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## AIR POLLUTION

Air sustains human life and provides mankind free drain of activities for throwing endless pollutants of many kinds. For long there was scant awareness that these pollutants may be dangerous nor was it necessary as the pollution could not have been hazardous under the then living and working conditions. Human beings have passed through many a phase of evolution. Accordingly their social needs and life-style have changed. The rapidly increasing socio-economic needs motivate people to secure various sources of power to lessen their burden. During the last two centuries human ingenuity has tapped tremendous power from nature by science and technology and developed machines and industries with the concomitant result of developing crowded urban areas. This process started throwing pollutants in the air in very large quantities. As time passed some adverse effects on human health were noticed and polluted air was regarded as a possible cause. Scientists and public started asking whether air pollution was lethal to life and whether it was harmful for crops and some other materials also. As time passed, the demand for an answer grew stronger leading to intensive studies in developed countries. The answer is yet not precise, but points to the potential dangers not only to human civilization, but to animal and plant life as well.

Let us consider mainly inhalant pollutants. The initial injury, if any, by these pollutants would be located in the respiratory system and cause chest diseases. Evidence so far collected also points in this direction. Considering the importance of workers in chest diseases keeping abreast with such developments, the Tuberculosis Association of India organised a symposium on the subject of air pollution at the National TB and Chest Diseases Conference last year. This issue of the Journal publishes an articles on the subject from Dr. B.B. Chatterjee who presided over the symposium in Lucknow. The article discuss the subject generally and indicates current position of air-pollution as a respiratory *hazard*.

The pathology in the early stage in generally located in the lining mucous membrane of the air-passages, causing chronic bronchitis. Chronic air-way obstructive phenomenon follows, leading to the dreaded complication of emphysema.

There are many sources of pollution. These may be natural, climatic phenomena like dust-storm, fog, etc. Excessive use of tobacco inhalation by smoking has so far been the most serious hazard of man-made air pollution which today accounts for maximum cases of incurable emphysema. Man-made industrial, agricultural and domestic sources are most important. Some

hazardous substances like mercury, lead, asbestos, etc. may get widely distributed in the atmosphere from chemical factories. But, the chief pollutants arise from combustion of fossil fuels mostly for generation of power and by automobiles. Most dangerous pollutants are sulphur dioxide, carbon dioxide, and carbon monoxide. Some others like fly ash, soot etc. and a small amount of radio-active particulates may also be present.

The atmosphere has cleansing mechanisms which keep the concentration of the pollutants in the atmosphere at low level. This is mainly by dilution caused by surrounding air. Besides, some chemical processes and currents of air and rain play generally important roles in keeping the concentration of the pollutants below the threshold of dangerous level.

Many workers who are deeply involved in this field believe that nature's processes alone can protect man from the health hazards of air-pollution. It can occur only under special conditions but not normally. Such special conditions may arise by inhalation of silica and other agents causing pneumoconiosis. They are, however, of a very limited nature and are discounted in this context. For the same reason, smoking, a personal pollutant and carcinoma with its probable association with pollution, have not been considered. The term "air-pollution" implies contaminants to which man at large is exposed. Documented evidence gathered so far tends to show that this kind of air-pollution can have adverse effects both on the function and structure of the respiratory system. For example, "Smog" in London in 1952 caused three thousand deaths from respiratory diseases, mostly however, in the older age-groups. A few other instances of such adverse effects on respiratory system from such natural episodes are also on record.

Most of the studies made are on the nature and concentration of pollutants in the atmosphere the sources of which are known. Technological advances have provided effective control measures against important pollutants, viz, Limestone injection for sulphurdioxide, gasification of coal before its use, mufflers for automobile exhaust, etc. How dangerous are these singly and jointly to human health, specially to the respiratory system, is not yet well established. To do so, study designs must eliminate major biases. Such planning and execution of studies appear difficult as many factors in the atmosphere are inseparably associated with the pollutants. Even so, it must be conceded that the primary objective of any study in this direction cannot be confined to mere identification and characterisation of pollutants, however important these studies may be; they must continue in order to determine their effect on health of human beings. Limitless ingenuity of man and pooling of knowledge and resources from workers from different faculties in this field should ultimately overcome difficulties and uncertainties.

In such co-ordinated ventures participation of supecialists in Chest Diseases may prove helpful as hazardous nature of a pollutant may initially manifest itself by chest symptoms and chest pathology only. It is also possible that this danger may escape its determination immediately, but may reveal itself by clinical manifestations on long-term exposure. This may also happen even when a hazardous paniculate substance is in sub-threshold concentration

in the atmosphere. Close and long observation and surveillance for chest diseases should therefore be a *must*.

