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Contents

Editorial: Community Health Service	...	171
Fungus Disease of the Lung —S.C. Chakravarty	...	173
Tuberculosis Mortality rates in a South Indian rural population —A.K. Chakraborty, G.D. Gothi, S. Dwarakanath and Hardan Singh	...	181
Correlation between Sulphadimidine Acetylation test in Urine and Plasma Isoniazid level in determining Isoniazid Acetylation status in Pulmonary Tuberculosis patients —C.P. Sinha and S. Sinha	...	187
Profile of Granulomatous hepatic tuberculosis in India —D.S. Singh, A.K. Das, A.L. Aurora, S. Chandrasekhar, D.8. Bisht and B.N. Tandon	...	193
Effect of Aspirin in the control of Hyperuricemia and Arthralgia due to Pyrazinamide (PZA) therapy —K. Nataraja Iyer and P. Srinivasan	...	197
An evidence in favour of tuberculous etiology of Kales disease —D.P. Varshney, K.C. Singhal and M.A. Siddiqui	...	199
Atlanto-axial dislocation associated with sub-occipital cold abscess —S. Mohanty, S.V. Sharma and T.P. Shrivastav	...	203
Spontaneous tension pneumomediastinum —R.K. Gupta, Y.D. Singh and H.C. Vaishnava	...	206
Tuberculosis osteomyelitis of the skull —H.R. Tata	...	208
Macleod's syndrome in an adult —R.K. Narang, J.N. Banavalikar, Bharat Kumar Gupta, A.L. Dubey and Narendra Kumar	...	210
The Twentyfourth World Conference on Tuberculosis and administrative meetings of the International Union Against Tuberculosis - A Short Report —P.N. Raman	...	213

News & Notes

Abstracts

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COMMUNITY HEALTH SERVICE

The present generation has probably for the first time in history shown real motivating concern about the problem of Primary Health Care and evinced the political will to take concrete steps in this direction. The Bhore Committee in 1946 for the first time suggested a solution. A number of schemes have since then been advocated and partially implemented. The impact, however, has been far from satisfactory. A scheme, acceptable to all, feasible economically and from the point of personnel and yet effective, is still elusive.

Meanwhile, the debate goes on. Should the Primary Health Care be provided on the conventional lines, even if it is not, for obvious reasons, a minor replica of the service being provided at present in the urban and semi-urban parts of the country? Should the service be delivered through bare-foot doctors or surrogate doctors? Should the para-medical health workers be uni-purpose or entirely multipurpose? What should be the quantum of their training, the scope and extent of their duties and their limitations? Should the service be based on the practice of Allopathy or should it be a *pot pourri* of Allopathy, Homeopathy, Ayurvedic, Unani and what not?

Whatever be the ultimately accepted pattern of the health care, it is obvious that it has to be practical and non-formal. It should be designed in keeping with the local customs and traditions. It should not only be curative but also promotive and preventive. It should be feasible economically. And above all, it should be acceptable to the community, and delivered preferably by one of the underserved community's own member. The recently introduced Community Health Scheme, while fulfilling some of these criteria in respect of general health has many shortcomings. And, alas, it is woefully deficient in respect of tuberculosis care.

The Community Health Worker's (C.H.W.) Manual makes just one reference to tuberculosis! It says that B.C.G. vaccination should be made available to every child before the age of one year. No one to-day believes that B.C.G. alone will or can bring tuberculosis under control. The only other reference which indirectly applies to tuberculosis, without saying so, is the action prescribed for those having cough. It is mentioned that such persons should be isolated, especially from children; should be advised to cover their *nose* while coughing and sneezing and should not spit indiscriminately. Isolation, even if necessary, would by and large, be impracticable for any length of

time. Covering the nose (and not the mouth) will not stop dissemination of droplets, when coughing. The only relevant and important advice for such persons viz. to get their sputum examined and to take regular treatment if found tuberculous is conspicuous by its omission.

Tuberculosis is a major health problem and any scheme of Primary Health Care must include the following minimum provisions, in addition to B.C.G. vaccination :-

1. Examination of sputum by direct smear of all those having cough and sputum, with or without fever, chest pain and haemoptysis.
2. Facility for collection of anti-tuberculosis drugs as near the residence as possible.
3. Means for prevention and retrieval of drug default.

There should be no difficulty in including the last in the schedule of regular duties of the C.H.Ws. There should also be no objection in incorporating preparation of sputum smears in the job chart of the C.H.W. For, after all, if he is being required and taught to make blood smears when Malaria is suspected, there can be no reason why he cannot make smears of the sputum of suspects, fix these and send to the nearest microscopy centre exactly as in the case of blood smears. The expertise needed for collecting sputum and making and fixing smears is definitely less than that required for making blood smears. Many microscopy centres, even now, are getting this work done by sweepers on other class IV staff, if no laboratory technician is available. Any one, even an illiterate person, with just a little intelligence can be taught to do so within a few days.

Regarding drug distribution, it is an accepted fact that facilities to collect drugs at nearby centres help to reduce drug default. C.H.Ws are to be provided at the rate of one per village or one per thousand of the population for the larger villages. It would therefore be logical that drug distribution in accordance with the instructions of the D.T.C. or the P.H.I. doctor should also be the responsibility of the C.H.W. But before that is done, the possibility of misuse or abuse of drugs has to be safeguarded. If he is tempted to use these drugs, on his own, without proper diagnosis at the D.T.C. or P.H.I. for every case of cough etc., the consequences will be frightful.

It is, therefore, essential that the diagnosis of tuberculosis must first be made by the D.T.C. or P.H.I, and the regimen, dose etc, decided by the diagnosing physician before they are allowed to issue any anti-tuberculous drug stocked by them. Their training must also include proper maintenance of records and ability to identify evidence of intolerance of the anti-tuberculous drugs to be dispensed by them (which in this case will only be INH and thiacetazone). Their manual must clearly specify the conditions under which they can issue drugs and for what period and conditions under which drugs should be stopped and the patients referred to the nearest health centre for further advice.

The scheme has already been introduced and is functioning in many villages. Training of workers is in full swing. It is imperative that the manual should be quickly revised and suggestions made above in respect of sputum examination, drug distribution and regularity of treatment be included in the revised version. These additional but nevertheless essential responsibilities are well within their capability and are not likely to increase their work load to any appreciable extent, but will pay handsome dividend in respect of tuberculosis control.



FUNGUS DISEASE OF THE LUNG

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Fungi can be differentiated from bacteria by their bigger size, cell wall which consists of Chitin and their method of reproduction by budding and branching of hyphae.

Fungal affection of lung may be termed as deep mycosis or systemic mycosis. Fungus disease of lung may be classified according to the types of fungi.

A. Diseases caused by endogenous fungi (i.e. fungi which are present in normal people as harmless commensal but on occasions cause diseases).

1. Actinomyces Israelii causes Actinomycosis.
2. Candida albicans causes candidiasis.

B. Diseases caused by Exogenous fungi (i.e. fungi which come from outside of body).

(i) Yeast and yeast-like fungi.

- (a) Cryptococcus neoformans causes Cryptococcosis.

(ii) Filamentous fungi.

- (a) Nocardia asteroides causes Nocardiosis.
- (b) Aspergillus fumigatus causes Aspergillosis.
- (c) Rhizopus species causes Mucormycosis (Phycomycosis).

(iii) Dimorphic fungi (Fungi which have two phases *i.e.*, yeast phase and filamentous phase).

- (a) Histoplasma capsulatum causes Histoplasmosis.
- (b) Blastomyces dermatitidis causes Blastomycosis.
- (c) Coccidioidomyces immitis causes Coccidioidomycosis.
- (d) Sporotrichum (Sporothrix) Schenckii causes Sporotricosis.

Diagnostic work up

If the physician keeps in mind fungus infection of lung in differential diagnosis the chance of diagnosis of fungus disease of lung will be multiplied.

Fungus infection is more common in following conditions:

1. Due to reduced cell mediated immunity in immunosuppressive therapy;
2. Due to inhibition of host cell response in Corticosteroid therapy;
3. In leukopenia specially white cells below 1500 per cu. mm. ;
4. Renal failure due to impaired immunoglobulin synthesis;
5. Diabetes mellitus;
6. Leukemia;
7. Prolonged antibiotic therapy;
8. Pulmonary disease due to tissue destruction;
9. Lymphomas.
10. In prolonged intravenous catheterisation specially candidiasis.

There are certain conditions which should be looked with suspicion for fungus diseases:

1. Pneumonia not responding to antibacterial treatment;
2. Undiagnosed fever in a patient treated with corticoids, antibacterial or immunosuppressive drugs;
3. Oral and pharyngeal ulcerations;
4. Draining Sinuses of skin;
5. Meningitis and brain abscess;
6. After surgery if endocarditis;
7. If biopsy report shows granuloma in prostatitis, epididymitis or orchitis.

Diagnostic skin tests

The following fungus diseases give positive reaction to specific antigens (*i.e.* Coccidioidin for Coccidioidomycosis, Histoplasmin for Histoplasmosis, Blastomycin for Blastomycosis and Sporotrichin for Sporotrichosis. Antigen is given intradermally in 1:100 dilution and read after 48 hrs. Induration of 5 mm or more is read as positive. Sporotrichin antigen for Sporotrichosis is given intradermally in 1:250 dilution, induration read after 24 hrs. Induration of 5 mm or more is taken as positive (Schneidau) *et. al.* (1964), Chakravarty (1968) *et. al.*

Laboratory Diagnosis (Ajello *et. al.* 1963)

1. Fresh sputum to be examined;
2. Urine — first urine in the morning to be examined.;
3. Blood — to be collected in heparinised 10 ml tube (sterilised);
4. Bronchial aspirate to be examined fresh;
5. Scalene node biopsy in histoplasmosis may show H. Capsulatum;
6. Bone marrow — in disseminated histoplasmosis is positive for H. Capsulatum in 75% cases;
7. Liver biopsy — may be helpful in disseminated histoplasmosis;
8. Cerebrospinal fluid in cryptococcosis.

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Direct examination of pus, blood, sputum on slide.

- (a) unstained with 10 % KOH solution added,
- (b) Gram stain — for actinomycosis and Candidiasis. (c) Ziehl-Nelson stain for Nocardia. (d) Lacto-phenol-cotton blue for filamentous fungi, (e) India ink — if cryptococcosis is suspected.

Tissue Stains

The following stains may be used for staining biopsied or autopsied tissues.

- (a) Hematoxyline or Eosin. (b) Periodic Acid Schiif (P.A.S.) stain except for actinomycosis. (c) Gram stain for Actinomycosis and Nocardiosis.

Culture methods

Materials may be cultured in the following media:

- (a) Sabourand glucose agar at room temperature (i.e. 22°-25° C). This medium contains glucose, agar and neoptone. It is easy to prepare,
- (b) Brain heart infusion agar (BH I).

Animal inoculation

- (a) Materials with equal portion of 5 % gastric mucin are injected in animals for establishing pathogenicity as also tissue phase of the fungus.
- (b) Embryonated hen's egg — for primary isolation of pathogenic fungi.

Serological methods

(a) Complement fixation test (C.F.T.) for Histoplasmosis, Coecidioidomycosis and Blastomycosis, gives positive reactions, (b) Agglutination test is good for Candidiasis, Cryptococcosis, Blastomycosis, Histoplasmosis and Sporotrichosis. (c) Precipitation test (gel-diffusion test) is good for Aspergillois, Blastomycosis, Coecidioidomycosis, Sporotrichosis. (d) Fluorescent antibody technique (F.A.T.) is good for Histoplasmosis, Cryptococcosis, Candidiasis, Sporotrichosis, Actinomycosis.

Autifungal Drugs

Amphotericin-B-(Fungizone). — This drug is a broad spectrum antifungal antibiotic. It is prepared from the fungus streptomyces nodosus. Not effective in Actinomycosis or bacterial infection. It is active against deep mycosis. It is available in 50 mg vial in a powder form. This is diluted in

500 ml. or 1000 ml 5 % glucose and infusion given lasting about 3 hours. It is preferable to start with 1 to 5 mg given over a 30-60 minutes period and on each subsequent days the dose can be doubled.

Toxicity and side effects of Amphotericin — B :

- (I) 1. Fever, chills, nausea, vomiting. 2. Local venous thrombosis (Phlebitis). 3. Nephrotoxicity — raised B u N and creatinins. 4. Convulsions and heart block. 5. Hepatic Coma. 6. Peripheral neuritis. 7. Bone marrow depression.

Other routes through which Amphotericin may be given : 1. Intrathecal. 2. Intra Pleural. 3. Aerosol — 25 mg/per day in distilled water. 4. Topical.

Precautionary measures for Amphotericin — B Side Effects :

- 1. For fever — acetylsalicylic acid— 15 to 30 grains. 2. Intravenous injection of Codeine Phosphate — 30-60 mg for chills. It ameliorates chills and aches within 5 minutes. 3. For vomiting — 5 — 10 mg of intramuscular injection of Prochlorperazine. 4. If BuN reaches 80 mg or more per 100 ml -- Discontinue amphotericin for 5 days (this is to protect from kidney damage).

(II) *Nystatin* : It is a tetraene antibiotic derived from the fungus, *Streptomyces noursei*. It is available in 500,000 units of oral tablet form. It is highly effective against *Candida albicans*. It is not absorbed through intestine but locally effective. It cannot be injected as it is too toxic.

(III) *Aerosol* : in respiratory tract infection: 50,000 units of sterile Nystatin powder suspended in 40% propylene glycol in 4 ml of normal saline. 1 ml of this suspension is given by serosal every six hours, daily for two-three weeks along-with oral nystatin (Chakravarty 1967) in respiratory Candidiasis.

IV Topical-Nystatin can be given topically : There are no side effects of nystatin.

- 3. *Hamycin* — This is produced by *Streptomyces Pimprina*, discovered in India in 1961.

The antifungal spectrum resembles that of Amphotericin B but result of clinical trials are variable.

Dose : 500 mg (2 capsules) daily for 1-2 weeks.
Side effects : Gastro-intestinal disturbances.

- 4. *5-Fluorocytosine* : Experimentally and Clinically it has protective action in Candidiasis and Cryptococcosis. It is contraindicated in

pregnant women as it is found teratogenic in experimental pregnant animals.

5. *Clotrimazole* : It has a protective action in Candidiasis, Aspergillosis, Histoplasmosis and Coccidioidomycosis. Effective orally in a dose of 100 mg/kg body weight. It has got no effect by parenteral route.

6. Saramycetin (x-5079C) Active against histoplasmosis, blastomycosis, Sporotrichosis and Aspergillosis, it is given Subcutaneously 6 hourly in 3-17 mg/kg/day.

Side Effects : Changes of liver function tests and Eosinophilia.

7. Natamycin — It is useful in broncho-pulmonary aspergillosis by inhalation (Pimaricin) (Edwards & La louche, 1964).

8. *2-Hydroxystilbamidine*: It has got suppressive effect on blastomycosis. It is given intravenously in a dose of 225 mg daily in 200 ml of 5 % glucose over 2 hours. Total dose is 8.0 gm. It may be repeated after 3 weeks.

Side Effects: Chill, fever, nausea, hypotensive episodes, abnormality of liver function tests.

9. *Penicillin* — This is the drug of choice in Actinomycosis of lung. 4-6 million units are given intra-muscularly for 6 weeks followed by 6,00,000 units daily for another 6 weeks. No resistance to drug has been observed.

10. *Sulphonamides* : This is the drug of choice for nocardiosis. 0.5-2.0 gms every 6 hourly daily by oral route for 6-8 weeks.

11. *Potassium Iodide* : It is very effective against lymphocutaneous form of sporotrichosis. On disseminated sporotrichosis it has got mild effect. In sporotrichosis of lung, Amphotericin B is the drug of choice. Side effects of Iodides: Metallic taste, Coryza, Symptoms of 'common' Cold, Laryngitis, Parotitis, Skin Rashes.

After describing the classification, diagnostic methods and antifungal drugs briefly the individual fungus disease will be described.

(1) *Actinomycosis* : This is caused by anaerobic, endogenous fungus *Actinomyces israelii* (*A. israelii*-ray-fungus). *A. israelii* is a normal inhabitant of mouth and tonsil. Primary infection of lung results from aspiration. By and large the symptoms and signs including radiological pictures mimic tuberculosis of lung or lung abscess. Characteristic findings is "Sulphur granules" in Pus. These contain ray-fungus.

Ind. J. Tub., Vol. XXV, No. 4

Sinus formation in skin and subcutaneous tissue is common. In a survey among tuberculosis hospital patients Chakravarty & Fernandez (1977) found 1.6 per cent tuberculosis patients had coexistence of actinomycosis of lung along with pulmonary tuberculosis.

Diagnosis : Direct Examination : Typical ray fungus in sputum and by gram stain branching filaments are found which are gram positive.

Culture media 1. Brain heart infusion broth, 2. Enriched thioglycollate broth. Both under anaerobic conditions. Anaerobic condition is easily obtained by pyrogallol — carbonate Seal (Ajello et. al. 1963) Culture from tissue: It is difficult to grow *A. israelii* from tissue. Chakravarty et. al. (1974) developed a new technique by injecting tissues suspected to be infected with *A. israelii* alongwith with "associate" bacteria i.e. *H. influenza* in animals.

By injecting the combination in animals they produced granules in lung of rabbits which showed clubs which were pencil shaped and echinulated and had spines on their surface, (see fig. 1 & 2). This was a type of *A. israelii*.

Treatment : Penicillin is the drug of choice.