What demands most careful consideration is vigorous action for prevention of air-pollution at this point of time when our industries are expanding and financial capabilities are still weak. Steps to implement control measures need education and persuasion of the industrialists and administrators and if necessary legislation. These involve cost. Besides, production may be retarded and cost may increase.

Though far behind many others, we have already started on the road of national re-construction and industrialisation. Special institutions and academic bodies in Nagpur, Ahmedabad, Calcutta, Asansol and Bombay are already involved in this subject. We are sure that with adequate support, they will deliver methods of prevention suitable under our conditions. Along with such efforts, intensive educational programme in under-graduate and post-graduate stages of medical studies should be planned. Price of pollution may rise unbelievably high in a few decades and people can rightly indict us for delay and denial of advantages of early start.

## ATMOSPHERIC POLLUTION AND RESPIRATORY DISEASES

B.B. CHATTERJEE

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For a little more than the last two decades a great deal of concern has been generated with regard to atmospheric pollution. But, it must not be thought that pollution of the atmosphere is of recent origin. The attention of scientists was forcefully drawn to it at about that period by episodes of widespread illness and death in certain industrial-urban localities, occurring during and immediately following, prolonged spells of severe degree of evident atmospheric pollution. There could be no doubts that the two phenomena were causally related. One of those severe episodes occurred in Demora in U.S.A. in 1948<sup>1</sup> and another one in London in 1952.<sup>2</sup> Although similar episodes of lesser severity and shorter duration must have been taking place in different localities from time to time for quite a few decades in the past, the associated excess morbidity and mortality, when observed, must have been attributed to inexplicable random fluctuations and not to the changes in the atmosphere. Search of literature reveals that only once before, in 1930, when the densely industrialised Meuse Valley in Belgium<sup>3</sup> was enveloped in dense smoke-filled fog (smog) for a few days and was accompanied with and followed by largely increased respiratory morbidity and mortality amongst the population of that region, suspicion was directed against atmospheric pollution as the causal agency.

Such acute visibly polluted states of air persisting for days, are rare occurrences brought about by freakish meteorological conditions which depend on the simultaneous operation of a number of circumstances. However, these initiated the widespread interest and investigations on the effects of atmospheric pollution on human health.

The manner in which air pollution is caused is obvious enough. The gaseous discharges from domestic ovens or from industrial furnaces and from stationary or mobile internal combustion engines such as the automobiles, are rich not only in a variety of potentially toxic gases but also contain a great deal of finely divided particulate matter. When such effluents are let out into the atmosphere it naturally becomes polluted. But then such pollution must have been occurring for decades and even centuries. Why were the effects not noticed before?

One of the important reasons is that the atmospheric envelope is huge and does not stay

polluted for long, as it can be cleaned by natural physical, chemical and mechanical self-purifying mechanisms. But such mechanisms must remain operative and the rate of supply of pollutants into the atmosphere should not be excessive. The greatly accelerated rate of growth of population as also of urbanisation and industrialisation in the recent decades have tended to overwhelm the natural atmospheric mechanisms specially in the densely populated areas with intensive degrees of industrialisation. In such areas, when the air is stagnant, cold, heavy and foggy, the pollution becomes not only visible but almost palpable and may cause the onset of sharply increased morbidity and mortality. But even under such conditions, the associated morbidity and mortality appear clinically either to be resulting from attacks of common respiratory diseases such as influenza, bronchitis etc., or from exacerbations of pre-existing chronic cardio-respiratory illnesses.<sup>4</sup> These are therefore not likely to be connected up with the abnormal pollutional states of the atmosphere unless such associations are specially being looked for. As a matter of fact, the recurring episodes of smog in London in winter were regarded by many as healthy and invigorating, before statistical and epidemiological studies during the severe and lingering smog of 1952 revealed that it had taken a toll of 4000 'excess' deaths from the population of the greater London area.

Polluted urban air may contain a very large number and variety of foreign substances, the individual concentrations and relative proportion of which is subject to great variations even within short periods of time depending upon a large number of factors. The types and intensities of human activities generating the pollutants, the types of fuel used for domestic, industrial, commercial, recreational or other pursuits; the physiography of the area and the prevailing meteorological conditions determine the pollutional state of the atmosphere. It is thus extremely difficult to objectively characterize atmospheric pollution over a given geographical area.

Very generally speaking, two broad types of polluted atmospheres may be described. In one of them, the London type, the products of coal burning viz., smoke and sulphur dioxide are the major constituents and in the other, called the Los Angeles type, pollutants from automobile exhausts viz., oxides of nitrogen, unburnt hydrocarbons and carbon monoxide are

the principal primary constituents. In the latter type, however, atmospheric reactions produce secondary pollutants such as ozone and other 'oxidants' the presence of which in relatively large concentrations characterizes it.<sup>5</sup> In other places, special types of industrial activity may contribute some specific pollutants which are unimportant or absent in the atmosphere of other areas.

Since the major human activities, industrial or domestic are more likely to generate atmospheric pollutants from the burning of coal, most epidemiological observations on the effects of atmospheric pollution on health have been based on measurements of sulphur dioxide and smoke *i.e.* air-floated dark particulate materials in the air. Many studies have also derived indirect estimates of likely intensities of atmospheric pollution in different localities from annual consumption of coal and other fuels and the density of populations.

However, although sharp increases in cardio-respiratory morbidity and mortality have been well-correlated by scientific observations with the acutely polluted states occurring from time to time in different parts of the world, it has not yet been possible to positively incriminate any of the pollutants either singly or in combinations as the causative agencies. They are each present in such small concentrations, even under conditions of acute pollution, that none could possibly be responsible for the observed effects. It, however, seems possible that some synergistic action of sulphur dioxide and the particulate materials in the air may be largely responsible for these. Reid<sup>6</sup>, for example, compared the respiratory mortality during two acute air pollution episodes in London occurring in 1952 and 1962. The mortality was much lower during the latter episode, although the average sulphur dioxide concentration was higher. But in the intervening period vigorous action for abatement of smoke had reduced considerably the air-floated particles in London air. Amdur<sup>7</sup> has also produced some experimental evidence for such synergism between sulphur dioxide and respirable particles in air.

But, naturally, the principal concern of enquiries on the effects of atmospheric pollution on health were eventually focussed on the question, what if any, were the long term repercussions of living in atmosphere in which the atmosphere is chronically polluted *i.e.* where the measurable pollutants are constantly present but in much lower concentrations than those obtained during the rare acute episodes mentioned earlier. Such enquiries are extremely

pertinent, for an ever increasing proportion of the world's population elect to live in such atmosphere namely, those of industrial townships and the cities.

So far as the respiratory tract was concerned, the exposure over long periods to pollutants in air, most of which have irritant properties, would be expected to insidiously produce a state characterized by hypertrophic and degenerative changes of its parenchymal tissues associated with manifest functional difficulties mainly in the middle and old ages due to the cumulative effects of minute repeated traumata of the pollutants. Chronic bronchitis and emphysema are conditions which admirably fulfil the above requirements. In U.K. these have gradually become one of the commonest illnesses of the population, particularly in the age groups above 45. There is a well marked urban-rural difference in its prevalence. Even amongst urban areas, prevalence is higher in the cities than in the towns<sup>8,9,10</sup>. Mortality from bronchitis<sup>11,12,13</sup>, as also mortality from respiratory diseases as a whole<sup>14,15</sup> were correlated with the degree of urbanisation and atmospheric pollution. Exacerbations, and remissions of symptoms of bronchitic patients were also correlated with fluctuations of atmospheric pollution by Lawther<sup>16,17</sup>. Fletcher<sup>18</sup> in a 5-year follow up of a group of subjects in London reported a steady decline of amount of phlegm which paralleled the decrease of atmospheric pollution in the city as a result of the operation of the Clean Air Act.

On the other hand, the prevalence of chronic bronchitis in other highly industrialised countries of Europe and North America is very much, even upto 15-16 fold, lower. It may however be that much of such differences can be attributed to differences in the diagnostic habits of clinicians in different countries so far as this ill-defined disease was concerned. Nevertheless, there has been noticed a distinct rise in the prevalence of chronic bronchitis during the two decades following the 2nd World War, in countries reporting low prevalence rates of the disease. Many authorities<sup>19,20</sup> are however inclined to regard cigarette smoking as the principal environmental factor determining the prevalence of this disease and mortality resulting from it. Prevalence and mortality of such diseases have been observed to be greater amongst the heavier smokers. Ex-smokers are affected to a greater extent than non-smokers but to a much lesser degree than the heavy smokers. A number of studies, such as those by Reid *et al.*<sup>6</sup> and Ferris and Anderson<sup>21</sup> show that the observed differences in the prevalence rates of chronic bronchitis amongst population exposed to different intensities of air pollution tend to disappear if the

rates are standardized for differences in the heaviness of cigarette smoking by the subjects.