2. *Nocardiosis* : This is caused by *Nocardia asteroides* which is aerobic and partially acid fast (0.5 to 2.0% Sulphuric acid). This disease

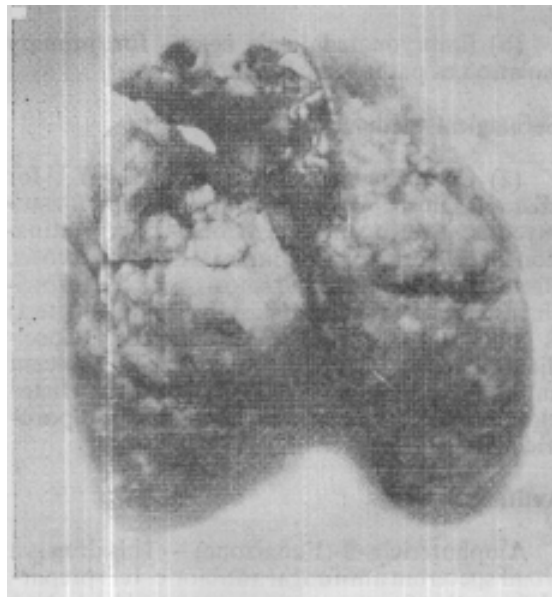


Fig. 1. *A. israelii* and *H. influenza* ("Associate bacteria") injected intratracheally in rabbits, produced miliary nodules of actinomycosis in lung.

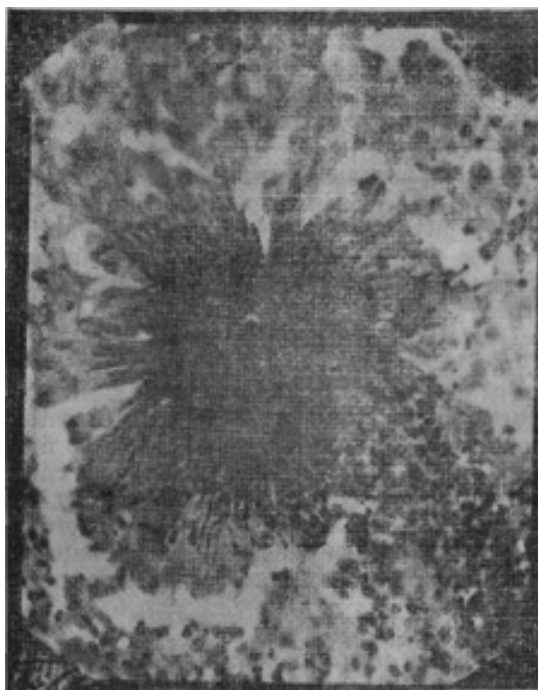


Fig. 2. *A. israelii* from granules of miliary nodules of experimental rabbit lung show :

- (1) Intensive staining with Eosin
- (2) Very elongated pencil-shaped clubs
- (3) Some clubs are clearly echinulated and show coarse spine which are not usually seen.

mimic tuberculosis and diagnosis becomes difficult when filaments fragment, as they are partial acid, fast and simulate *M. tuberculosis*. Like other opportunistic fungi this disease is associated with conditions which lower resistance of host (i.e. Diabetes mellitus, Leukemia, Corticosteroid treated patients, tuberculosis).

In a survey in tuberculosis patients in a tuberculosis hospital Chakravarty et. al. (1968) found about 2.4% tuberculous patients had pulmonary Nocardiosis along with Pulmonary Tuberculosis.

Moquown (1955) reported 5.0% Nocardiosis in a tuberculosis Hospital. Shome (1974) recorded 83.9% of 31 cases recorded were diagnosed administered initially as tuberculosis and 68% of the cases were administered Corticosteroids and/or antibiotics prior to diagnosis.

Diagnosis: Gram stain — *N. asteroides* is gram positive, filamentous or Diphtheroid forms. Ziehl — Nelson stain — Partial acid fast filament.

Culture : In Sabouraud's agar as also in sterile broth with sterile paraffin coated glass rod (Meclung method).

Treatment : Drug of choice is sulfadiazine. In case this drug is not tolerated Tetracycline may be given.

3. *Cryptococcosis (Torulosis)* : It is caused by the fungus *Cryptococcus neoformans*. Pigeon feces is reported to be a good source of infection. Pulmonary cryptococcosis is associated with Hodgkin's disease. Patients with Hodgkin's disease should avoid handling Pigeons.

Pulmonary form of *Cryptococcosis* is generally of low virulence and some time go unrecognised. X-Ray of chest shows consolidation but cavitation is uncommon. Mediastinum is not usually involved.

In India Khan et. al. (1959) and Shome (1974) described *Cryptococcosis*. Shome (1974) described 50 cases of Pulmonary *Cryptococcosis* and felt that there is a shift towards pulmonary infection in contrast to meningeal infections as also there is rising of incidents of *Cryptococcosis* in India.

Diagnosis : Thick walled yeast cells in sputum. Capsule of the yeast is better demonstrated in India ink. Capsule stands out as white in dark medium of India ink.

Culture : Sabouraud's medium at room temperature. Serological test: Agglutination and Fluorescent antibody technique.

Treatment : Amphotericin B is the drug of choice.

4. *Aspergillosis* : The disease is generally caused by inhalation of spores of *Aspergillus fumigatus*, under conditions of increased host susceptibility. *A. flavus*, *A. niger*, *A. nidulans* also can, cause *Aspergillosis*. This disease may be presented as following :

(i) *Aspergilloma* : A solid growth of fungi (Fungus ball). These affect a cavity such as lung abscess, bronchiectasis tuberculosis.

(ii) *Asthmatic type* : Presents with asthmatic symptoms, fever, prostration following inhalation of spores. Recovery is completed within 12-36 hours.

(iii) *Disseminated* : This is predisposed by underlying disease. Not common.

It may spread to other organs. X-Ray picture

of aspergilloma looks like a solid mass with an air crescent and in fluoroscopy—movement of the fungus ball with change of posture of the patient as the fungus ball is not attached to the cavity.

Diagnosis : Repeated demonstration of mycelial fragments in sputum.

Media : Sabouraud's medium at 37°C.

Serological study (Henderson et al., 1968)

(i) Precipitin (Gel Diffusion Test) — 95 % are positive

(ii) Complement fixation tests.

Treatment : (i) Natamycin (Pimaricin) aerosol Natamycin in dose of 2.5 mg in 1.5% suspension given 2-3 times daily, (ii) Amphotericin B (iii) Resection for Aspergilloma.

In India Pamra et al. (1972), Shome (1974) described aspergillosis of lung.

5. *Histoplasmosis* : This disease is caused by the fungus *Histoplasma capsulatum*. The fungus affects the cells of reticuloendothelial system. Mainly three clinical types are described (Emmons et al. 1970).

(i) *Primary histoplasmosis* : about 90-95% cases are asymptomatic. The rest present in various expressions of chest diseases such as cough fever etc. X-Ray of chest shows multiple lesion in both lung fields. These lesions are slowly calcified. These calcified lesions if sectioned and stained show *H. Capsulatum* — specially the core of lesion which is still caseous. Bilateral lymphadenopathy is present. Diagnosis is based on Histoplasmin skin test and complement fixation test.

(ii) *Re-infection type* : This is similar to reinfection tuberculosis. Patients show multiple calcification of lung with fibrocaseous type of lesion. Histoplasmin skin test is strongly positive.

(iii) *Progressive disseminated Histoplasmosis*. About 1 % of patients progress and go to fatal form of disease. This is mostly at extremes of age i.e. infants and old age. Dissemination is also in other organs. Bone marrow will show *H. Capsulatum* and there will be anemia, leukopenia and splenomegaly or Hepato-Splenomegaly.

Diagnosis : By Giemsa's Stain —intracellular yeast like organism is found.

Culture media : Yeast form is cultured in B.H.I, agar and mycelial form and chlamy-

dospore (which is characteristic of *H. Capsulatum*) is produced in Sabouraud's medium.

Serological test :
1. Agglutination
2. Complement fixation tests.

It should be remembered that if the patient is skin tested with Histoplasmin before C.F.T. —it might show false positive results. It is advisable to withhold Histoplasmin skin test before the blood is taken for Serological tests.

Treatment : Amphotericin — B.

The first case of Histoplasmosis in India was reported by Panja and Sen (1954) from Calcutta. Chakravarty et al. (1968) reported for the first time a case of histoplasmosis of lung in a child by lung biopsy by identifying intracellular *H. Capsulatum* from caseous portion of the core of calcified nodules in lung. Lal & Mohapatra (1964) reported another case of Histoplasmosis from Delhi.

6. *North American blastomycosis* : This disease is caused by a fungus *Blastomyces dermatitidis*. Pulmonary blastomycosis is a progressive suppurative disease with pulmonary symptoms. In x-ray—mediastinal adenopathy and infiltrations of lung noted.

Blastomycin skin tests is positive except in severe disseminated disease. Complement fixation test is positive in high titre.

Treatment : The drug of choice is Amphotericin B.

It is debatable whether North American blastomycosis exists in India.

7. *South American blastomycosis* : Is caused by *Blastomyces brasiliensis*. It is mostly reported from South America, specially Brazil. Fungus invades lungs. Secondarily by hematogenous spread. Treatment is Sulphonamides.

8. *Coccidioidomycosis* : This is caused by the fungus *Coccidioides immitis*. Primary pulmonary diseases is benign and self-limiting and mostly asymptomatic. Progressive form is serious and some time fatal.

Primary type simulates influenza and generally have arthralgia. X-ray of chest show patchy opacities in lungs. Sometime a shadow like tuberculoma of lung. Hilar adenopathy is found. Progressive form: 1 in 1000 patients of primary type become progressive. It simulates Pneumonia or tuberculosis of lung.

Diagnosis : The fungus is non-budding thick walled structure filled with endospore. Precipitin test is positive in early cases. Complement fixation test more than 1 : 4 is suggestive of active disease.

Treatment : Drug of choice is Amphotericin B.

9. *Candidiasis* : This disease is caused by Yeast like endogenous fungus *Candida albicans*. Occasionally other species of *Candida* may cause the disease. Bronchopulmonary candidiasis may be classified as (i) Bronchial (ii) Pulmonary (iii) A combination of bronchial and pulmonary types.

Bronchial type : simulates chronic bronchitis and bronchial asthma. Pulmonary type simulates tuberculosis. Primary pulmonary candidiasis is rare.

Diagnosis : Since *Candida albicans* is endogenous, mere presence of *C. albicans* in sputum does not mean candidiasis. The surest test for candidiasis of lung is to identify *Candida albicans* in biopsied lung tissue. But this is not practicable in daily routine case. So there are criteria defined by Chakravarty (1967) for diagnosis of candidiasis of lung.

- (i) Repeated culture of *C. albicans* in sputum with heavy growth.
- (ii) Culture of *C. albicans* in bronchial aspirate help further in confirming candidiasis.
- (iii) Patients should have clinical symptoms and signs.
- (iv) The patient should be cured with anti-fungal antibiotic independent of other treatment.

Candida albicans may be grown at Sabouraud's media at 37°C.

Drugs : Suspension of sterilised Nystatin powder in 40% Propylene Glycol in normal saline should be given by aerosol with IPPB machine at 10-15 cm of water pressure or by hand nebuliser every six hours. Each time 12,500-15000 units of Nystatin to be given by Aerosol (Chakravarty, 1967). This should be given 2-3 weeks.

If needed when Nystatin aerosol fails Amphotericin B be given intravenously. In India, Chakravarty (1967) Shome (1974) have reported candidiasis of lung.

10. *Sporotrichosis* : This disease is caused by *Sporothrix (Sporotrichum) schenckii*. This disease mainly affects skin of subcutaneous tissues. Lungs are rarely affected. Only a few cases of pulmonary sporotrichosis are reported in literature. If lung is affected it simulates lung abscess.

Diagnosis : Culture of *S. Schenckii* in BHI agar at 37°C. (ii) Latex agglutination test to be done.

Treatment: Potassium Iodide in skin infection and Amphotericine B in lung infection.

In India, cases are recorded by Dey (1959) from Assam and Calcutta. Chakravarty et. al. (1968) did sporotrichin skin test survey in Delhi area (both urban & rural areas) it is mostly positive in agricultural population and gardeners. More the duration of occupation of gardeners and farmers more the positive reaction to sporotrichin. This shows that this infection is present but may be the disease is mostly a self-limiting disease.

11. *Mucormycosis (phycoiycosis)* this disease is caused mainly by *Mucor* Species in diabetic patients or in Leukemia. It presents as bronchopneumonia or pneumonia.

Diagnosis : Presence of *Mucor* in sputum.

Treatment : Amphotericin B.

Recommendation : In India still now tuberculosis is the biggest problem. Fungus disease is also important because many patients of tuberculosis may be associated with fungus disease of lungs as also fungus disease is increasing.

Moreover, in tuberculosis clinics many patients come with tuberculosis but they do not respond to antituberculous treatment as they are not tuberculous but fungus disease. There are also some cases of tuberculosis patients diagnosed as resistant to antituberculous drugs because after initial improvement they do not improve further. Probably some of these tuberculous patients have also associated fungus infection. At the present moment there is no mycological unit (except at one or two places in India) attached to tuberculosis clinics. As a result these patients go undiagnosed.

I would suggest that in all provincial capitals as also with all important tuberculosis clinics there should be a mycological unit. District tuberculosis centres can refer the specimen to these units. Mycologists and technicians may be trained at the V.P. Chest Institute, Delhi University, All India Institute of Medical

Sciences, New Delhi, School of Tropical Medicine, Calcutta or other recognised mycological laboratories in this country. This will help to cater better treatment in our existing tuberculosis clinic.

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TUBERCULOSIS MORTALITY RATE IN A SOUTH INDIAN RURAL POPULATION

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Introduction

Tuberculosis mortality rate in general population is one of the important epidemiological parameters for denning the problem, in any country, from time to time. In some of the advanced countries, mortality rates have been widely used to determine the trend of tuberculosis over the years^{1,2}. Comparative study of mortality rates due to different causes helps in deciding the relative priorities. In India in the absence of adequate diagnostic facilities, notification of cause of death as well as regular and systematic post-mortem examinations, it is difficult to obtain precise information on mortality. In this report, an attempt has been made to estimate mortality due to tuberculosis in comparison with total mortality. Data obtained from "A five year epidemiological study in a rural population in South India"³ has been analysed for this purpose.

Study Area and Method

Four surveys were conducted using the same methods in 119 randomly selected villages in the 3 taluks of Bangalore district — Channapatna, Magadi and Nelemangala during 1961-68. Intervals between I and II and between II and III surveys were 1½ years whereas it was two years between III and IV surveys. The total population studied constituted 15% of the population in the 3 taluks. The coverages for examinations were uniformly high in all the surveys, being about 80% on the average.

The entire population of the villages was registered and those who were aged 5 years and over were offered X-ray examination in the villages. From persons who had any X-ray abnormality two samples of sputa were collected. X-rays were interpreted by two readers independent of each other, and those for which interpretations were at variance between the two readers were submitted to an umpire reader for final interpretation. On the basis of this interpretation X-rays were classified as³ :

N : Normal, A : non-tuberculous, B : Tuberculous, inactive, C : possibly tuberculous, probably active, D : possibly tuberculous, probably or definitely active.

Sputum specimens were examined by smear microscopy and by culture; Identification tests

for Mycobacterium tuberculosis were carried out on all positive cultures.

Persons who had radiological abnormalities classified as C or D but sputum negative for tuberculosis ("suspect disease") together with persons whose sputa were positive on culture and also had any radiological abnormality at the current survey ("cases") were taken as patients of pulmonary tuberculosis for this report. They will be referred to in this paper as "TB".

Persons who were radiologically normal (N) together with those having shadows classified as A or B whose sputa were negative will be referred to in this paper as "NAB".

Material

At the I survey 32,009 persons aged 5 years and over without BCG scars were examined and they were followed up at II survey. Similarly, 29,489 persons were examined at II survey and followed up at III survey and 29,241 between III and IV surveys. Their age-wise distribution is presented in Table 1. The proportion of persons with BCG scars, excluded from this report, ranged from 2.5 % at I survey to 3.7 % at the IV survey³. Of those examined at I and II surveys, 31,502 were NAB and 507 were TB. Persons who had NAB or TB in four age groups are presented in Table 2, by survey at which they were examined. Material is not presented sex-wise but no differences were observed in crude mortality rates between males and females by different age groups.³

Findings

Mortality rate due to all causes (Table 1)

Of the total 32,009 persons of I survey and followed-up at II survey, 439 died between the period (888.7 per 100,000 per year), of 29,489 between II and III surveys 383 died (826.0 per 100,000 per year), and of 29,241 between III and IV surveys 560 died (963.3 per 100,000 per year). The annual mortality rate due to all causes on five year observation could be calculated as 893 per 100,000 population aged 5 years and over.

The age specific death rates were lowest in 5-14 years and highest in 55 years and over. In the latter age group it was more than 4134 per 100,000 annually. Age specific death rates were significantly higher in 55 years & over age

Table 1

Mortality Rate due to all causes between different surveys by age

Age group	Survey MI (1½yrs.)			Survey II-III (½yrs.)			Survey III-IV (2 yrs.)		
	Popula- tion examin- ed	Died	Annual all causes mortality rate per 100,000*	Popula- tion examin- ed.	Died	Annual all causes mortality rate per 100,000*	Population examined	Died	Annual all causes mortality rate per 100,000*
5—14	11936	52	282.3	10935	51	296.6	10645	75	354.4
15—34	10616	100	610.4	10060	78	493.1	10123	112	556.5
35—54	6596	101	992.2	5925	87	933.9	6007	125	1046.7
55 +	2861	186	4212.8	2569	167	4134.4	2466	248	5058.6
Total	32009	439	888.7	29489	383	826.0	29241	560	963.3

* To arrive at annual rate correction factors applied:

I-II survey ...0.648 II-III survey ... 0.636 III-IV survey ...0.503.

group as well as in total 5 years & over population at the third period (III-IV surveys) as compared to those in the earlier two periods (I-II surveys & II-III surveys).

Deaths Attributable to Tuberculosis

Deaths due to all causes among the group 'NAB' and group TB' are presented in column 3 and 5 respectively between different surveys (Tables 2, 3, 4). All the deaths among persons with sputum positive disease or suspect disease i.e., in group 'TB' cannot be attributed to tuberculosis alone. Presuming that persons with active tuberculosis have a similar chance of dying from other causes as for the group who are normal or suffering from diseases other than tuberculosis, only the excess proportion of deaths occurring among the TB group over that in the NAB could be attributable to tuberculosis. The observed all causes death rate among the NAB have been applied to the group TB age-wise (figures in cols. 3 x col. 4 -I- col. 2 in Tables 2, 3 and 4), and thus the absolute number of deaths among the TB group supposed to be due to causes other than tuberculosis have been estimated (col. 6 in Tables 2, 3 and 4). It is seen that between the I and II surveys, estimated number of deaths due to other causes among TB were negligible in 5-14

years, one in 15-34 years, 2.1 in 35-54 years, 10.6 in 55 years and over and 13.9 among the age groups combined (Table 2). The estimated number of deaths between II and III as well as between III and IV surveys are similarly presented in Tables 3 and 4. On subtracting the estimated number of deaths due to other causes among the TB as mentioned above (col. 6 of Tables 2, 3 and 4) from the observed number of deaths from all causes among the group TB age-wise (col. 5 of Tables 2, 3 and 4), the estimated number of excess deaths are calculated separately between I and II, II and III and III and IV surveys (cols. 3, 6, 9 of Table 5). All these deaths among the group TB, which are in excess of those that could be due to causes other than tuberculosis, may reasonably be attributed to tuberculosis.

Based on the above calculations it is estimated that the annual death rates due to tuberculosis in 100,000 population between 1 and II surveys were 10.00 in 5-14 years, 79.0 in 15-34 years, 146.0 in 35-54 years, 394.0 in 55 and over years and 95 in combined are group (Table 5). The tuberculosis mortality rates as estimated between II and III as well as between III and IV surveys in the above age groups are presented in columns 7 and 10 of Table 5. Age-wise as well as overall rates were not different between surveys I and II,

Table 2 Deaths between I and II surveys among population groups classified as active Tuberculosis (TB) and otherwise (NAB)

Age group	Normal and non-TB (NAB)	Deaths among normal and non TB (NAB)	Sp. pos. & Suspect cases (TB)	Deaths from all causes among-TB	Estimated absolute no. of deaths due to non-TB causes among TB
					(Col. 3xCol. 4)
					Col. 2
1	2	3	4	5	6
5—14	11888	50	48	2	0.2
15—34	10498	86	118	14	1.0
35—54	6435	84	161	17	2.1
55 +	2681	158	180	28	10.6
Total	31502	378	507	61	13.9

Table 3

Deaths between II and III surveys among population groups classified as active Tuberculosis (TB) and otherwise (NAB)

Age group	Normal and non-TB (NAB)	Deaths among normal and non-TB (NAB)	Sp. pos. & Suspect cases (TB)	Deaths from all causes among TB	Estimated absolute no. of deaths due to non-TB causes among TB
					(Col. 3 x Col. 4)
					Col. 2
1	2	3	4	5	6
5—14	10912	50	23	1	0.1
15—34	9971	72	89	6	0.6
35—54	5812	73	113	14	1.4
554-	2437	146	132	21	7.9
Total	29132	341	357	42	10.0

Table 4

Deaths between III and IV surveys among population groups classified as active Tuberculosis (TB) and otherwise (NAB)

Age group	Normal and non-TB (NAB)	Deaths among normal and non-TB (NAB)	Sp. pos. & suspect cases (TB)	Deaths from all causes among-TB	Estimated absolute no. of deaths due to non-TB causes among TB
					(Co. 3 x col. 4)
					Col. 2
1	2	3	4	5	6
5—14	10633	75	12	—	0.1
15—34	10059	101	64	11	0.6
35—54	5908	111	99	14	1.9
55 +	2403	214	63	34	5.6
Total	29003	501	238	59	8.2

II and III and III and IV. The average of the periods is calculated to be 84/100,000 annually. The death rates were the highest in 55 years and over age groups, the lowest in 5-14 years, and showed an increasing trend with age.

The age-specific tuberculosis mortality rate showed some decrease between II and III surveys over that between I and II surveys; but these generally increased between III and IV surveys, though none of these differences were statistically significant. Since similar changes were also observed in the incidence and prevalence of the disease,³ this could be attributed to the effect of drought between III and IV surveys.

Discussion

From a study of the same material as in the present report, the annual crude death rate for population in all ages both sexes, registered at I survey was 1100 per 100,000.³ The death rate due to all causes was 1900 per 100,000 in population who were aged 0-4 years at the time of I survey. Deaths in between two surveys among the new borns were not recorded in the study. The crude death rates in total population were

lower than 1640 per 100,000 reported by Central Bureau of Health Intelligence for Indian rural population during the same period.⁴ This is because of high mortality among the newly born between two surveys being excluded in the study. It is also possible that the crude death rates in the study area were lower than the all India rate since crude death rates are known to be somewhat lower for Karnataka than for the entire country.⁵ Annual all causes death rate of 893 for 100,000 among both sexes in 5 years and over age group, in this study, were also significantly lower than 1211.3 per 100,000 calculated from Frimodt Moller's study material (236 deaths in 4138 persons in 4.7 years).⁶

The absence of information about cause of death is one of the reasons for the remarkably meagre knowledge on mortality rates due to tuberculosis in India. McDougal in 1949⁷ quoted the cause specific mortality rate due to tuberculosis in India as 250 per 100,000. Frimodt Moller⁸ estimated the mortality rate in Madanapalle town to be 253 per 100,000 in 1949, and 64 to 21.2 per 100,000 between repeat surveys in urban and rural Madanapalle areas.⁶ In these surveys deaths among all persons diagnosed as

Table 5

Estimates of annual tuberculous mortality rates in population between different surveys (by age)

Age group	Between surveys I&II			Between surveys II & III			Between surveys III & IV		
	Total population	Estimated no. of deaths	Annual* mortality rate per 100,000	Total population	Estimated no. of deaths	Annual* mortality rate per 100,000	Total population	Estimated no. of deaths	Annual* mortality rate per 100,000
1	2	3	4	5	6	7	8	9	10
5—14	11,936	2	9.8	10,935	1	5.8	10,645	—	—
15—34	10,616	13	79.4	10,060	5	34.1	10,123	10	51.7
35—54	6,596	15	146.4	5,925	13	135.2	6,007	12	101.3
55 +	2,861	17	394.1	2,569	13	324.3	2,466	29	580.3
Total	32,009	47	95.4	29,489	32	69.2	29,241	51	87.6

* To arrive at annual rate correction factors applied:

I-II survey... 0.648

II-III survey ... 0.636

III-IV survey ... 0.503

having active tuberculosis were regarded as deaths due to tuberculosis. In the study under report, however, an attempt has been made to arrive at the tuberculosis mortality rate by subtracting the proportionate number of deaths due to causes other than tuberculosis from the deaths due to all causes among persons with active tuberculous disease. The rates presented in the study may thus be better estimates of mortality rates attributable to tuberculosis.

No doubt the mortality rates presented by Frimodt Moller in 1949⁸ were over estimates due to the methodology in arriving at the rate. Yet, in the absence of better estimates and owing to similar figures reported elsewhere⁷ (250/100,000) it could be safely presumed that tuberculosis mortality rates of 1949, upto which time no specific anti-tuberculosis drugs were available, represented the natural tuberculous mortality rate in Madanapalle. Compared to that the rates reported in the present study (84/100,000) per year could be taken as indicative of a declining trend.

The remarkable difference in death rates reported from survey to survey in Madanapalle

during 1949-55 period were due to facility of treatment made available to tuberculous patients diagnosed in Madanapalle area since 1949.⁹ No organised anti-tuberculosis services were available for the area of the present study and there was no such marked difference in the death rates due to tuberculosis over a period of five years. The latter may be taken to represent a natural situation without anti-tuberculous control programme.

Proportional mortality rate from tuberculosis i.e., ratio of cause specific death rate due to tuberculosis to the total death rate in 5 years & over age group in the present study was about 11 % at the first interval (I-II survey) and between 8 and 9% at the later two intervals (i.e., II-III & III-IV surveys). This contribution was the least in age group 5-14 years (2-4%) and the highest in age group 35-54 years (about 15% in first two periods and about 10% in the last period of follow up). Though tuberculosis mortality rates increased with age and the highest was in 55 years and over age, yet, the contribution made by tuberculous deaths to the total deaths did not show an increasing trend with age.

Table 6

Proportional mortality rates due to tuberculosis

Age group	Deaths due to tuberculosis as proportion of total deaths (%)			
	1 st period (I-II survey)	2nd period (II-III survey)	3rd period (III-IV survey)	Total (I-IV survey)
1	2	3	4	5
5—14	3.5	2.0	—	1.6
15—34	12.9	6.9	9.3	9.9
35—54	14.8	14.5	9.6	12.7
55 +	9.4	7.8	11.5	9.8
Total	10.7	8.4	9.1	9.4

In 55 years and over age group it was around 10% i.e., almost the same as for age group 15-34 years and the average of entire 5 years and over age group (Table 6). This was inspite of the highest prevalence of tuberculous disease in 55 years and over age group.

Summary

Information on cause specific mortality rates due to tuberculosis in India is inadequate. In the study under report, these have been estimated based on the data obtained from a five year epidemiological study in a rural population in South India. For this purpose, the estimated number of excess deaths due to causes other than tuberculosis among patients of tuberculosis, have been attributed to the disease. The annual cause specific death rate due to tuberculosis was 84 per 100,000 population aged 5 years & over and it represented about 9 % of mortality due to all causes in the same population. The death rates were the highest in 55 years and over age group, the lowest in 5-14 years and showed an increasing trend with age.

Compared to the estimates of tuberculous deaths in India available for 1949 (about 250/100,000), the present rates were lower.

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CORRELATION BETWEEN SULPHADIMIDINE ACETYLATION TEST IN URINE AND PLASMA ISONIAZID LEVEL IN DETERMINING ISONIAZID ACETYLATION STATUS IN PULMONARY TUBERCULOSIS PATIENTS

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Introduction

In the liver hepatic-N-acetyl transferase enzyme is present which takes part in the acetylation of Isoniazid. The larger the amount of this enzyme, the greater the speed of acetylation (Evans⁵, and White; 1964 and Hearse,¹⁰ and Weber; 1973).

The sulphadimidine is also acetylated in liver by the same enzymatic processes and its acetylation rate runs closely parallel to that of Isoniazid acetylation (Peters, et. al;¹² 1965 white,²³ and Evans 1968). The technique of estimation of both free and acetylated (Total minus free) sulphadimidine is comparatively simpler than those of Isoniazid estimation.

The present study has been carried out to determine the suitability of sulphadimidine acetylation test by the urine method as an indicator of Isoniazid inactivation status. For this purpose, a comparative evaluation has been made among the same tuberculosis patients by employing sulphadimidine acetylation test in urine as well as chemical extraction method for free Isoniazid level in Plasma.

Materials and Methods

(1) Selection of Subjects

The 60 patients of pulmonary tuberculosis who were having radiological lesions in lungs and A.F.B. in their sputum smears (W.H.O. expert committee on Tuberculosis)²⁴ - - were chosen. All of them were the patients of Rajendra Medical College Hospital, Ranchi. The patients with history of asthma, eczema, hay fever and major abdominal surgery, or like ones who reacted adversely to any drug were excluded from the present study. Chemotherapy was stopped for two days prior to investigation. The urine was tested early in the morning by S.N.P. test (Rao,¹³ et. al; 1967) to detect the presence of Isoniazid and acetyl-Isoniazid. Only such patients whose urine was free from Isoniazid and acetyl-Isoniazid were further investigated.

2. Doses of Sulphadimidine administered

The patients were administered sulphadimi-

dine 44 mg/kg. body weight early in the morning with a glass of water on empty stomach. They were not allowed to take anything by mouth for the next two hours. No other restriction was imposed on them (Rao,¹⁴ et. al; 1970 and Sen¹⁸ et. al. 1972).

3. Collection of Urine

The patients were asked to empty their bladders five hours after administration of sulphadimidine and were once again given a glass of water each to promote urine formation. The urine was collected in dry and clean glass vessels 6th hour after administration of Sulphadimidine and stored at room temperature. Rao¹⁴ et. al.; (1970 Sen¹⁸ et. al. 1972).

4. Estimation of Sulphadimidine

The total and free sulphadimidine in urine were estimated by Bratton and Marshall Method¹ Varley H. (1962). Total minus free sulphadimidine were estimated, is the acetylated form and their percentage was calculated. (The decimal figures obtained were rounded up to the nearest of the whole number to simplify the calculation).

5. Doses of Isoniazid

After 6 to 10 days all the above patients were administered oral Isoniazid 10 mg/kg body weight (Evans³ et. al; 1960) on an empty stomach. No other restriction was imposed on them.

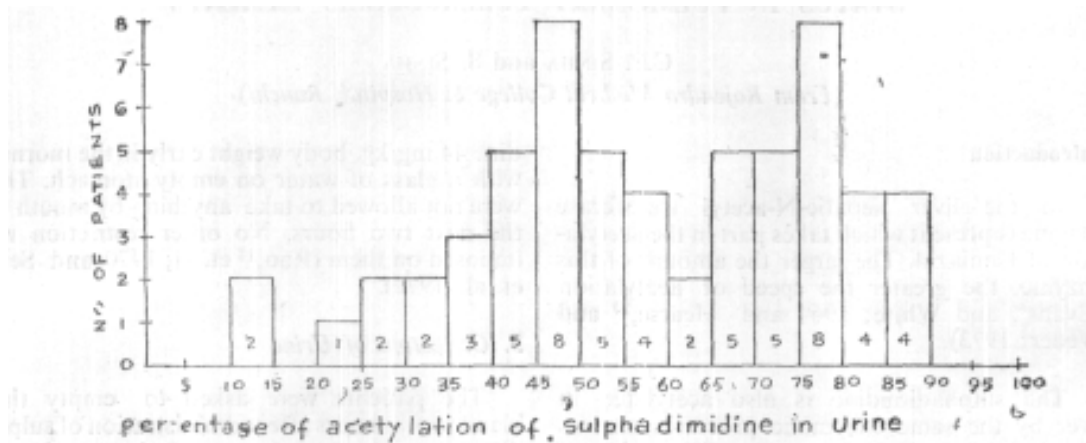
6. Collection of blood

10 ml. of venous blood was collected in heparinised vials after 6 hours of drug ingestion. The solid portion were allowed to settle and the supernatant fluid was transferred in clean centrifuge tube. The supernatant fluid was centrifuged for 5 minutes at a speed of 1000 rpm. The supernatant fluid was transferred thereafter in clean dry glass vessels and stored in deep freezing chamber of a refrigerator at -20°C .

7. Estimation of Free Isoniazid in Plasma

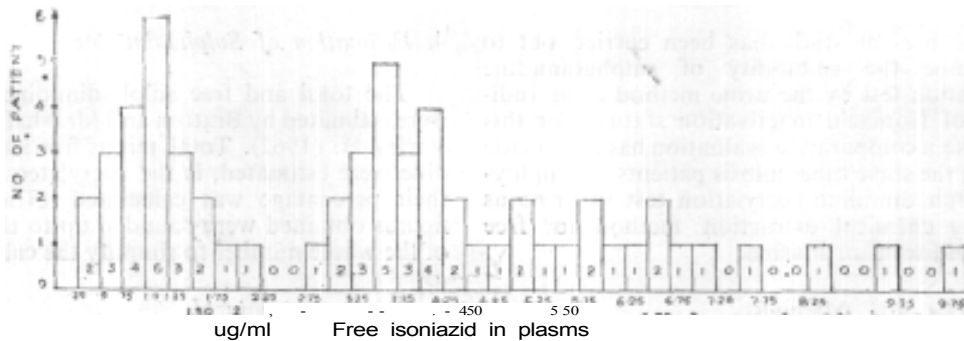
The free Isoniazid level in Plasma was estimated by Chemical extraction method of Rao¹⁵ et. al. (1971).

HISTOGRAM NO. 1



Histogram , showing percentage of acetylation in 60 subject of Pulmonary Tuberculosis in Urine

HISTOGRAM NO. 2



Histogram , showing free isoniazid µg/ml in Plasma of 60 subjects of Pulmonary Tuberculosis

Observation

In the 60 pulmonary tuberculosis patients percentage of acetylation of sulphadimidine in urine was estimated and plotted in histogram no. 2, which is bimodal in distribution. The discriminating point was observed in 65% of acetylation of sulphadimidine, which separated into two bimodal distribution

In the same 60 pulmonary tuberculosis patients free plasma Isoniazid level was determined and plotted in histogram No. 1 which is bimodal in distribution. The discriminating point was observed in 2.5 µg/ml, of free Isoniazid plasma level which separates into two unimodal distribution.

In the sulphadimidine acetylation test in urine 34 (57 %) patients out of 60, had 65 % or less of acetylation of Sulphadimidine in urine and they were classified as slow acetylators and

the rest 26 (43 %) out of 60 patients, had more than 65% of acetylation of sulphadimidine in urine and they were classified as rapid acetylators. In the isoniazid plasma level 22 (37%) out of 60 had free isoniazid plasma level of 2.5 (µg/ml. or less, and they were classified as rapid acetylators and 38 (63 %) out of 60 had free Isoniazid plasma level of more than 2.5 µg/ml. or less and they were classified as rapid acetylators and 38 (63%) out of 60 had free isoniazid Plasma level of more than 2.5 µg/ml. and they were classified as slow acetylators. In order to know the extent of association between the percentage of acetylation of sulphadimidine in urine and Isoniazid plasma level, the co-efficient correlation 'r' is work out and found to be (-1). The comparative value of estimate is shown in Table 1. Classification of the patients as slow and rapid inactivators obtained by both methods is shown in table 2.

Table 1

Shows in do pulmonary tuberculosis subjects distribution of slow and rapid acetylators by two different methods.

SI. No.	Item	Estimates of free Isoniazid in plasma.			Estimates of % of acetylation of sulphadimidine in urine;		
		Total no. of patients	Slow acetylator no. of patients.	Rapid acetylators no. of patients.	Total no. of patients.	Slow acetylators no. of patients.	Rapid acetylator no. of patients.
1	2	3	4	5	6	7	8
1.	No. of Patient	60	38	22	60	34	26
2.	Percentage	100	63	37	100	57	43
3.	Mean value	3.62 µg/ml	4.006 µg/ml.	1.13µtg/ml	59	45	77
4.	Standard deviation	2.2	2.01	0.86	19	11	7
5.	Standard Error of Mean	0.29	0.41	0.17	2.5	3	2

The co-efficient correlation $\mu = (-1)$

Table 2

Table 2 shows distribution of patients under two methods of estimations.