To avoid the complicating factor of cigarette smoking a few studies were carried out amongst school children. Douglas and Waller's<sup>22</sup> studies on cohort children from different localities, revealed an increased occurrence of lower respiratory tract infection amongst children residing in areas with higher degrees of pollution. Lunn *et al.*<sup>23</sup> also found greater prevalence of upper and lower respiratory tract infection in children from localities with more polluted atmosphere. These authors carried out a follow up survey three years later<sup>24</sup> amongst the same group of children. It revealed that the differences in the degree of pollution in the areas from which the children were examined had been greatly reduced within this period due to anti-pollution measures, leading to reduction in the differences in prevalence of respiratory diseases among them observed in the earlier study.

In this context, it must be remembered that in the chronic obstructive disorders of the lungs the aetiology is multifactor. Age, sex, social class, room overcrowding and occupation all seem to have some measure of influence in their genesis. Infection is also one of the important factors whose relationship with the disease vis-a-vis atmospheric pollution is imperfectly understood. Smoking seems to have a very great role in determining the onset of the disease. But in spite of the evidence put forward here and a much larger number of others which could not be cited, the contribution of atmospheric pollution in bringing about chronic bronchitic disorders does not seem, to the present reviewer, to have been established unequivocally as yet.

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# NIACIN PRODUCTION TEST IN MYCOBACTERIA: REPLACEMENT OF BENZIDINE—CYANOGEN BROMIDE REAGENT BY o-TOLIDINE—CYANOGEN BROMIDE

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## Introduction

The identification of *M. tuberculosis* depends primarily on the niacin production test. Runyon and others (1959) described a method based on the observations of Konno (1956) using aniline as the reagent. However, the aniline reagent gives a yellow colour which can cause difficulty in the interpretation of the results, particularly in the case of the chromogenic mycobacteria. Hence several workers prefer the test employing benzidine (Medveczky, 1960) or o-tolidine (Gutierrez-Vazquez, 1960), since the pink colour produced in these tests is easier to read.

The standard method for niacin production test at this Centre has been the one using benzidine. However, satisfactory supplies of benzidine are no longer available, as the manufacture of this compound has recently been stopped. Hence it was decided to investigate the test using o-tolidine. Though other workers (Tarshis, 1960, 1961; Gangadharam and Droubi, 1971) have compared the benzidine and o-tolidine methods on small numbers of cultures, no large scale investigation of these two methods has been reported. Therefore a direct controlled comparison of these two methods was undertaken, the results of which are reported here.

## Material and Methods

A total of 560 cultures of mycobacteria was used for this comparison. These cultures formed part of a survey.

A standard suspension was prepared from each culture by shaking it with sterile distilled water and glass beads. One loopful of this suspension was inoculated on to a pair of Lowenstein-Jensen slopes and incubated at 37°C. At the end of four weeks, the two sets were given code numbers and processed. The investigation was carried out in three batches, using 150-200 cultures per batch. Both the tests were performed and read by the same person.

*Benzidine test* -- To approximately 0.25 ml of the autoclaved culture extract was added 0.25 ml of a freshly prepared 3% w/v solution of benzidine (E. Merck, GR) in ethanol followed by an equal volume of approximately 10% cyanogen bromide (saturated aqueous solution).

*o-tolidine-test* - - The procedure was essentially similar to that of the benzidine test except that the benzidine was replaced by a freshly prepared 1.5% w/v solution of o-tolidine (BDH Analar) in ethanol.

With both the tests, the formation of a pink or red precipitate was considered to be a positive reaction for niacin while a white or dirty-white precipitate was taken as a negative reaction. The positive results were graded as 1+ (faint perceptible pink precipitate) or 2+ (pink or red precipitate).

## Results and Conclusions

Of the 560 cultures tested (Table 1) 174 were negative and 380 were positive by both tests, that is, an agreement of 99%. Of the remaining six specimens, 4 yielded a positive reaction only by the o-tolidine method (1 was 1+ and 3 were 2+), and 2 by the benzidine method only (both 1+). It may be concluded that the efficiency of the o-tolidine method is very similar to that of the benzidine method in detecting niacin production.

Table 1

*Comparison of the benzidine and o-tolidine methods for the detection in niacin production in mycobacteria*

Benzidine	o-tolidine method			Total
	Neg.	1 +	2 +	
Neg.	174	1	3	178
1 +	2	1	0	3
2 +	0	6	373	379
Total	176	8	376	560

## Summary

The benzidine and o-tolidine methods for niacin production were compared on 560 cultures. There was an excellent agreement (99%) between the two methods.