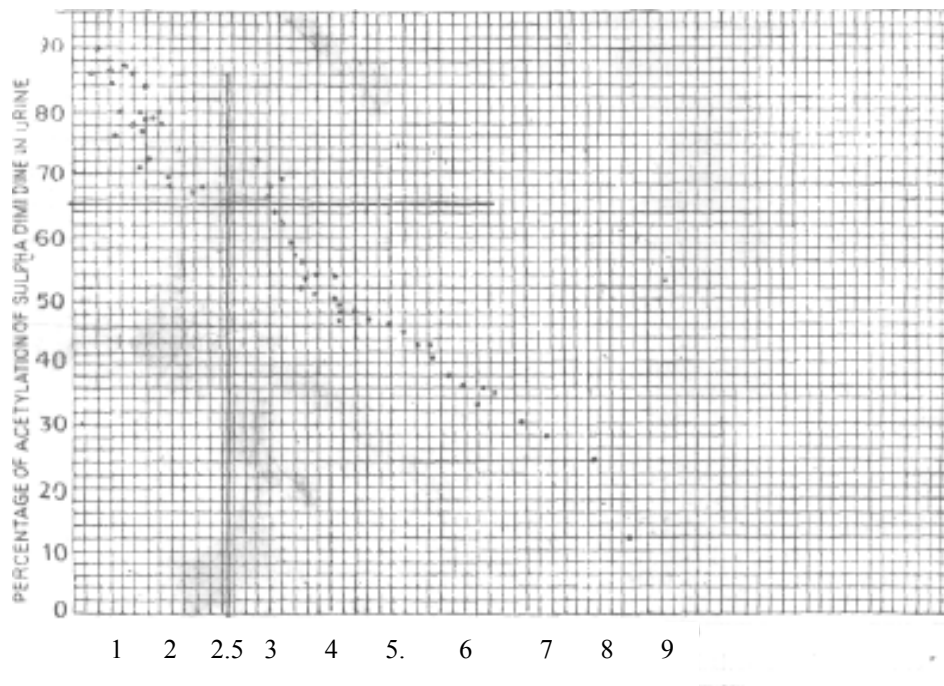
Sulphadiinidine acetylation test in urine	Isoniazid plasma concentration method.		Total no. of patient.
	No. of slow inactivators	No. of rapid inactivators.	
1	2	3	4
1. No. of slow inactivators	34	0	34
2. No. of Rapid inactivators.	4	22	26
Total	38	22	60

Misclassification of slow inactivators 7%.

Considering the merit of the sulphadimidine test in urine in comparison with the standard isoniazid plasma concentration method in classifying the patients as slow and rapid inactivators of isoniazid, it is seen that all the 22 patients classified as rapid inactivators by the isoniazid

plasma level test were also identified as such by the sulphadimidine test, an agreement of 100%. However agreement in respect of slow inactivator was obtained only in 34 (89%) out of 60 patients. Since 4 (11%) slow inactivators were misclassified as rapid inactivators. Considering

GRAPH NO. 1



the two groups together only 47%) out of 60 patients were misclassified.

As can be expected, low level of free Isoniazid in blood is associated with higher percentage of acetylation of sulphadimidine in urine and the same is evident from graph-1.

Discussion

The determination of Isoniazid acetylation (inactivation) status is of great therapeutic interest in once weekly therapy with isoniazid containing regimens. Subjects have been classified into two groups, slow and rapid acetylators. The rate of acetylation of sulphadimidine runs parallel to Isoniazid (Evans⁵ and White 1964 White²³ & Evans 1968, Evans⁶ 1969 and Weber and Brenner²² 1974). The present study is based on the presumption that sulphadimidine and Isoniazid acetylation in human body runs parallel (Evans⁵ & White 1964 and Weber²² and Brenner 1974).

In the present study 65% acetylation of sulphadimidine in urine is the discriminating point between slow and rapid acetylators according to histogram no. 2 and graph no. 1. The result of the present study confirms to White²³ and Evans (1968) Schroder^{16,17} (1972a and

1972b) Weber²² and Brenner (1974) and approximately conforming to Rao, et. al; (1970) British Medical Research Council (1973) but are dissimilar to Sen¹⁸ et. al. (1972). In the latter study, subjects have been divided in three groups Slow, Rapid and Intermediate group instead of the two, If slow and intermediate groups are clubbed together in the study of Sen¹⁸ et. al; (1972) their observations also become identical.

In the present study 2.5 µg/ml Isoniazid concentration in plasma is the discriminating point between slow and rapid acetylators according to histogram no. 1 and graph no. 1. Those having free Isoniazid concentration in plasma up to or less than 2.5 µg/ml are rapid acetylators and those having over 2.5 µg/ml in plasma are slow acetylators. These findings, too, are in close similarity with Evans⁵ et. al; (1960) Evans⁴ et. al; (1961) and Smith²⁰ and Kyi (1968). Indian patients suffering from pulmonary tuberculosis consist of 60 % Slow acetylators and 40% Rapid acetylators; Gangadharan, et. al; (1961a) and (1961b) Tuberculosis Chemotherapy Centre Madras²¹ (1973) Sharma¹⁹ et. al; 1974).

The present study on pulmonary tuberculosis subjects indicates that 57 % cases are slow acetylators and 43 % cases are rapid acetylators by sulphadimidine acetylation test in urine, and 63 %

cases are slow acetylators and 37% cases are rapid acetylators by estimation of free Isoniazid in plasma by chemical extraction method. The present finding is also marginally similar to Evans⁶ (1969) British Medical Council Research² (1973) and Viznerova²⁵ et al; (1973) on Pulmonary Tuberculosis by the sulphadimidine acetylation method and Evans,³ et al; (1960) Evans,⁴ (1961) Smith²⁰ and Kyi (1968) and Farhat⁷, et al; (1973) by the estimation of Isoniazid plasma level by different methods. A comparative study of sulphadimidine in urine and Isoniazid plasma level in sixty patients has revealed reverse correlation. The co-efficient correlation 'r' is (—1).

Sulphadimidine test detected all the (22) (100%) rapid in activators on the standard test and 34 (89 %) of slow inactivators. Considering all the 60 patients misclassification by the sulphadimidine acetylation test was only 4(7 %). The misclassification had been reported by Rao et. al; (1970) to be 1 % and Sen et. al; (1972) 9%.

Summary

Isoniazid inactivation status was compared in 60 pulmonary tuberculosis patients by sulphadimidine acetylation test in urine as well as free Isoniazid plasma level by chemical extraction method.

Out of 60 pulmonary tuberculosis patients 34 (57 %) were slow acetylators and 26 (43 %) were rapid acetylators by sulphadimidine acetylation test in urine. But 38 (63 %) were slow acetylators and 22 (37 %) are rapid acetylators by free Isoniazid plasma level determined by chemical extraction method.

Considering the merit of the sulphadimidine acetylation test in comparison with the standard Isoniazid plasamaconcentration method in classifying the subjects as slow and rapid inactivators of isoniazid, it was seen that all the 22 patients classified as rapid inactivators by the isoniazid plasma level test were also identified as such by the sulphadimidine test, and agreement of 100 %. However agreement in respect of slow inactivators was obtained only in 34 (89 %) out of 38 since 4 slow inactivators were misclassified as rapid inactivators. Considering the two groups together only 4 (7%) of 60 patients were misclassified.

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PROFILE OF GRANULOMATOUS HEPATIC TUBERCULOSIS IN INDIA

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Introduction

Tuberculosis in its varied forms is widely prevalent in tropics and still continues to be the leading cause of morbidity and mortality in the developing regions of the world. While diagnosis of pulmonary tuberculosis is easy to establish patients of abdominal and hepatic tuberculosis present mostly as a diagnostic problem. Paucity of literature on clinical and laboratory profile of hepatic tuberculosis from Indian subcontinent prompted us to record our experiences of 20 histologically proved cases of granulomatous hepatic tuberculosis diagnosed and treated during 1972 to 1976 out of total 57 cases of prolonged pyrexia and obscure hepatosplenomegaly.

Material and Methods

Patients attending medical out-patient department and gastroenterology clinic of A.I.I.M.S. Hospital, New Delhi and JIPMER Hospital, Pondicherry were taken up for the present communication. Thirty-two cases of prolonged pyrexia of uncertain origin and 25 cases of obscure hepatosplenomegaly without pyrexia were studied. Standard techniques were employed for routine haematological and biochemical investigations. (Dacei, 1958, Wootton and King, 1964). Postero-anterior skiagram of the chest was done in all the cases for the evidence of pulmonary tuberculosis. Mantoux test was done by using one and/or five tuberculin units. Induration of 10 mm. or more was considered as positive and less than 10 mm. as negative reaction.

Liver biopsy was carried out using Menghini aspiration needle/Vimsilverman biopsy needle (Sherlock, 1975). Sections were stained by haematoxyline losin and reticulin for detailed histological study. Barium contrast study was performed in all the cases to find out the presence and type of the intestinal tuberculosis. Of the total 57 cases initially taken up for the study definite histological diagnosis of granulomatous lesions suggestive of tuberculosis could be established in 20 cases. All the 20 were treated with antitubercular drugs namely streptomycin, thiacetazone and 40 mg. predinsolone. Patients were followed up in a speciality clinic for the assessment of the response of the therapy. Steroid was tapered after three weeks and was continued for 12 weeks.

Results

The age and sex distribution of the cases is

shown in table 1. The maximum number of cases 60 % were between the age groups of 21 -40 years. Males to females ratio was 3:1.

Table 1
Age and Sex Distribution (20 Cases)

Age group	Male	Female	Total	Percentage
13—20	3	1	4	20
21—30	7	2	9	45
31—40	2	1	3	15
41—50	2	—	2	10
51—60	2	—	2	10

The commonest presenting symptom was prolonged pyrexia 75 %, fever was continuous in 40% and remittent in 35%. Other symptoms according to their decreasing order were: loss of weight (70%), dull aching pain right upper abdomen (40 %), and loss of appetite (55 %) (Table 2).

Table 2
Symptomatology in Granulomatous Hepatic Tuberculous (20 cases)

Symptoms	No. of cases	Per cent
Prolonged fever	15	75
Continuous low grade	3	15
Intermittent	12	60
Loss of weight	14	70
Loss of appetite	11	55
Pain abdomen	8	40
Fullness in abdomen	7	35
Lump in right upper abdomen	5	25

Important physical finding was, variable

degree of hepatomegaly seen in (90%) cases. Liver was palpable 5 cms. below right coastal margin in 55 % cases. Hepatomegaly was more than 5 cms. in another 10% cases. In 25% cases liver was macronodular. Mild to moderate splenic enlargement was encountered in 40% cases and ascites was present in 25 % cases (Table-3).

Table 3

Physical Findings in Granulomatous Hepatic Tuberculosis (20 cases)

Findings	No. of cases	Percentage
A. General Examination		
Pallor	5	25
Oedema feet	3	15
Lymphadenopathy	2	10
Glossitis	1	5
Clubbing	2	10
Temperature	11	55
B. Abdominal Examination		
Palpable liver	18	90
Upto 5cms	11	55
>5cms	2	10
Macronodular liver	5	25
Splenomegaly	8	40
Mild ascites	5	25
C. Associated illness		
Ostearticular tuberculosis	2	10
Healed Pulmonary	3	15

Mantoux test was positive in 11 cases. Ulcerative reaction was observed in another 3 cases. It was interesting to record negative mantoux in 6 cases with tuberculous granuloma of the liver. Posteroanterior skiagram of the chest showed healed pulmonary tuberculosis in 3 cases. Radio-

logical evidences of active osteoarticular lesions suggestive of tuberculosis was recorded in 2 cases. Barium meal follow through study was done in 12 cases which revealed no evidence of gastrointestinal and ileocaecal tuberculosis.

Histopathological study of the liver showed multiple confluent caseating granulomas in 17 cases and non-caseating discrete granulomas in the remaining 3 cases. Other morphological findings were: langhans giant cells, mild portal fibrosis and focal necrosis. Biochemical profile showed raised serum alkaline phosphatase in all cases and enzyme values over 30 KAU in 45% cases. Serum bilirubin of more than 2 mg. % was present in only 3 cases. Mild elevation of SGOT was present in 2 cases. Hypoalbuminaemia (<2.7 grams. %) was noted in 60% cases. Prothrombin time was prolonged in 60% cases (Table 4).

Table 4

Liver Functions in Granulomatous Hepatic Tuberculosis (20 cases)

Findings	No. of cases	percentage
Elevated Alk. Po4	20	100
<30K.A.U.	11	55
>30 K.A.U.	9	45
Serum albumin (<2.7 grams%)	12	60
Serum bilirubin (>2.0 grams%)	3	15
Raised SGOT (N=20IU) (>20IU)	2	10
Prolonged prothrombin time	12	60

Discussion

Tuberculosis is a disease with protean manifestations and is widely prevalent all over the tropics including Indian subcontinent (Rao, 1972). Although classical forms of pulmonary and intestinal disease are widely recognised and accepted, clinical and laboratory profile of hepatic tuberculosis is still not well understood (Hughes and Fox, 1972). Hence tuberculosis of the liver is rarely considered in the differential diagnosis of hepatic disorders in day to day clinical practice. Results of the present study indicate that significant proportion of cases of pyrexia of unknown origin may be due to isolated hepatic tuberculosis or hepatic involvement in miliary tuberculosis.

Clinical features of granulomatous hepatic tuberculosis observed in the present series are: pyrexia of unknown origin, variable degree of hepato-splenomegaly along with constitutional symptoms like loss of weight, appetite and anaemia. Jaundice is rare. These findings suggest that prolonged pyrexia and hepatomegaly are important findings in hepatic tuberculosis. Conditions simulating the clinical features of tuberculosis of the liver are: anicteric viral hepatitis, collagen vascular disorders and hepatic involvement in systemic infections including enteric fever. However, definitive diagnosis of hepatic tuberculosis only on clinical and biochemical findings is difficult because of the close similarity in clinical and laboratory profile of various disorders mentioned above. Marked anorexia and high elevation of serum transaminase levels favours the diagnosis of anicteric viral hepatitis (Schiff, 1960). Multi-systemic involvement like polyarthrititis, endocarditis, fever and hepatomegaly are diagnostic features of collagen vascular disease like systemic lupus erythemia. Similarly past history of dysentery, raised and restricted movement of right dome, demonstration of *E. histolytica* in the stool or aspirate and positive indirect haemagglutination test (I.H.A.) for amoebiasis confirm the diagnosis of hepatic amoebiasis. Positive blood culture for *Salmonella* and high titres widal reaction are diagnostic of typhoid fever. However, most important single parameter for the diagnosis is liver biopsy. Authors believe that paucity of literature on granulomatous hepatic tuberculosis from Indian subcontinent is due to the fear of doing liver biopsy in acutely ill and feeble patients. These findings indicate that prolonged pyrexia and hepatomegaly in an anicteric patient suggest the possibility of tuberculous granuloma of the liver.

When patients of hepatic tuberculosis have macro-nodular liver and jaundice, it poses diagnostic problem: Important conditions which needs consideration are: macro-nodular cirrhosis of liver, hepatoma and metastatic malignancy of the liver, hepatic infiltration in leukaemias and lymphomas. Associated symptoms, biochemical and haematological findings may be helpful in arriving at definite diagnosis but liver biopsy is essential for histopathological diagnosis. Hence liver biopsy is very useful diagnostic procedure in patients presenting with prolonged fever, hepatomegaly with or without icterus for confirmation of diagnosis and rational therapy. Other laboratory parameters helpful in the diagnosis of hepatic tuberculosis are: high E.S.R., strongly positive montoux reaction, raised alkaline phosphatase due to focal liver disease. However, these parameters are not adequate for the diagnosis

and need to be supported by histopathological findings. Rarity of hyperbilirubinaemia and elevation of serum transaminase levels are worthnoting. These observations are comparable with previous series (Wig et. al. 1972, Simon and Wolff, 1973; Mir-madjlessi and Farmer, 1973).

Granulomatous hepatic lesions have been reported in conditions other than tuberculosis e.g., sarcoidosis, leprosy, syphilis, infectious mononucleosis and fungal infection etc. (Simon and Wolff, 1973). Absence of associated clinical and laboratory evidence typical of above illness almost excludes these possibilities in the present series. Presence of multiple, caseating, confluent granulomas on liver biopsy in 17 cases was diagnostic of granulomatous hepatic tuberculosis. Non-caseating granulomas in 3 cases who had healed pulmonary lesions are highly suggestive of tuberculous granuloma. Other histological findings besides granuloma observed in the present series are: langhans giant cells, epithelioid cells, mild non-specific hepato-cellular reactions and variable degree of portal fibrosis.

Summary

Clinical, biochemical and histological profile of twenty cases of granulomatous hepatic tuberculosis are presented. Pyrexia of unknown origin and variable degree of hepatomegaly were the main features observed in 75% and 90% cases. Clinical jaundice was rare and was present in only 3 cases. Other findings noted were: loss of weight 70%, appetite (55%) anaemia (25%) and protein malnutrition (60%). Liver biopsy is the most useful procedure for confirmation of the diagnosis of granulomatous hepatic tuberculosis and rational therapy.

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EFFECT OF ASPIRIN IN THE CONTROL OF HYPERURICEMIA AND ARTHRALGIA DUE TO PYRAZINAMIDE (PZA) THERAPY

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PZA has been found to be an invaluable drug in the treatment of Tuberculosis. But many patients on PZA therapy often complained of pain in various joints. Even though studies about arthralgia and hyperuricemia due to PZA therapy are reported by Cullen et. al. 1957, Shapiro et. al. 1957, Petty T.L et. al. 1964, a study in Indian patients was considered overdue by us. It is worthwhile to note here that Kass has reported death following PZA induced hyperuricemic nephropathy.

Materials and Methods

The study was carried out at the Sanatorium for Chest Diseases, Pulayanarkotta, Trivandrum. All the patients in the hospital receiving PZA as a part of treatment for at least one month (range 2-6 months) were selected for the study. Those who had at any time prior to PZA therapy any joint complaint were excluded from the study. Thus 45 patients qualified for the study, 23 males and 22 females. Their age varied from 16 to 60 years. All of them were subjected to thorough clinical examinations. Routine Urine, stool and hematological examinations were carried out. Radiological examination of the joints was carried out in all those who complained of joint pains. Serum uric acid was estimated in all the patients. This constituted the first part of the study.

In the second part, these patients were divided into two groups at random, 22 in Group 'A' and 23 in Group 'B'. Acetyl Salicylic acid in a dose of 600 mg 3 times daily was added to the regimen of Group 'A' patients, while PZA was continued. In Group 'B', PZA was discontinued. Both groups were observed for a period of 15 days, at the end of which serum uric acid was estimated in all the patients belonging to either group.

It may be mentioned here that a serum uric acid level of 6.5 mg% and above was considered as hyperuricemia (Leading article BMJ—7th Oct. 72).

Results — Part I.

All the patients showed a serum uric acid level above 6.5 mg%. Of the total 45 patients studied, 30 showed arthralgia of one or more joints. The severity of the pain varied. 7 patients complained of severe pain in the involved joint.

In 8 patients arthralgia was associated with painful restriction of movement of the joint. There was no direct relationship between the level of serum uric acid and the degree of arthralgia. Duration of PZA therapy did not seem to influence the level of serum uric acid or the onset of symptoms. Occurrence of arthralgia was not found to be influenced by the age of the patient. Excepting two patients, none had any significant joint swelling. Of the 30 patients developing arthralgia 16 were males and 14 females showing that both sexes are involved to the same extent. X-ray of involved joints did not show any radiological changes in any case.

Part II

All the patients in Group 'A' who were given aspirin while continuing PZA and all patients in Group 'B' in whom PZA was discontinued showed a normal serum uric acid level at the end of 15 days except in two patients belonging to Group 'B' who showed a serum uric acid level of 6.5 mg which is considered as the upper limit of normal. Joint pains had completely disappeared within one week in all patients.

Discussion

Hyper uricemia is a common finding in patients treated with PZA (Shapiro et.al. 1957) Hyperuricemia results from decreased renal excretion of urate (Gleason et.al. 1957 Cullen et al 1957). This can be due to increased tubular reabsorption rate (Gleason et al 1957) or due to decreased tubular secretion rate (Gutman et. al. 1959). We found hyperuricemia to be a constant accompaniment of PZA therapy.

The mean serum uric acid level in general population is 4-4.5 mg%. 9% males and 3.5% females have serum uric acid levels above the arbitrary upper level of normal of 6.5mg%. (BMJ — Leading article 7th Oct. 1972). In our study the level of serum uric acid returned to normal in almost all patients in whom PZA was stopped, showing that in none of these the serum uric acid was elevated normally. Relatively higher proportion of patients on PZA therapy complained of arthralgia as against the observation in "Modern Drug treatment of Tuberculosis" (A publication of Chest and Heart Association — London) which states this symptom to be rare.

Both PAS and aspirin may prevent PZA hyperuricemia (Shapiro et. al. 1957). But Acetyl Salicylic acid becomes uricosuric in daily doses of more than 5 grams a day. (Madan Lal, 1976) and in smaller doses it has a urate retaining action. But we found that in daily doses of 1.8 gms it very effectively brings down PZA induced hyperuricemia. Similar results were observed by Petty et.al.(1964) who found that Acetyl Salicylic acid in daily doses of 2.4 gms daily effectively brings down PZA induced hyperuricemia and arthralgia.

Summary and Conclusion

45 patients on Pyrazinamide therapy were studied. All of them showed an increased serum uric acid level. Thirty of them had arthralgia of varying intensity affecting one or more joints. Radiography of the involved joints showed no abnormality in any patient. On withdrawal of PZA or addition of 1.8 gm aspirin daily, uric acid level returned to normal and arthralgia disappeared in 15 days.

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AN EVIDENCE IN FAVOUR OF TUBERCULOUS ETIOLOGY OF EALES DISEASE

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Introduction

Bales disease is characterised by repeated intraocular haemorrhage in young males leading to gross visual impairment and finally loss of vision. The pathogenesis of this disease, which shows low grade grumbling inflammatory reaction with proliferative and degenerative changes, is still an obscurity, though it is generally agreed that retinal vasculitis or periphlebitis is the initial lesion. A widely held belief was that the disease is the result of tissue allergy to tuberculo-proteins (Elliot, 1959). This presupposes the existence of tuberculous infection in the body which again is likely to be associated with blood and tissue changes characteristic of the tuberculous infection.

The present investigation shows that changes in the pattern of serum proteins in patients suffering from Bales disease are identical with those observed in tuberculosis.

Material and Methods

Forty one cases of spontaneous vitreous haemorrhage due to venous inflammation, of less than 3 months duration, diagnosed as Bales disease were referred to the Tuberculosis and Chest Out-patient department from the Institute of Ophthalmology, J. N. Medical College, Aligarh, for investigating any evidence of associated tuberculous lesion. After recording detailed history and thorough physical examination, the

patients were subjected to a battery of tests including haematological (T.L.C., D.L.C., E.S.R., Hb%), sputum, urine, stool examination, Mantoux test with 1 T.U., P.P.D. R.T. XXII and x-ray of the chest. Associated tuberculosis was detected in two cases only and they were excluded from the present study. Rest of the patients were otherwise healthy and had no associated metabolic disorder, hypertension or any known cause that could be responsible for diminution of vision. The serum of these patients was subjected to electrophoretic study for proteins.

Five ml. blood was collected from each patient in a clean glass vial, and the serum was separated and stored in the refrigerator. Protein fractions in the serum were studied by electrophoretic separation on agar gel using barbitone buffer (pH 8.4) and D.C. current of 120 volts. The electrophoresis was stopped after one and half hour. The slides were fixed with alcohol and stained with amido-shwartz. The stained slides were analysed using a densitometer and the readings were plotted on a graph. The areas occupied by individual fractions were obtained from the graph and the corresponding standard area was calculated.

Results

Table I shows the distribution of cases of Bales disease according to age, sex, family or

Table 1

Showing distribution of patients of Bales disease according to age, sex, family contact history and B.C.G. Vaccination

Age in years	No. of Cases	Sex		Family/Contact h/o. tuberculosis	B.C.G
		M	F		
10—20	11	11	—	2	—
21—30	15	15	—	2	1
31—40	10	10	—	—	—
41—50	3	2	1	—	—
Total	39	38	1	4	1

contact history of tuberculosis and of earlier B.C.G. vaccination. None of the patients in the present series was below 10 years in age. The majority of the patients belonged to age group 21 to 30 years and only 3 were above 40 years of age in the present study. Only one patient out of 39 included in the present study was female. One patient had prior B.C.G. vaccination and 4 had family or contact history of pulmonary tuberculosis.

Table II shows tuberculin status of patients of Bales disease. It is evident from the table that 28% cases were tuberculin negative (<10 mm) and rest 72% were tuberculin positive.

Table 2
Showing the size of the tuberculin reaction in 'cases of Eales disease.

Size of tuberculin reaction in mm. (induration)	Patients	
	Number	Percentage
0—4	9	23
5—9	2	5
10—14	11	28
15—20	8	21
Above 20	9	23

Table III shows distribution of cases according to socio-economic status. Sixty-one and 34 % patients respectively belonged to very poor and poor categories. Only five per cent were from families having per capita income of Rs. 80 or above p.m.

Plasma protein pattern of 20 normal individuals who served as controls and 39 patients of Eales disease are summarized in Table IV.

Total serum proteins showed no deviation from the control. However, there was a significant decrease in the level of albumin and a corresponding increase in globulin fraction. The albumin/globulin ratio was changed from 1 : 1.26 in the controls to 1 : 0.43 in the patients of Eales disease. Amongst the various fractions of globulin, α_1 showed no significant difference as compared to the control. On the other hand, α_2 , β and γ -fractions significantly increased over the control values. The albumin/ α_2 ratio was

Table 3

Showing distribution of cases of Eales disease according to their socio-economic status.

Per capita income Rs. per month	Patients	
	Number	Percentage
Very poor		
Below 40	24	61
Poor	13	34
40-79		
Lower middle class	1	2.5
80-129		
Upper middle class	1	2.5
130-269		

markedly decreased from 7.1 in the control group to 1.9 in the disease group.

Values of ESR, T.L.C., D.L.C., Hb% were not different in patients of Eales disease from those observed in the control group.

Discussion

From the values of total serum proteins and their fractions in normal persons (controls) and in those suffering from Eales disease (Table IV) the following observations are made:-

(a) There was no significant change in total serum protein level, (b) The serum albumin was significantly lowered and serum globulin was increased in patients of Eales disease, (c) Amongst the fractions of globulins, α_1 showed no change while α_2 , β and γ -fractions were significantly increased, (d) Albumin: globulin ratio was increased and Albumin/ α_2 ratio was decreased. These changes exhibit a similarity with those reported in patients suffering from manifest tuberculosis (Seibert et. al. 1947; Baldwin and Hand 1953, Gilliland et. al. 1956; Khanijo 1964; Kavinde et. al. 1964; Johnson et. al. 1967).

The fall in serum albumin may result from deficient intake, excessive loss or lack of synthesis. There is evidence to suggest that impairment of protein synthesis is associated in tuberculosis due to lowering of enzyme activity, besides accompanying tissue destruction, which may manifest in lower albumin levels (Kanhere and Rao, 1967). It is difficult to assign lower albumin

Table IV

Showing mean α_2 -values of serum proteins and albumin/ α_2 ratio in the present series of Bales disease as compared to figures reported in tuberculosis by other workers.

Series	Total Proteins	Albumin	Globulins	Globulin Fractions				Ratio -Albumin/ α_2
				α_1	α_2	β	γ	
Present study								
Control (20)	7.56 ± 0.08	4.23 ± 0.05	3.33 ± 0.046	0.28 ± 0.08	0.59 ± 0.028	0.87 ± 0.036	1.58 ± 0.048	7.1
Bales disease (39)	7.59 ± 0.14	2.36* ± 0.1	5.23* ± 0.12	0.25 ± 0.028	1.12* ± 0.076	1.33* ± 0.069	2.24* ± 0.093	1.9
Gilliland et. al. (1956)								
Control (28)	7.27	4.15	3.11	0.29	0.59	0.83	1.40	7.06
Pul. Tub. (60)	7.60	3.57	4.03	0.39	0.81	1.02	1.81	4.53
Kanhare and Rao (1963)								
Control (6)	7.10	3.56	2.54	0.36	0.60	0.93	1.65	5.93
Skeletal Tub. (10)	5.99	1.86	4.13	0.45	0.89	0.97	1.82	2.09
Khanajo and Khanajo (1964)								
Control (16)	7.49	4.11	3.43	0.41	0.75	0.89	1.38	5.42
Pul. Tub. (15)	7.39	3.34	4.03	0.55	0.95	0.82	1.71	3.52
Kavindeet. al. (1964)								
Control (32)	7.50	4.35	3.15	0.35	0.61	0.79	1.40	7.13
Extra Pul. Tub. (80)	6.85	3.08	3.79	0.44	0.69	0.79	1.87	4.75

*p = <0.001

Figures in brackets indicates number of cases.

levels in Bales disease to any of the above factors as the patients were seen in good general condition and tissue destruction in this disease is so minimal that it is unlikely to affect the albumin level to such an extent.

In spite of lowered serum albumin total protein level is maintained by a corresponding increase in the level of globulins resulting in the reversal of albumin/globulin ratio.

The α_1 globulin fraction was normal in patients with Bales disease. Normal levels have been reported in tuberculosis as well as where the disease is minimal (Chievitz and Thide 1960; Kanhere and Rao 1962). Increase in α_1 globulin in tuberculosis has been shown to be associated with fever, extent of disease and low body weight (Chievitz and Thide 1960; Johnson et. al. 1967). It may be argued that Bales disease is only a localized manifestation resulting from the allergy to tuberculo-proteins (Kavinde et. al 1964) and does not affect the general health of the individual.

Increase in α_2 globulin is less sensitive to the extent of the disease or the weight of the patient (Johnson et. al. 1967). However, rise in α_a globulin fraction exhibits a correlation with sensitization of the patient to the protein of tuberculin (Kavinde et. al. 1964). This sensitization is manifest in all forms of tuberculosis resulting in increased levels of this fraction (Table IV). In Bales disease the hypersensitive reaction of peripheral retinal vein walls to tuberculo-proteins has been considered as a cause of vitreous haemorrhage (Elliot, 1959). It may be argued that the presence of antibodies against tuberculo-proteins may be responsible for the increase in α_2 globulin in Bales disease.

β -globulins are known to be increased in patients with lesser extent of the disease and in early tuberculosis (Barua, 1972). Its level declines with increase in the extent or the duration of the disease (Johnson et. al. 1967). Significant increase in the levels of β -globulins were observed in the patients of Bales disease. Since all the patients included in the present study presented themselves

early in disease it may be argued that the two conditions viz. early tuberculosis and Eales disease, having similar effect on β -globulins have something in common which triggers the rise in their level. Conclusion can be drawn that allergy is responsible atleast in part for this rise in β -globulins.

Increase in the level of γ -globulins supposedly represents activity (Volk et. al. 1953), chronicity (Maheret. al. 1957), or caseation (Virgilio and Anzano 1958). Bulk of the increase in γ globulin, undoubtedly, does not represent antibodies, but rather a nonspecific reactivity of the reticulo-endothelial system (Baldwin and Hand 1953; Schroeder 1960). Significant increase in the level of γ globulins observed in the present study is similar to that observed by some workers in patients suffering from various forms of tuberculosis (Table IV).

Reduction of albumin/ α_2 ratio has been regarded proportional to the activity and extent of the tuberculous disease and is considered as a better index for the assessment of a case than other conventional laboratory procedures (Kavinde et. al. 1964). The change in albumin/ α_2 ratio in Eales disease have been found to be similar to what have been reported in various forms of tuberculosis by different workers (Table IV). It is difficult to explain satisfactorily the changes of such great magnitude in such a localized condition. It will be premature to draw any conclusion regarding the etiology of Eales disease on the basis of similarity in the response of Albumin/ α_2 ratio.

Summary

The changes observed in serum proteins and their fractions in patients of Eales disease exhibit similarity with those observed in manifest tuberculosis by various workers. There was no change in total proteins while albumin level decreased and globulins increased thus resulting in the reversal of albumin/globulin ratio. Amongst the fractions of globulins α_1 remain unchanged while α_2 , β and γ -fractions increased significantly. Albumin/ α_2 ratio was also decreased.

The prevalence of Eales disease was found more in persons belonging to poor economic status, amongst males and in younger age group. Although many common factors are observed in tuberculosis and Eales disease but more evidence is required to assign it a tuberculous etiology.

Acknowledgements

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Ind. J. Tub., Vol. XXV, No. 4

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CASE REPORTS

ATLANTO-AXIAL DISLOCATION ASSOCIATED WITH SUBOCCIPITAL COLD ABSCESS

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Craniovertebral region is an uncommon site of tubercular involvement. (Tuli, 1974; Massalawala, 1973; Nicholson, 1956; Fung et. al. 1962). When the lesion occurs in the odontoid process or the transverse ligament of the atlas, it is easy to explain the associated atlanto-axial dislocation (Pandya, 1971; Osborn, 1874). The present case is an unusual case of atlanto-axial dislocation with a tubercular cold abscess in the suboccipital region behind atlanto-axial membrane and the posterior arch of atlas.

Case Report. A 22-year old female (Hosp. No. 10229) was admitted to orthopaedic unit of S.S. Hospital, Banaras Hindu University on 29.6.76. Two months prior to admission, she developed pain in neck which used to be increased during neck movements. Fifteen days back she started with tingling and numbness in left upper limb followed soon after by weakness which rapidly progressed to involve all the four limbs. She developed overflow incontinence and severe constipation. There was no definite history of trauma, upper respiratory infection, diffuse joint pain or tuberculosis. On examination, she was of average built and mildly anaemic. She had no lymphadenopathy. Cardiovascular and respiratory system were normal. She had torticollis and painful limitation of neck movements. Upper cervical spine was tender. Cranial nerves were normal. She had only grade 2 power in all four limbs with increased tone and brisk deep tendon reflexes. Abdominal reflexes were absent. Plantar response was upgoing on both sides. She had overflow incontinence. Investigations revealed E. SR-51 mm first hour; total W.B.C. 10500 per cu.mm; differential leucocyte count normal; haemoglobin-12.5 gm%; blood sugar 85 gms% (Fasting); blood urea 25 mg%. Plain x-ray of cervical spine (Fig. 1) revealed marked degree of atlanto-axial dislocation.

She was given skull traction and referred to neurosurgery. After continuous skull traction with increasing weight upto 12 lbs., she showed excellent neurological recovery. Motor power returned to grade 4. Operation was done on 23.8.76 with the aim to fuse the craniospinal region. Under continuous skull traction, patient was anaesthetised and bone grafts taken from ileac crest. Then she was put in prone position. Through a midline incision extending from occi-

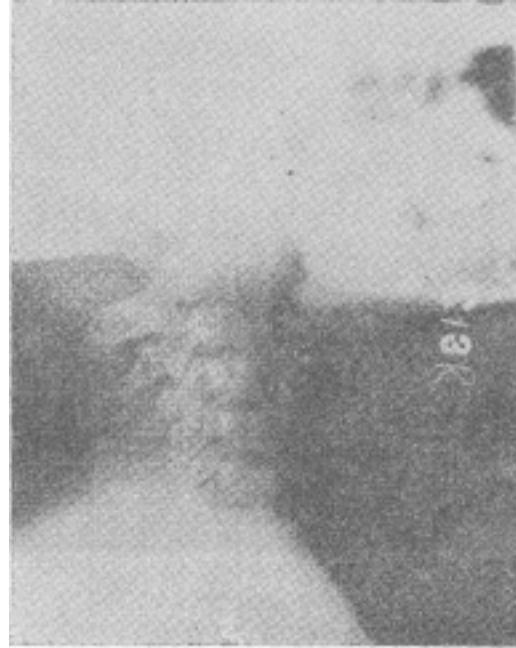


FIG. 1

Preoperative x-ray of craniocervical region (lateral) showing marked atlanto-axial dislocation.

pital protuberance to midcervical region, suboccipital region was exposed. While approaching the posterior lip of foramen magnum and posterior arch of atlas. 5 to 10 ml of thick caseous material came out. After removing the caseous material, the atlanto-axial fusion done by modified Gallic method. Two burr holes were made in the occipital bone. With the help of stainless steel wire (No. 22) which passed through the burr holes and around C2 spine, fixation was done and bone grafts were laid down and wound closed in layers. (Fig. 2). Histology confirmed the caseous material to be tubercular.