## ACKNOWLEDGEMENT

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## DIABETES AND TUBERCULOSIS

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The frequency and enhanced severity of infections in uncontrolled diabetes were well known before and after the discovery of Insulin. The availability of antibiotics has made a great difference, but infection is probably a more serious threat to life in a diabetic than in the non-diabetic.

The association between tuberculosis and diabetes was noted by Avicenna<sup>1</sup> more than a thousand years ago. About a hundred years ago, half the diabetic patients who died were found on post-mortem to be suffering from pulmonary tuberculosis. Tullock<sup>8</sup> in 1962 reported that the incidence of tuberculosis in diabetics in underdeveloped countries has varied from 0.9% in Jamaica to 14% in Hongkong, 2.8% in Africa, 5.8% in Indians in Natal. Association between diabetes and tuberculosis has been reviewed by Deshmukh and his colleagues<sup>3</sup>; he has reported the incidence of pulmonary tuberculosis to be 8.3 % in diabetics. The proportion of diabetics was 11 % in 825 patients suffering from pulmonary tuberculosis over the age of 40 years. The incidence of tuberculosis in this country has been reported by various workers, 63 out of 1,882 diabetic cases in Bengal,<sup>8</sup> 157 out of 720<sup>s</sup> cases in South India.<sup>8</sup> Nanda and Tripathy (1968)<sup>7</sup> found 2.4% tuberculosis amongst 200 diabetics. Dingley (1969)<sup>5</sup> observed the incidence of tuberculosis amongst diabetics about five times more than in the general population. Lahiri and Sen (1974)<sup>9</sup> found the incidence of tuberculosis with diabetes 1.2 % in the age group between 20 to 39 years, 5.4% in age group of 40 to 59 years and 0.8% in 60 years, and over. Bahulkar and Lokhandwala (1975)<sup>2</sup> have reported an incidence 7.8 % in 400 diabetics which is four times more than that seen in the general population.

The mortality rate of diabetes with tuberculosis has been reported as ranging from 100% in 1955 to 11 % in 1958.<sup>9</sup> The majority of workers have reported mortality between 40 to 60 % in developed countries.<sup>6</sup>

### Method and Material

We have analysed 4,349 cases of diabetes admitted in the Bombay Hospital between 1967 to 1974. From the records of the case papers, we found 251 case records showing infection of tuberculosis *i.e.* an incidence of 5.77%. Tuberculosis affecting the organs have been listed in Table I.

Table I

### *Organs affected by Tuberculosis*

Organs Affected	No. of cases	% Incidence
Lungs	179	71.2
Meninges	22	8.8
Tuberculoma (Brain)	2	0.8
G.I. Tract	12	4.8
Fistula-in ano	6	2.4
Spine and bones	19	7.6
Lymph glands	7	2.8
Pericardium	2	0.8
Urinary bladder	1	0.8
Fallopian tubes	1	0.4
	251	100

Pulmonary tuberculosis was associated with two cases of tuberculous meningitis, one case of ileocaecal tuberculosis, one case of tuberculosis of the lumbar spine and one case of tuberculosis obstruction of the small intestine.

### Age and Sex Incidence

Of the 251 cases, 172 were males and 79 were females. The male/female ratio was 2.15 : 1. The majority of patients were between 51 and 60 years of age, as shown in Table II.

These patients were admitted for symptoms pertaining to tuberculosis or diabetes and in the course of investigation the presence of either diabetes or tuberculosis was discovered. Five cases were admitted in diabetic coma and later were found to be suffering from tuberculosis.

### Duration of Diabetes

Out of the 251 cases, 146 were known to be

Table II Age incidence of  
Diabetes with **Tuberculosis**

Age in years	Number of patients
Below 10 years	1
11—20 years	3
21—30 years	11
31—40 years	32
41—50 years	67
51—60 years	76
61—70 years	46
71—80 years	14
81 years and above	1
	251

diabetic, while 105 were diagnosed to be suffering from diabetes on investigation. The duration of diabetes on admission is shown in Table III.

Table III Duration  
of Diabetes

Duration in years	No. of cases of tuberculosis admitted (1967 to 1974)	Total no. of diabetics	% incidence of tuberculosis
Diabetes of less than 1 year duration			
113	2206		5.12
1_5 years	34	510	6.7
6—10 years	16	397	4.0
11—15 years	16	214	7.5
16—20 years	11	115	9.6
21—30 years	3	47	6.4
Duration unknown	58	860	6.74
Total	251	4349	5.77

The duration of diabetes of 55 cases was not known due to lack of records. When the percentage incidence was calculated from the total number of diabetics with a similar duration of diabetes, it was seen that the incidence of tuberculosis was not related to the duration of diabetes.