Post-operatively, traction was maintained for a week and anti-tubercular treatment started. After removal of stitches, a four poster collar was given and patient was helped to walk. Three months after she is in excellent health without neurological deficit.



FIG. 2

Postoperative x-ray after craniospinal fusion.

Discussion

Cranio-vertebral tuberculosis is a relatively uncommon condition. Tuli (1974) reported 25 cases of cranio-vertebral tuberculosis of which 14 had atlanto-axial dislocation/subluxation and 21 had significant prevertebral shadow. Other reported series (Pandya, 1971; Massalawala, 1973; Bosworth et al. 1953) have mentioned presence of prevertebral shadow associated with atlanto-axial dislocation which indicates tubercular aetiology. Such dislocation produce marked neurological symptoms and even sudden death (Osborn, 1874). However, in none of the reported series, suboccipital tubercular cold abscess associated with atlanto-axial dislocation have been reported. Without any positive history of tuberculosis and absence of prevertebral shadow or erosion of odontoid, the diagnosis of congenital atlanto-axial dislocation was considered preoperatively. In this case, the atlanto-axial dislocation is possibly due to hyperaemic condition produced by the inflammatory process as has been reported by Watson Jones (1932).

Ind. J. Tub., Vol. XXV, No. 4



FIG. 3

Postoperative photograph of the patient.

Summary

A case of atlanto-axial dislocation with quadraparesis, diagnosed preoperatively as cranio-vertebral anomaly, was found to have a tubercular cold abscess in the suboccipital region. Literature on cranio-vertebral tuberculosis reveals such presentation unusual.

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SPONTANEOUS TENSION PNEUMOMEDIASTINUM

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An infrequent but grave medical emergency is that of tension pneumomediastinum which is defined as presence of free air in the mediastinum. Laennec (1819) was the first to recognise this condition. In recent years mediastinal emphysema has been recognised with increasing frequency principally in healthy young men and parturient women and is found most frequently without an apparent precipitating cause (Munsell 1967).

Spontaneous tension pneumomediastinum complicating pulmonary tuberculosis is described rarely (Hurrell 1941, Class & Pascheco 1965) Spontaneous tension pneumomediastinum complicating miliary tuberculosis has been reported by Bodey 1961 and P. Ravindram et. al., 1975. We are also reporting one case of tension pneumomediastinum complicating miliary tuberculosis.

Case Report

G.D. 55 Years old male was admitted in Nehru Hospital, Medical College Gorakhpur with history of dry cough, low grade fever and marked loss of weight for six months. There was no definite history of contact. On examination patient was anaemic, thin built without any evidence of clubbing or dyspnoea. Lungs were clear. Examination of other systems did not show any abnormality. Sputum examination by smear proved negative for acid fast bacillus. Mantoux test was negative (7 m.m.). E.S.R. was 69 m.m. in 1st hour (Wintrobe). Total W.B.C. count was 9864/c.m.m. with moderate lymphocytosis. Fundus revealed miliary tubercles. Skiagram Chest showed presence of miliary mottling in both lung fields in all Zones, more confluent in left upper zone (Fig. 1). The patient was put on antitubercular drugs, Streptomycin, Isonex, P.A.S. in standard doses and prednisolone 20 Mg. daily. After five days of therapy he complained of severe retrosternal pain. On examination, subcutaneous emphysema was observed at the root of neck, down the Chest wall and the arms (Fig. 2). On auscultation crackling sound was heard over the precordium near the left border of sternum (Hamman's Sign). On both the sides breath sounds were normal. Skiagram chest confirmed the diagnosis of pneumomediastinum. Analgesics and intermittent oxygen inhalation were given in addition to antitubercular treatment.

Ind. J. Tub., Vol. XXV, No. 4

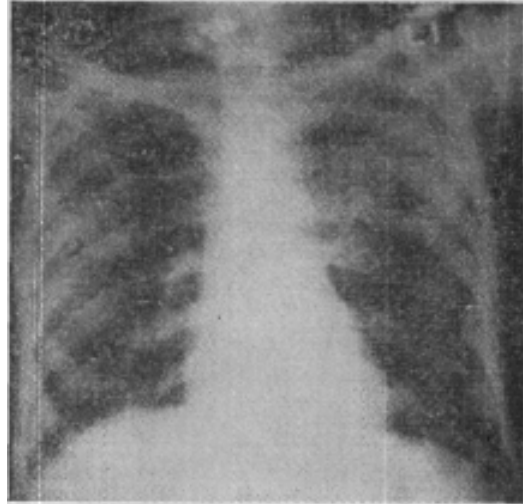


FIG. 1

Skiagram chest P.A. view showing miliary mottling in both lung fields which are more confluent in left upper zone.

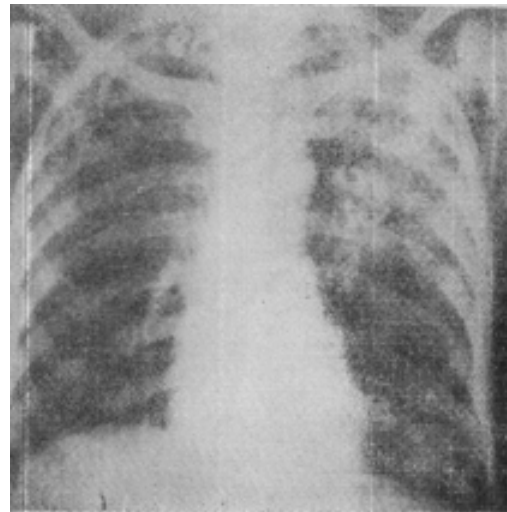


FIG. 2

Skiagram chest showing miliary mottling with confluent lesion in left upper zone and air strip in mediastinum on left side with subcutaneous emphysema in both axillae.

Discussion

Air may gain access to the mediastinum via four pathways, (a) From the exterior by extension along the fascial plane of the neck, (b) Through perforation of the oesophagus, trachea and bronchus, (c) By dissection along the retroperitoneal space, (d) By way of interstitial tissue of lungs. The last one is the commonest. The cause of alveolar rupture is an extreme rise in intra-alveolar pressure due to trapping of air.

Macklin and Macklin (1944) classified alveoli into marginal and partitional. (a) Marginal—which are related to perivascular sheath and have no intercommunicating pores, (b) Partitional—which represent all other alveoli and do possess intercommunicating pores. Rupture of marginal alveoli results in escape of air in perivascular sheath. The air leak from alveolar rupture is propelled towards the hilum along the vascular sheath by compressing action of respiratory muscles. The air now builds up tension in mediastinum and the air may track up along the fascial plane or down to abdomen along the fascial sheath of aorta. It may rupture mediastinal pleura leading to pneumothorax.

Our case had mediastinal emphysema with subcutaneous emphysema at the root of neck and down the chest wall and in the arms.

Although in recent years pneumomediastinum has been recognised with increasing frequency particularly in Children (Jewett 1962, Ibrahim 1964) occurrence in association with pulmonary tuberculosis is rare. Four cases of pneumomediastinum with miliary tuberculosis have been reported.

Summary

One case of spontaneous tension pneumo-

mediastinum complicating acute haematogenous miliary tuberculosis is described. The mechanism of production of pneumomediastinum is briefly stated.

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TUBERCULOUS OSTEOMYELITIS OF THE SKULL

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Introduction

Tuberculous osteomyelitis of the skull bones is fairly rare. The incidence of calvarial tuberculosis is probably less than 0.5 % of all types of bone diseases. Even though tuberculosis is so common in India, there are very few recorded cases of skull bone involvement.

Case Report

R.K., a 12 years old girl was admitted with us in early June 1977 with a soft fluctuant swelling over the scalp, almost over the region of the anterior fontanelle.

According to the history, she had that swelling for 3 months and had been having headache and fever off and on.

She had been treated for persistent cough and fever in March 1977 and since there was no response to routine antibiotics, she had been put on anti-tuberculous drugs. Unfortunately she developed reaction to Streptomycin and PAS and so was only on Isoniazid.

On examination, she was an average female child, with a soft, fluctuant swelling over the scalp. The swelling was slightly tender and there was a palpable defect in the underlying bone measuring about 3 cms x 4 cms. There was no lymphadenopathy. Clinically, the chest was normal.

Blood count and urine examination were normal. The erythrocyte sedimentation rate was 96 cm. for the 1st hour (Westergren). Skull x-ray revealed a circumscribed, osteolytic lesion in the fronto-parietal region (Fig. 1). Chest x-ray was clear.

The patient was operated upon on 13.6.77. On exploration it was seen that both tables of the bone were destroyed and there was extensive yellowish caseous matter, extending into the epidural space. The dura matter was intact. Most of the necrotic tissue was removed and the bone edges debrided.

The biopsy material consisted of irregular bits of soft tissue, which on histopathological examination, revealed areas of caseous necrosis surrounded by epithelioid and giant cells with lymphocytic reaction. The microscopic diagnosis was tuberculosis.

The patient was asymptomatic for 1 month after surgery, when she returned with persistent discharge from the wound. At re-exploration, some more necrotic dead bone was removed. This time the ESR was 126 mm. in 1st hour and so the patient was put on Ethambutol in addition to Isoniazid.

At regular follow up, she has shown good response. Her E.S.R. has come down to 60 mm. in the 1st hour. The wound is clean and granulating and she is symptom free.

Discussion

Tuberculosis of the vault of the skull is an uncommon condition. Even in a country like India, where tuberculosis is so common, involvement of the calvarium has been reported in not more than 5 cases.

The incidence of cranial involvement in all form of tuberculosis varies and has been reported as varying from 0.2% to 1.3% (Straus). In common with tuberculosis of bones and joints, lesions in the skull are almost never primary, unless there has been direct inoculation of the bone by a penetrating injury. Such a route of infection must be very uncommon but Straus has recorded a number of cases where no other lesion was found. In almost all cases, a primary lesion elsewhere in the body — most commonly in the lung — can be shown.

The majority of these cases occur at an early age. It has been reported by Straus in 1933 that 50% of all cases occur before the age of 10 and 80 % before the age of 20. Recent literature also seems to validate these report. There is no sex predilection and both sexes are almost equally affected.

Trauma has been suggested as playing a role in the genesis of this disease. The degree of importance that should be attached to this is doubtful, as the disease has a fairly long incubation period, and the condition does not present for a considerable time after the onset. Trauma, by increasing the local vascularity, may help in localising the lesion, to a particular part of the skull.

It is accepted that the skull lesion is secondary to tuberculosis elsewhere and the probable route of infection is via the blood stream. Skull lesions are seen more commonly in the fronto-parietal

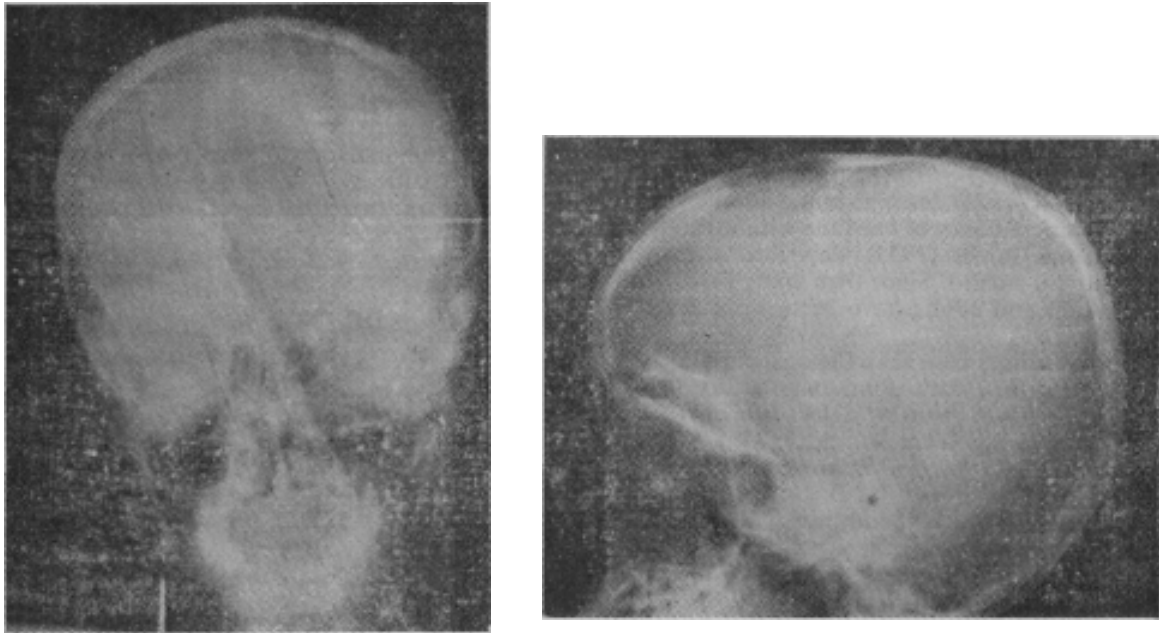


Fig. 1

Skull x-ray antero-posterior and lateral view showing osteolytic lesion in the fronto-parietal region.

region than in the occipito-temporal region. The tuberculous focus in the skull starts in the diploe and may erode either one or both tables of the skull, giving a clear punched out appearance on skull x-ray. Minute sequestra show up as bone sand in the osteolytic lesion. If the outer table of the skull is eroded, a fluctuant swelling of the scalp develops, and when the inner table is also eroded, the disease spreads extensively in the extra dural space as was seen in this case. Depending upon the individual resistance, the lesion may be either of a circumscribed punched out type or may be diffuse and infiltrating. The dura matter forms an excellent protective barrier to the spread of the disease to the brain and meninges.

Treatment-wise, the disease responds very well to the usual anti-tubercular chemotherapy in the early stages. In later stages, with sequestration of bone and extensive caseation, surgical removal of all diseased tissue is essential.

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MACLEOD'S SYNDROME IN AN ADULT

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Introduction

Macleod (1954) described nine adult cases of Hyper-transradiancy of one lung without collapse. Swyers and James (1953) described a similar condition in a child. Since then many cases both in children and adults have been reported.

Some authors believe in the congenital origin of the syndrome with pulmonary artery hypoplasia being the primary defect (Belcher and Pattison, 1957; Belcher et al., 1959). Others (Sywer and James 1953, Rakower and Moran, 1962; Reid and Simon 1962), however, believe that it is an acquired condition with bronchopulmonary lesions being the primary abnormality and the pulmonary vascular hypoplasia developing later. Reid (1967). Reid et al. (1967) and Houk et al. (1957) reported cases in which the radiograph was normal before the onset of infection thus providing a strong support for the acquired bronchial disease theory.

We report below a case of Macleod's syndrome affecting the left lung of an adult. A radiograph taken one and a half year earlier showed a normal left lung.

Case Report

M. a 15-year old girl complained of cough,

The patient came for routine check in May 1969. An x-ray chest showed abnormal transradiancy of the left lung. On Physical examination diminished movements, prolonged expiration and fine crepitant rales were found in the left chest. She presented at the hospital again in May 1972 with dry cough and a small hemoptysis. An x-ray chest (Fig. 2) showed persistence of abnormal transradiancy of the left lung. A bronchogram (Fig. 3) showed patent main bronchi and normal segmental distribution of peripheral branches and a complete lack of bronchiolar filling of the left lung ("pruned tree appearance"). Some of the smaller bronchi showed dilatation or terminal clubbing. In contrast the bronchogram of the right lung was

normal. A pulmonary angiogram (Fig. 4) revealed a decrease in calibre of the pulmonary artery and its branches traversing the hyperlucent lung.

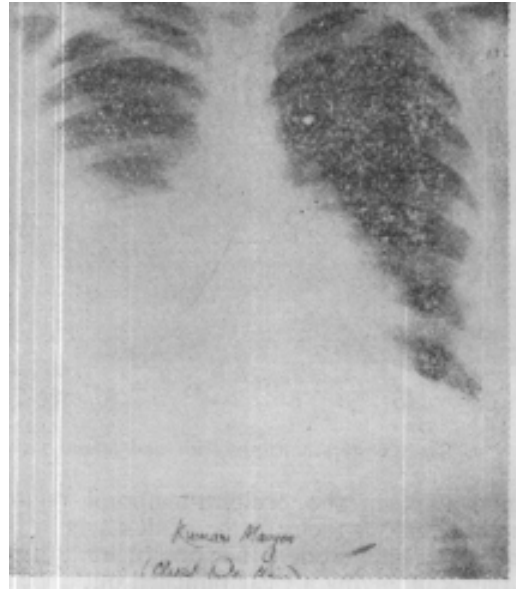


Fig. 1

X-ray chest showing a right pleural effusion. The left lung appears normal.

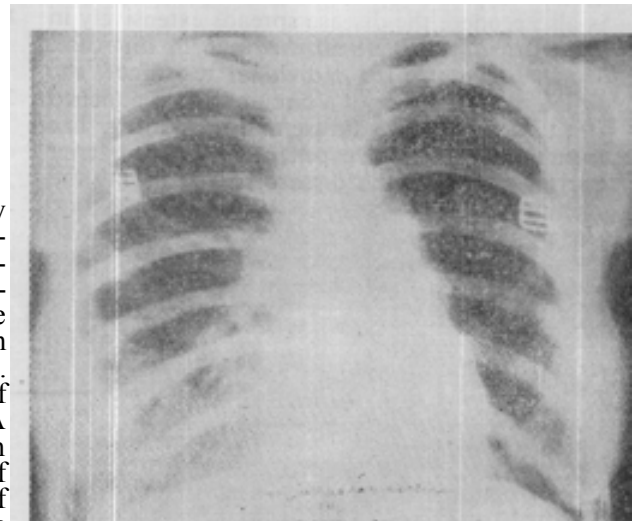


Fig. 2

X-ray chest showing transradiency of the left lung;

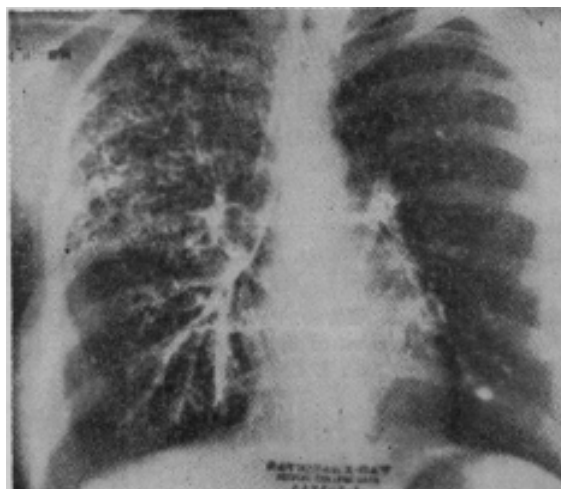


Fig. 3

Bronchogram showing poor filling of the peripheral branches and a complete lack of bronchiolar filling.

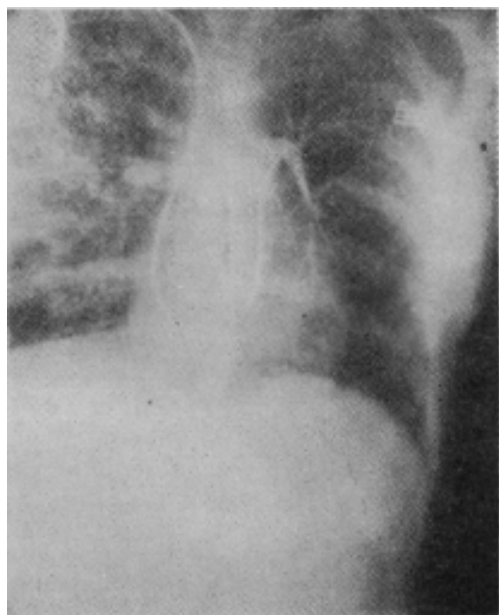


Fig. 4

Pulmonary angiogram showing decrease perfusion of the left lung. The opacities in the right lung are due to retained contrast material used for bronchography.

The patient has been followed up. She is alright except for occasional episodes of cough and small hemoptysis responding to broad spec-

trum antibiotics. There is no change radiographically; crepitant rales on the left side of chest persist.

Discussion

The diagnosis of Macleod's syndrome was suspected on routine chest x-ray and confirmed by bronchography and pulmonary angiography.

It is evident that the patient developed the syndrome sometime between December 1967, when the left lung was normal clinically and radiographically and May 1969 when trans-radiancy was first noticed.

The factors leading to the development of the syndrome can only be conjectured. A primary tuberculous infection caused a pleural effusion on the right side. Haematogenous dissemination to the left lung apparently resulted in peribronchial fibrosis with bronchial obliteration with some emphysema. Reid & Simon (1962) have described two cases in whom damage consequent to childhood tuberculosis was the cause of the syndrome. In one of these cases histopathological examination showed widespread damage throughout a lobe although it was not obvious on naked eye examination of the lung nor apparent on the radiograph. Thus a normal lung on x-ray does not exclude the possibility of presence of disease causing sufficient damage leading to the development of the syndrome. Macleod excluded from his series cases with radiological evidence of primary tuberculosis infection. But the later workers (Reid & Simon, 1962, Kulpati et. al. 1977) have pointed out that tuberculosis can be a cause of Macleod's syndrome.

The classic study of Dunnill (1962) indicates that the alveolar multiplication occurs post-natally upto the age of eight. So, damage to the lung due to an infection in early childhood will result in hypoplasia of the involved lung. However, the data of Thurlback and Angus (1975) indicate that alveolar multiplication with increase in volume can continue to occur upto the age of 18 years. Hence, emphysema with hypoplasia as found in Macleod's syndrome can also possibly occur due to injury in later childhood or early adult life.

Summary

A case of Macleod's syndrome affecting the left lung in a 15 years old girl is reported. An x-ray chest done one and a half year earlier showed a normal left lung. The etiology was probably tuberculosis.

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**THE TWENTYFOURTH WORLD CONFERENCE ON TUBERCULOSIS AND
ADMINISTRATIVE MEETINGS OF THE INTERNATIONAL UNION
AGAINST TUBERCULOSIS**

A Short Report

P.N. RAMAN*

The 24th World Conference of the I.U.A.T. was inaugurated in the "PaLals des Congress" by the Hon'ble Mr. L. Tindemans, the Prime Minister of Belgium before a gathering of about 2,000 of which about 1,500 were said to be delegates. From India Prof. R. Viswanathan, Lt-Genl. J.C. Chatterjee, Dr. B.N.M. Barua, Dr. H.B. Dingley, Shri P.N. Raman (all from Delhi), Dr. S.P. Tripathy (Madras), the Hon'ble Shri Margada Mallappa (Health Minister, Karnataka), Dr. T. Manickam and Shri M. Ramaswamy Reddy (from Bangalore), Dr. M.L. Mehrotra (Agra) and Dr. A.G. Patel (Baroda) attended the Conference.

In his inaugural address Mr. Tindemans made an impassioned appeal for international cooperation and solidarity in fighting Tuberculosis. Prof. V. Farga, Executive Director of the IUAT, in his address drew attention to the fact that while the economically advanced countries were thinking in terms of eradication of Tuberculosis, the situation was dramatically different in the developing countries. The latter were still struggling with problems relating to case-finding and treatment of patients largely due to lack of funds and non-availability of powerful drugs in large quantities. A change in the present unsatisfactory situation in these countries could be brought about only by finding all infectious cases and treating them effectively as quickly as possible. He therefore pleaded for international collaboration in bringing down the price of the most effective drug "Rifampicin" and also in making it available in large quantities to developing countries so that a tremendous impact could be achieved on the TB problem.

The Session was also addressed by Prof. A. Gyselen, President of the IUAT and President of the Conference, Dr. K.L. Hitze of the World Health Organisation, Dr. J.A. Aluoch (Kenya), Dr. J.S. Sodhy (Malaysia) and Dr. E. Crockett (United States).

The arrangements made for the Conference were excellent and left nothing to be desired. Besides the official languages of the Conference viz. English and French, simultaneous interpretations in Spanish and German were also avail-

able in all the Conference Halls. There were interesting film shows on Tuberculosis and allied diseases. For the benefit of the delegates a Commercial and Scientific Exhibition had also been organised.

Scientific Sessions

The Scientific Sessions of the Conference were held simultaneously in three different Halls from the 6th to 9th September. About 220 papers spread over 35 sessions were presented from different countries. Of these, six papers were from India. The main subjects discussed included Short Course Chemotherapy in the Treatment of TB, Chronic Obstructive Pulmonary Diseases, TB Bacteriology and Immunology, Tuberculosis Case-finding, Extra-pulmonary Tuberculous Diseases, TB and Community Health Care, Epidemiology of Tuberculosis and Non-tuberculous diseases, Chemoprophylaxis, Atypical Mycobacteria, Leprosy, Smoking, etc.

All the Sessions were well-attended. The sessions on "Short-Term Chemotherapy" were easily the most popular. Over 30 papers were presented on the subject and there was also a Plenary Session in which Dr. W. Fox (BMRC) discussed some of its main features in the light of the studies presented at the Conference including the choice of the drugs, duration of Chemotherapy, etc. There were three sessions devoted to surveillance which showed the need to monitor the application of tuberculosis measures and to assess the epidemiological impact of the programmes. The 5-year results of the cooperative international study on INH prophylaxis in individuals with fibrotic lesions of the lung as well as the study on evaluation of treatment results at national level in routine practice were presented. The work of the Tuberculosis Surveillance Research Unit was synthesised in a general presentation with illustrative examples. There were also two sessions on Leprosy and Tuberculosis and twelve sessions on non-tuberculous respiratory diseases.

General Assembly

The General Assembly of the Union met on the 9th September, with Prof. A. Gyselen in the

*Secretary-General, Tuberculosis Association of India.

Chair. Dr. H. Coudreau, Chairman of the Executive Committee of the IUAT, announced that the Union was still in correspondence with the Brazilian Association about holding the 25th Conference in Rio-de-Janerio in 1981 and that until a firm decision is taken and a new President appointed, Prof. Gyselen will continue as the President of the Union. He also announced the appointment by the Council of Dr. Annik Rouillon to succeed Prof. V. Farga as Executive Director of the Union.

Conference of Executive Directors

The Conference of Executive Directors and Secretaries General of National TB Associations was held on 4th September at the "PaLals des Academies". The Proceedings were conducted by a Board consisting of Dr. I. El Rifai (Syria) as Chairman and Drs. O. Schweiger (Hungary) and T. Shimao (Japan) as members. Representatives of 60 out of 97 constituent members of the Union participated in the Conference. The Tuberculosis Association of India was represented at the Conference by Prof. R. Viswanathan and Shri P.N. Raman.

The Conference reviewed the position in regard to the assessment of contributions payable by constituent members to the Union and suggested that since the quota system could not be applied rigidly to every country, the Quota Committee should work out new criteria which would satisfy the Union as also the constituent members.

Another important subject discussed at the Conference was the proposal to expand the activities of the Union to include respiratory Diseases and Community Health Programmes. A majority of representatives expressed themselves in favour of this proposal. The consensus of opinion was that by diversifying its activities the Union will not be losing anything. On the contrary it will be drawing into its fold new groups of people and the Union may be in a better position to focus attention not only on TB but other allied respiratory diseases as well. The experience of some of the National Associations which had expanded their activities to include diseases other than TB had proved this beyond doubt. On behalf of India Prof. Viswanathan pointed out that historically the IUAT had been established primarily for the purpose of controlling and later on eradicating TB. Since TB still continued to be a major public health problem in countries covering more than 3/4 of the World population he felt that the time had not yet come for the Union to diversify its activities and that it should continue to devote all its attention to TB only. Dr.N.C.Sen Gupta from

Singapore spoke about the need for the development of community health programmes and an adequate health infra-structure even in developing countries and said that this could be achieved only by expanding the Union's activities. Dr. J.N. Giri from Nepal said that whatever may be the final decision of the Union in regard to expansion of its activities there should be some distinction in dealing with countries like Nepal where even the extent of the TB problem had not yet been assessed, not to speak of controlling it. Dr. Sodhy, speaking on behalf of the Eastern Region, said that while industrialised countries like Australia welcomed the change and Malaysia had accepted it with grace, TB still continued to be the main concern in some of the countries of the Region and this factor had to be taken into account in coming to a decision.

Dr. K.L. Hitze (WHO) referred to a resolution recently passed by the World Health Organisation on "Active Programme on essential drugs" and said that urgent action to bring about a reduction in the cost of Rifampicin and also to make it available in large quantities to all developing countries to enable them to treat their TB cases is contemplated. How this will be achieved and how the funds necessary for this purpose will be raised etc. are details yet to be worked out.

Council Meeting

The meeting of the Council of the Union was held from 8 A.M. to 2 P.M. on 5th September. Prof. A. Gyselen, President of the Union presided over the meeting. Out of 97 countries affiliated to the Union 44 were represented at the meeting. Prof. R. Viswanathan and Shri P.N. Raman acted as Councillors from India and Dr. H.B. Dingley attended the meeting as an observer. After the roll call of the Councillors and adoption of the Agenda, the minutes of the Council meeting held in Istanbul on 24.10.1977 were confirmed.

Dr. H. Coudreau, Chairman of the Executive Committee recorded the high appreciation of the Council of the valuable services rendered by Prof. Farga during his term of office as Executive Director of the Union.

The Council adopted the report on the activities of the Union for the period October 1977 to September 1978 and approved the statement of accounts of the Union for 1977 and the budget for 1979. The Council also received reports from Dr. K. Styblo, Coordinator of Scientific Committees, Dr. W.W. Holland, Chairman of the Committee on Respiratory Diseases, Dr. I. El Rifai on the Conference of Executive Directors held on the previous day, and reports on the

activities of the six Regional bodies of the Union presented by the Chairman/Secretary-General of the respective Regions.

With regard to the proposal to change the name of the Union to include Respiratory Diseases and Community Health it was decided that the Secretariat of the Union may prepare a report and circulate it to the constituent members for their comments and the question may be examined again next year.

The Council approved the admission of four new members thus bringing the total number of constituent members of the Union to 101. As for the admission of the Republic of China, it was decided that the Secretariat should get in touch with the TB Association of China and obtain further clarification about its status etc. with a view to decide about its membership.

The Council was informed that the next Annual meetings of the Union will be held in Paris towards the end of October or early in November 1979. It was also announced that the Union was in correspondence with the Brazilian TB Association on the question of holding the 25th World Conference in Tuberculosis in Rio De Janeiro in 1981.

Eastern Region Meeting

The meeting of the Council of the Eastern Region was held on 7th September with Dr. J.R. Wilson, President of the Region, in the Chair. About 40 delegates representing 16 of the 19 Constituent Members of the Region attended the meeting. From India Prof. R. Viswanathan and Shri P.M. Raman attended as Council Members and Drs. H.B. Dingley, A.G. Patel and M.L. Mehrotra attended as observers. The meeting was also attended by Dr. H. Coudreau and Dr. A. Rouillon, Chairman and Executive Director respectively of the IUAT, and Dr. E.I. Hershfield, Medical Director of the Canadian Lung Association.

After confirmation of the minutes of the meeting of the Council held on 13th October, 1976, at Seoul, Korea, the Report on the activities of the Region during the period October 1977 to September 1978, presented by Dr. J. S. Sodhy, the Secretary-General, was adopted. The Council approved the statement of accounts for the years 1976 and 1977 presented by Dr. N.C. Sen

Gupta, the Treasurer and passed the budget for 1979 with the modification that the provision for the Bulletin may be increased slightly, if found necessary.

The Council directed the Secretary-General to redraft the Constitution of the Region by incorporating the proposed amendments and circulate it to members for their comments 90 days in advance of the next meeting so that it may be considered and got approved at the Council meeting to be held at the time of the next Regional Conference in Sri Lanka. The Council noted that the Proceedings of the Seoul Conference were being printed in Korea and that copies were expected to be ready for distribution early next year. The Council also noted that efforts were being made to enrol Papua, New Guinea, Western Samoa, etc. as members of the Region.

Prof. Viswanathan suggested that the dates for the Sri Lanka Conference may be fixed in such a way that delegates attending the Paris -meeting of the IUAT in the first week of November 1979 can go over to Colombo to attend the Regional Conference there and thereafter proceed to Bombay to attend the Asian Pacific Conferences on Diseases of the Chest in the 3rd week of November 1979. This was agreed to.

Dr. Rouillon expressed her happiness in being able to attend the Regional meeting and thanked the delegates from the Eastern Region for coming in such large numbers to attend the World Conference at Brussels. She appreciated the good work being done by the Region and said that she was very much looking forward to attend the Regional Conference in Sri Lanka. Dr. Hershfield conveyed the greetings of the Canadian Lung Association and said that his Association would continue their aid, under the mutual assistance programme of the IUAT, to the various projects sponsored by the Eastern Region. The delegates from Indonesia and Nepal expressed their appreciation and thanked the IUAT and the Eastern Region for their help and guidance in promoting voluntary anti-TB work in their respective countries.

In conclusion, Dr. J.R. Wilson, the President of the Region extended a cordial invitation to one and all for the Sri Lanka Conference and requested the member countries to send as large delegations as possible to this Conference.

NEWS & NOTES

SECRETARY-GENERAL, TUBERCULOSIS ASSOCIATION OF INDIA

Shri B.M. Cariappa, Secretary General, Tuberculosis Association of India, retired on 30.6.78 and Shri P.N. Raman has taken over as Secretary-General of the Association. Shri Raman has been closely associated with the work and working of the Central and State Tuberculosis Associations for over 35 years.

29TH SEAL CAMPAIGN

Shri Neelam Sanjiva Reddy, President of India, inaugurated the 29th TB Seal Campaign on 2nd October at Rashtrapati Bhavan by making a token purchase of TB Seals. The function was organised by the Tuberculosis Association of India.

President Reddy in a message said:

“The TB Seal Campaign provides an opportunity to educate our people that TB is preventable and curable and should be prevented and cured. I appeal to all my countrymen to buy TB Seals in large numbers and wish the Tuberculosis Association of India every success in its efforts to fight this scourge.”

Shri S. Ranganathan, President of the Association, in a message said:

“TB Seal Campaign is one of the most effective and constructive endeavours systematically carried out by our TB Associations to strengthen the anti-TB movement in the country.... I am

glad that the Association has decided to celebrate its Foundation Day—the 23rd February — as “ANTI-TUBERCULOSIS DAY” and to terminate the Campaign on that day. I appeal to the people of India to support the Seal Campaign and help their Associations to strengthen themselves to serve this humanitarian cause”.

33RD NATIONAL CONFERENCE

The 33rd National Conference on TB & Chest Diseases will be held in the auditorium of the Mahatma Gandhi Medical College, Bhopal, from the 22nd to 26th November, 1978. The Conference will be inaugurated by His Excellency the Governor of Madhya Pradesh, Shri C.M. Poonacha and the inaugural session will be addressed by the Chief Minister and Health Minister of the State. Prof. J.L. Bhatia, Professor and Head, Department of TB & Chest Diseases, Medical College Hospital, Adviser in Tuberculosis to the Government of Punjab, Amritsar and Chairman, Standing Technical Committee of the Tuberculosis Association of India, will preside over the Conference. The main subjects to be discussed at the Conference are (1) National TB Control Programme - Problems of treatment, (2) Chemotherapy, (3) TB in Children including Tuberculosis of the Central Nervous System and TB Meningitis, (4) Community Participation in TB Control Programme, (5) Bronchial Asthma, (6) Air Pollution, (7) TB and Leprosy, etc. For details of the programme, registration of delegates, etc. please contact the Secretary General, Tuberculosis Association of India, 3, Red Cross Road, New Delhi-1.