Of the 251 cases, 179 cases were found to have pulmonary tuberculosis. There were 135 males and 44 females, the maximum number of cases, as shown in Table IV, occurred in the age group 50-60 years. 22 cases were found to have tuberculous meningitis. There were 12 males and 10 females. The number of female cases were nearly equal to the males. The maximum number of cases, as shown in Table IV, occurred in the age group 31 to 40 years. 19 cases were found to have tuberculosis of the spine or bones; of these 17 cases were tuberculosis of the spine, 1 case tuberculosis of the wrist joint, 1 case tuberculosis of the ankle joint. The maximum number of cases of tuberculosis of the bone as shown in Table IV, occurred in the age group 51 to 60 years.

We have also graphically represented the incidence of the cases of pulmonary tuberculosis, tuberculous meningitis and tuberculosis of the spine or bone according to age and from graph No. I, it is evident that in pulmonary tuberculosis the highest incidence is in patients aged 51-60 years. The curve starts to rise from the age of 30 years and decline from the age 61 years. Prevalence of tuberculosis in diabetes and prevalence of diabetes in general population is in the same age groups as can be seen in the graph except tuberculous meningitis occurs earlier. Diabetes usually occurs in the middle age, but tuberculosis is a disease of young adults and our graph shows the incidence rising from 30 years. So there must be an association between tuberculosis and diabetes. Tuberculous meningitis shows a peak in the 31-40 group, earlier than pulmonary tuberculosis but also has small rise between 51 to 60.

Appropriate treatment was given to all patients for diabetes as well as tuberculosis.

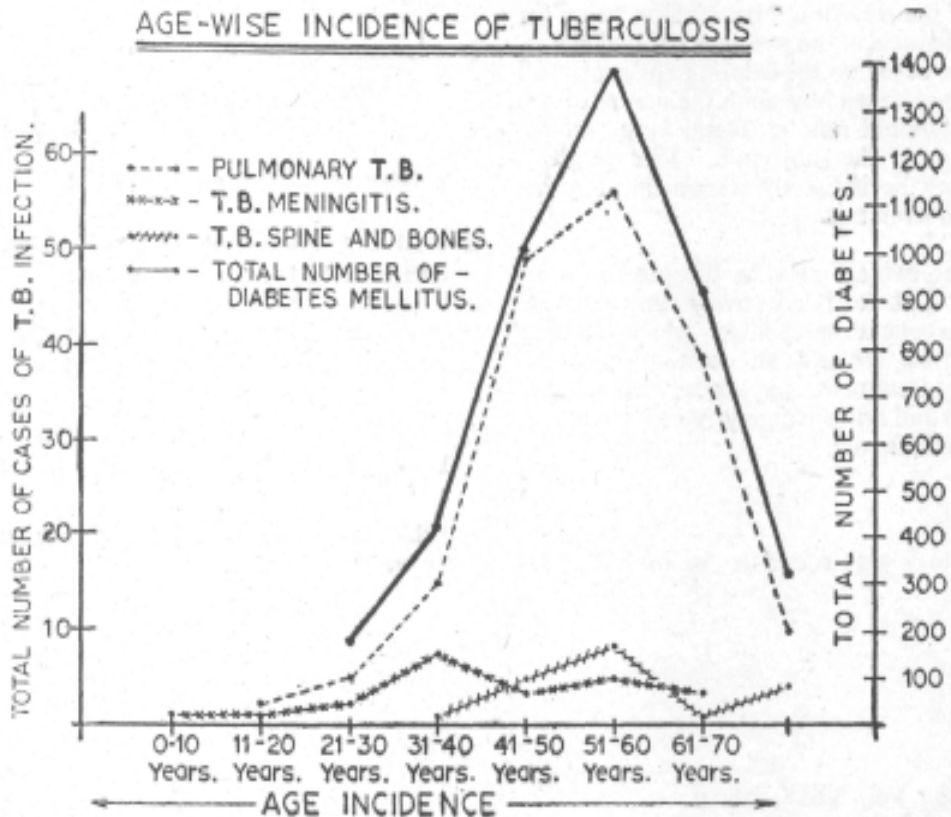
The mortality rate in our series was 12.35%, i.e. 31 deaths.

### Discussion

The incidence of tuberculosis infection with diabetes in 4,349 diabetics admitted in the Bombay Hospital was 5.77%. Out of these, infection of the lung formed 71.2%, tuberculosis of the meninges was the next common cause, 8.88%, tuberculosis of the spine or bone comprised 7.6%, that of the gastro-intestinal tract was 4.8%. Tuberculosis of other organs was found in one or two cases.