NEWS FROM STATES

REFRESHER COURSES

An intensive Refresher Course on TB was organised on 15th July at TB Centre, Irrumnuma, Hyderabad, under the joint auspices of TB Association of Andhra Pradesh and City and District TB Association of Hyderabad. The course was inaugurated by Dr. A. Chandra-sekhar, Deputy Secretary to the Government, Medical and Health Department and the function was presided over by Dr. S.N. Mathur, Director of Medical and Health Services. About 150 doctors including Medical Officers of various industrial undertakings participated in the Refresher Course.

A two-day Refresher Course in TB was organised by the West Godavari District TB Association at Eluru on 12th and 13th August, 1978. Shri D. Subba Rayalu, District Revenue Officer and In-Charge Collector, inaugurated the Course. Dr. V. Nagabhushanam, Regional Director of Medical and Health Services, presided over the Scientific Sessions which included lectures on ‘Pitfalls on the diagnosis of Tuberculosis’ by Dr. A. Chakrapani Rao, “Childhood Tuberculosis” by Dr. P.S.N. Murthy, “Recent trends in the management and Chemotherapy of Tuberculosis” by Dr. T. Rama Rao, and “Non-Tuberculous lung conditions” by Dr. K. Kotilingam. Dr. V. Chittiseshu, Assistant

Director of Medical and Health Services, Andhra Pradesh, spoke on the District TB Control Programme. The second day's programme which was for para-medical staff working in public health centres and other Government hospitals was presided over by Dr. T. Rajaratnam, District Medical and Health Officer. The local branch of the Indian Medical Association, the West Godavari District Red Cross branch, Messrs. Alembic Chemicals, Parke Davis and Pfizer extended their cooperation in organising the Course which was attended by about 150 Medical Officers and private Medical Practitioners in the district.

SEMINAR

The District TB Association, Kurnool, conducted a Seminar on TB on 20th August, 1978. Dr. D. Umapathy Rao, Honorary General Secretary, State TB Association, Hyderabad, presided. Dr. Venkataswamy, Additional Professor of Medicine, Kurnool, presided over the first session. The papers presented included "Epidemiology of Tuberculosis" by Dr. C. Sreenivasa Rao, "Incidence of Tuberculosis in rural population of Nellore" by Dr. I.L. Narasaiah, "Changing Concepts of BCG Vaccination" by Dr. Kul Bhushan "Radiological diagnosis of Pulmonary Tuberculosis and Pitfalls" by Dr. T. Jayanarasimhulu and "Tuberculosis Tubercular pleural effusion" by Dr. R. Venkateswara Rao.

Dr. N. Ramachandra Rao presided over the second session which discussed the National Tuberculosis Control Programme and the feasibility of collecting sputum slides at the doorstep of tire suspected cases in the periphery. Papers were also read by Dr. T. Rama Rao on "Chemotherapy of Tuberculosis", Dr. B.P. Narasimhulu on "Diagnosis complications and treatment of tuberculosis of Spine" and Dr. I. Dinakaran "Neuro-Tuberculosis."

BCG VACCINATION DRIVE

The Kodagu District TB Association organised a mass BCG vaccination Camp at the premises of Government Primary School, Chettali, Kodagu District, on 22nd June. A total number of 878 persons were given BCG vaccination.

SHIBIRS

The Maharashtra State Anti-TB Association participated in a multi-check-up Camp held at Chinchipokli on 20th August jointly organised by the Insurance Medical Practitioners' Association, Chinchipokli, Ganeshotsav Mandal and Maharashtra State Anti-TB Association. In all

68 persons were examined and 27 persons were BCG vaccinated. The State Association also carried out, in cooperation with the Indian Medical Association and Sitaram Mill Social Welfare Centre, a multi-check-up Shibir at the Mill Compound on 30th July. A team of 15 specialists examined 800 workers and their families.

The Maharashtra Association also organised an anti-TB Shibir on 24th September, 1978 at Prince Aly Khan Hospital in cooperation with Lions Club of Byculla, Bombay Central and E-Ward Medical Association. At this Camp 246 persons were examined and 139 were screened. 30 were x-ray positive and 6 were sputum positive.

SURVEYS

Some of the District TB Associations of Madhya Pradesh have undertaken a survey on 'Hair Cutting Saloon Workers' in cooperation with the official agencies. A Protocol for the survey was drawn up by a Committee with Dr. N.L. Bordia as Convener. The Associations have decided to take up this survey as a regular activity and a report of this survey is expected to be ready shortly.

STATE CONFERENCES

The Rajasthan State TB Association organised its second Provincial TB and Chest Diseases Workers Conference on 28th and 29th August at the Medical College Campus, Ajmer. Shri Trilok Chand Jain, the Health Minister inaugurated the Conference. The Scientific Sessions were presided over by Dr. N.L. Bordia, Dr. G.S. Jhala, Dr. S.P. Pamra, Shri V.I. Rajagopal, Dr. G.C. Sharma and Dr. Rameshwar Sharma. The papers presented included "Short-term Chemotherapy", "Differential Tuberculin Test", "Pitfalls in National Tuberculosis Programme", "Role of Para-Medical Personnel", "Socio-Economic Aspects of Tuberculosis", "Surgery in Pulmonary Tuberculosis", etc. Shri P.N. Raman, Secretary-General, Tuberculosis Association of India, presented a paper on 'Role of Voluntary TB Associations'.

The TB Association of Andhra Pradesh proposes to hold its 7th TB & Chest Diseases Workers Conference at Vijayawada in December next. The Krishna District TB Association will be the host.

The TB Demonstration & Training Centre and Chest Institute, Agra, proposes to hold the second State TB and Chest Diseases Conference on 16th and 17th December, 1978 at the premises of the Centre.

INDIAN PHARMACEUTICAL CONGRESS ASSOCIATION

The XXX Annual Session of the Indian Pharmaceutical Congress will be held from 26th to 28th December, 1978 at Calcutta. The different sections of the Scientific meetings include (1) Medical Chemistry, (2) Analytical Chemistry, (3) Pharmacology and Drug Metabolism, (4)

Phytochemistry and Pharmacognosy (5) Industrial Pharmacy and Microbiology, (6) Biopharmaceutics and Clinical Pharmacy, (7) Hospital Pharmacy, (8) Unani & Ayurvedic Pharmacy, (9) Pharmaceutical Education and (10) Professional and Forensic Pharmacy. For details write to the Convener, Scientific Services Committee, D-312, CIBA-GEIGY Research Centre, Goregaon East, Bombay-400 063.

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ABSTRACTS

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Abst. No. 4

Pulmonary airway clearance mechanisms : A Reappraisal

A. Van As, Amer. Rev. Resp. Dis.; 1977.115, 721.

An inhaled particle does not get automatically transported after deposition but only after it has come into contact with mucus. Owing to discontinuity of mucus in the pulmonary airways, the particle movement will be sporadic and uneven. The role of mucus, thus, as a physical barrier in the airway defence mechanism seems to be minor. The respiratory epithelium is vulnerable, because it is continually exposed to exogenous irritants, pollutants and infective agents. Because mucus is sparse in the airways, other secretions such as IgA, bronchial lactoferrin, bronchial lysozyme and some serum proteins become more important in their role of protecting the airways from primary invasion. These substances could well reside in the fluid layer related to the cilia.

S.P.P.

Clinical aspects of mucociliary transport

Adam Wanner, Amer. Rev. Resp. Dis.; 1977, 116, 73.

Mucociliary transport in the lung is capable of removing inhaled particulate matter from the ciliated airways; however, the importance of mucous transport as a pulmonary defence mechanism has not been demonstrated experimentally. Normal mucociliary function depends on a morphologically and functionally intact ciliated epithelium as well as normal rheologic properties and quantity of respiratory secretions for optimal mucociliary interaction. The pathophysiologic features of the mucociliary apparatus and the presence of impaired mucous transport mechanisms under various conditions both demonstrate the extreme vulnerability of this non-respiratory function of the lung. Thus, decreases in temperature and humidity, exposure to inhalants such as cigarette smoke, atmospheric pollutants, and supplemental O₂ or certain pharmacologic agents all depress mucous transport. Likewise, mucociliary dysfunction appears

to be associated with obstructive lung diseases and acute respiratory infections. Stimulation of mucociliary transport by drugs, notably B-adrenergic agonists, cholinergic agents and methylxanthines has been clearly observed and may be of clinical relevance in patients with retained tracheobronchial secretions.

Because several studies have demonstrated that impairment of mucous transport may represent the earliest detectable sign of lung injury, it is not surprising that mucociliary dysfunction has been implicated as a pathogenetic factor in a number of pathologic conditions of the lung. Although this has not been documented experimentally, the possibility remains that an intact mucociliary apparatus may protect against the development of certain lung disorders.

S.P.P.

Respiratory abnormalities among grain handlers

G.A. Dopico et. al. Amer. Rev. Resp. Dis.; 1977, 115,915.

A survey of 300 grain elevator workers revealed that 77% complained of eye symptoms; 64% of nasal symptoms; and 88% of one or more respiratory symptoms on exposure to airborne grain dust. Symptoms on exposure were independent of age and length of employment. Cough and wheezing on exposure were more common among smokers than non-smokers ($P < 0.025$). Nineteen per cent of the workers had had episodes of grain fever. The prevalence of chronic bronchitis was 37% (42% of smokers and 30% of non-smokers). Wheezes on auscultation were found in 23%. Measurements of lung ventilatory function, as well as diffusing capacity, correlated significantly with age and smoking habits, but not with length of employment. Thirty-seven per cent of the workers had an abnormal mean forced expiratory flow during the middle half of the forced vital capacity (47% of smokers and 13% of non-smokers) and 34% had an abnormal maximal expiratory flow after exhalation 50% of the forced vital capacity (40% of smokers and 13% of non-smokers) whereas only 13% had an abnormal ratio of 1-sec forced expiratory

Ind. J. Tub., Vol. XXV, No. 4

volume to forced vital capacity. There was no correlation between precipitins to fungi, bacteria, grain, or grain dust antigens and acute or chronic respiratory symptoms, lung function, or grain fever. There was however, a significant correlation between cutaneous reactivity to grain dust and wheezing on exposure ($P < 0.02$). Abnormal flows at low lung volumes were more common among cutaneous reactors to common allergens. It is concluded that exposure to airborne grain dust can cause acute inflammatory reaction in the exposed mucosa, and it is highly probable that grain dust contributes and, in some cases, cause chronic airway disease.

S.P.P.

Pulmonary function in granite dust exposure: A four-year follow up.

A.W. Musk et. al. Amer. Rev. Resp. Dis.; 1977, 115, 769.

Pulmonary function studies were performed on 974 workers in Vermont granite sheds in 1974. Of these subjects, 668 had been studied 4 years earlier and had remained in jobs in which their exposure to granite dust had not changed based on dust concentrations measured during 1970. The yearly decrement in pulmonary function observed in the 668 granite shed workers was excessive (0.07 to 0.08 liter per year for forced vital capacity and 0.05 to 0.07 liter per year for forced expiratory volume in 1 sec). This exceeded the expected decrement derived from several other occupational and population groups. Studies from this laboratory consistently indicate a decrement of no more than 0.03 to 0.04 liter per year in both forced vital capacity and forced expiratory volume in 1 sec. The observed decrements were independent of exposure groups and not accounted for by cigarette smoking. In 528 additional granite shed workers, decrements in ventilatory capacity had been measured for one, two or three years and were consistently of the same order of magnitude. Dust concentrations, within defined jobs and between granite sheds showed great variability. Despite this, a suggestive relationship between exposure and decrement in ventilatory function was demonstrated at the end of 2 years; however, at the end of 4 years the relationship could no longer be shown with these exposure groupings. The difficulty in characterising individual dust exposure and projecting dust concentrations for several years is considered to account for the absence of a dose-response relationship at the 4-year follow up. The results of this study suggest that previous estimates of annual deterioration in ventilatory capacity attributable to work in granite sheds are under-estimates.

S.P.P.

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The significance of Asbestos Exposure in the diagnosis of Mesothelioma: A 28-year experience from a major urban hospital

Fay sal M. Hasan et. al. Amer. Rev. Resp. Dis.; 1977, 115, 761.

A continued increase in the incidence of diffuse mesothelioma has been attributed to greater industrial use of asbestos but is also due in part to wider acceptance of this tumour by pathologists. In this retrospective study, the epidemiology, clinical presentation, and pathology of asbestos and non-asbestos related mesothelioma from a major urban hospital were reviewed. Of the 36 cases of mesothelioma on file, 19 were not associated with exposure to asbestos. Although a retrospective study raises the possibility of inadequate occupational histories, the lack of history of asbestos exposure correlated with postmortem histology by light microscopy. When postmortem material was reviewed, evidence of asbestos exposure was present in all cases of mesothelioma with history of exposure to asbestos and in no cases in which the patient denied history of asbestos exposure. Using strict histologic and histochemical criteria, the diagnosis of mesothelioma was confirmed in 8 of 9 patients with asbestos-related mesothelioma but in only 4 of 13 cases of non-asbestos related mesothelioma.

The diagnosis of diffuse mesothelioma is often difficult to make even with complete autopsy examinations. It should be entertained only with adherence to strict clinical and pathologic criteria, especially in women with no history of exposure to asbestos dust.

S.P.P.

Co-existence of Actinomycosis of Lungs with Pulmonary Tuberculosis

S.C. Chakravarty & J. Fernandez. J. Ind. Med. Assn.; 1977, 69, 89.

Two cases of bacteriologically confirmed pulmonary tuberculosis are reported where Actinomycosis of the lung was co-existent with tuberculosis. In both cases there was no evidence of empyema or chest wall sinuses due to the fungus. Investigations for fungus were carried out when the tuberculous patients (125 in all) failed to respond satisfactorily to anti-tuberculous treatment. *Actino. Israeilli* was repeatedly isolated from anaerobic cultures of sputum on brain-heart infusion agar and liquid thioglycollate broth medium in both cases. Improvement became satisfactory when treatment for Actino-

mycosis was added to the anti-tuberculous treatment. Absence of chest wall lesions could have been due to anti-biotic treatment received by these patients already or due to pulmonary Actinomycosis being in early stage.

[S.P.P.]

Lymphomatoid Granulomatosis: Association with Retriperitoneal Fibrosis and evidence of impaired cell-mediated immunity.

Samuel P. Hammer et. al., Amer. Rev. Resp. Dis.;1971, 115, 1045.

Lymphomatoid granulomatosis is a non-neoplastic lymphoreticular disorder that usually presents as primary lung disease. A case is reported of 61-year old white man who developed persistent right sided abdominal pain with associated nausea and vomiting in 1965. All investigations proved negative. The abdominal pain recurred in 1972 with polyuria and polydipsia. Excretory urogram showed obstruction and slight medial deviation of the right ureter and a small shrunken right kidney. The left kidney was mildly hydronephrotic and the left ureter was partially obstructed. Chest x-ray was normal. Right nephrectomy was done and left ureter was dissected and relocated. Biopsy of the retroperitoneal tissue showed infiltrate of lymphocytes, plasma cells and large mononuclear cells with infiltration around small blood vessels and peripheral nerves. The resected kidney showed end-stage renal disease consistent with longstanding obstruction and/or pyelonephritis. In 1975 there was marked loss of weight and chest x-ray showed 4x7 cm mass in the right anterior lung field that was diagnosed tentatively as a lung tumor. He had not smoked since 1940. Right middle lobectomy was performed. The resected lobe contained a 4 x 8 x 6 cm gray-white firm nonencapsulated mass that replaced almost the entire parenchyma. The mass did not arise from a bronchus. Multiple sections showed an appearance similar to the earlier retroperitoneal biopsy suggesting that the disease may have begun in the retroperitoneum. The histological appearance on the whole was of lymphomatoid granulomatosis. An extensive immunological evaluation of the patient revealed impaired cell-mediated immunity.

S.P.P.

Intra-Cavity Suction and Drainage in the Treatment of Emphysematous Bullae.

A.M. Macarthur and S.W. Fountain. Thorax, 1977, 32, 668-672.

Thirty-one patients were treated by intracavitary suction and drainage. There were two

(65 %) operative deaths. Apart from infection no other significant post-operative complications ensued. Radiographic improvement occurred in all patients but one (96.7%) symptomatic improvement occurred in 28 (90.3%).

H.B.D.

Percutaneous Drainage in the Treatment of Klebsiella Pneumoniae Lung Abscess.

E.W.J. Cameron and I.D. Whilton. Thorax, 1977, 32, 673-676.

Operative management of seven patients of lung abscess involving klebsiella pneumoniae with or without other pathogens presented with gross expansion of the involved lobe or segments and severe clinical illness despite medical treatment by rib resection and tube drainage was successful in all the patients.

H.B.D.

Lymph Node Tuberculosis : A Comparison of various methods of Treatment.

I. A. Campell and A.J. Dyson: Tuber, 58 (1977) 171-179.

The treatment of lymph node tuberculosis has been studied in 108 patients. Chemotherapy consisted of 18 months isoniazid with either rifampicin or ethambutol plus an initial supplement of streptomycin.

19 patients who had previous excision of lymph nodes, 56 patients who had previous biopsy and 33 patients without surgical intervention were allocated to randomly to these two drug regimens.

Histological evidence of tuberculosis was obtained in 64% and M. tuberculosis was cultured in 30%. No other mycobacterium were grown.

Progress during treatment was uneventfully in 65%. Fresh nodes appeared during treatment in 12%. Existing nodes enlarged in 13% and fluctuation developed in 11% of patients. Discharge and/or sinus formation was in 7%, breakdown of surgical scar in 4%. Excision or aspiration after the start of chemotherapy was performed in 19% of chemotherapy. No difference was seen in the treatment between the two groups. Satisfactory results were obtained in 98 % of the patients by the end of treatment, although 13% had slight node enlargement.

H.B.D.