Table IV  
Age and Sex Incidence of Pulmonary Tuberculosis

	0-10 years	11-20	21-30	31-40	41-50	51-60	61-70	71-80	81 years & above	Total
Male	—	—	2	13	36	50	30	4	—	135
Female	—	2	3	2	14	7	9	6	1	44
Total	—	2	5	15	50	57	39	10	1	179
Age and Sex Incidence of Tuberculosis Meningitis										
Male	1	—	—	4	2	3	2	—	—	12
Female	—	1	2	3	1	2	1	—	—	10
Total	1	1	2	7	3	5	3	—	—	22
Age and Sex Incidence of Tuberculosis of the Spine or Bone										
Male	—	—	—	—	2	8	—	3	—	13
Female	—	—	—	1	3	—	1	1	—	6
Total	—	—	—	1	5	8	1	4	—	19



The salient findings were: the majority of cases were between 40-60 years, males predominated, and the duration of diabetes in majority were between one year to five years.

Antidiabetic treatment was mostly instituted but occasionally the treatment of diabetes had been ignored by the physician in the hope of alleviating the disease by instituting treatment of tuberculosis, but most patients received insulin alone or in combination with sulphonylureas or a biguanide. However, it was observed that quite a number of patients changed to the oral drugs on discharge from hospital regardless of the duration and severity of diabetes or the effectiveness of oral drugs. This was usually done at the request of the patients, who were unwilling to continue the injections of insulin under the misguided belief that oral hypoglycaemic agents are as effective as insulin, the physicians succumbing to the persuasion by the patients. The physician should adequately assess the effectiveness of oral treatment before agreeing to discontinue insulin.

In the treatment of tuberculosis uptill now a high caloric nourishing diet has been followed. Is this indicated in a patient of tuberculosis complicated by diabetes? It has been shown by W.H.O. experiments that diet does not play an important part as an adjunct in the treatment of tuberculosis as it was in the pre chemotherapy period. Higher caloric nourishing diet is still advocated by some of the physicians usually because of the traditional belief. Diet being an important feature in the treatment of diabetes—it should be based on the caloric requirement of the individual depending upon the nature of his work, diet should not be liberal even though containing all the nutrients. Uncontrolled diabetes may be indirectly responsible for the spread of tuberculosis.

It is our earnest plea to the physician to ensure that diabetes is effectively controlled by oral antidiabetic drugs failing which, insulin should be used. We have noticed that failure of antidiabetic treatment may cause spread of tuberculosis and bring in agony and spread of the disease in the family.

#### Summary

Tuberculosis was found to be in 5.77% in

4,349 cases of diabetes. The largest area affected was lungs in 71.2%. The next common site was meninges in 8.8%. Less frequent areas were bones or joints, least was gastro-intestinal tract. The commonest age group was 40-60 years. The largest number of cases occurred when the duration of diabetes was less than 5 years. The importance of correct treatment of diabetes has been stressed. The physician should not fall a prey to the patients' pleading for oral drugs in a situation where they are ineffective. Diabetic treatment should be that required for a diabetic.

#### ACKNOWLEDGEMENTS

We thank the Superintendent, Bombay Hospital and the Board of Trustees, Bombay Hospital Trust, for allowing us to utilize the hospital records of the cases included in our series and for financial assistance.

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## A STUDY OF MYCOTIC FLORA OF RESPIRATORY TRACT IN PULMONARY TUBERCULOSIS

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### Introduction

Superinfection of the respiratory tract by mycotic organisms in chronic broncho-pulmonary disorders has been widely documented (Khan *et al* 1959, Mittal *et al* 1959, Muktabhai 1970 and Shome *et al* 1976). The problem has also been highlighted in pulmonary tuberculosis too (Beatty and Saliba 1963, Sandhu *et al* 1964, 1966, Chakravarti *et al* 1964, 1967, and Shome *et al* 1974, 1975 & 1976). Commonest pathogens identified are *Candida albicans* (9.2 % by Shome *et al* 1976), *aspergillus fumigatus* (Jha *et al* 1974) no-cardia asteroids (3.7% by Shome *et al* 1976) and cryptococcosis (.75% by Jha *et al*).

The prevalence of the mycotic superinfection (or the infection) has been found to be related to a large number of variables, the most important of which, no doubt, is the geographical location. Other factors include the nature of basic pulmonary lesion, presence of cavities and their size (Sandhu *et al* 1966, B.T.A. 1968, and Misra *et al* 1971) and the nature and duration of the chemotherapy received by the patient in the past (Wood *et al* 1951, Browne 1954, Elinger 1957 and Shome *et al* 1974, 1976).

Mycotic superinfection may be present as a coincidental finding or may affect the progress of the lesion and its response to chemotherapy (Minikiewiez and Limak 1960, and Shome *et al* 1976).

The present study was undertaken to elucidate this aspect of the problem in tuberculous patients admitted to Kasturba T.B. Hospital.

### Material and Methods

110 cases of pulmonary tuberculosis admitted to Kasturba Tuberculosis Hospital were selected for the study/The patients according to the history of previous chemotherapy, were divided into two sub-groups e.g. untreated (who had no or less than 2 weeks of chemotherapy) and treated (these who had over 2 weeks of antituberculosis chemotherapy. There were 38 cases in the former group and 72 in the latter.

All the patients were asked to provide 6 samples of sputum (collected at 48 hours interval). These samples were examined by smear examination after staining the smears with 10% KOH,

India ink, Gram's and Ziehl-Neelsen's stain. All these samples were cultured on the sabouraud's media with and without antibiotic. Identification of the fungi was done by fermentations and by growth characteristics on cornmeal media.

### Observations and Results

Table 1  
*Comparison of direct microscopy of sputum smear and culture results*

Cases screened	No. of (+) by direct microscopy	% by culture	No.of (+)	V /o
NO	36	34 .34	40	36.36

Table 2  
*Table showing species of fungus isolated by fermentation and by growth on corn meal*

Species	No. of cases	V
Candida Albicans	25	62.5
Candida Krusei	2	5
Candida Stellatoides	2	5
Asp. Fumigatus	3	7.5
Asp. Sulphuricus	3	7.5
Asp. Niger	3	7.5
Other species	2	5

Table 3  
*Table showing percentage of fungal positive cases in treated and untreated cases.*

Group	No. of fungal positive cases	%
Untreated group	12	30
Treated group	28	70
Total	40	100

Table 4

*isolation of fungal organism in relation to duration of past anti-tubercular drug therapy*

Duration of therapy in months	Total No. of cases	Total positive cases	%
1—6	36	13	36.11
7—18	22	9	40.9
18—onward	14	6	42.85

Table 5

*Table showing comparative incidence of Aspergillus isolation in cavitory and non-cavitory cases.*

	No. of cases	No. of Aspergillus positive cases	%
Cavitory	54	7	12.96
Non-cavitory	56	4	7.14

### Discussion

Pulmonary tuberculosis is essentially a chronic destructive disease of the lungs. Caseation, necrosis and fibrosis tend to lead to the formation of cavities and bronchiolectatic dilations. These destroyed areas of the lung will no doubt continue to remain in the body even after the tubercle bacilli has been totally eliminated. The cavities form an ideal culture plate for the tubercle bacilli and for many other organisms including the fungi by providing plenty of oxygen and necrotic tissue material.

The fungal organisms tend to settle in these cavities and destroyed dilated bronchi, as a rule, after the tubercle bacilli has disappeared from these areas. However, the prolonged chemotherapy in tuberculosis, which may last for well over two years with or without corticosteroids by itself, becomes a potent predisposing factor for the onset of super-infection by the fungal organisms (Wood *et al.*, 1951; Browne, 1954; Sharp, 1954; Bartland and Halton, 1954; Seligman, 1959; and Sydransky and Pearl, 1961).

Therefore, it is not surprising to come across

frequent references in the literature regarding the association of mycological super infection with active pulmonary tuberculosis (Beatty and Saliba, 1963; Shome *et al.*, 1969; Sandhu *et al.*, 1964, 1966; Chakravarti *et al.*, 1962, 1963, 1964, 1967).

The frequency and the type of organisms isolated has varied from workers to workers. This is also likely to be affected by the specimen selected for the examination. It is a well known fact that 10 % of normal people will have *Candida albicans* in their throat. (Bansal, 1973). The percentage increases after antibiotic therapy (Wood *et al.*, 1951; Browne, 1954). The same has been found to be more or less true for *Aspergillus* infection too.

What conventionally is called sputum in a tuberculous patient is mixture of necrotic material from cavities, bronchial mucous secretion and debris from respiratory passages. As this "sputum" passes from lower respiratory passage to the upper, it gets contaminated by the organisms residing in the upper respiratory passages which, as a rule, even in a normal person contain normal resident flora. It is these organisms which are cultured from the sputum of any individual. So far, it has been a matter of great concern to the chest physician because sputum does not reflect the bacterial flora of the lower respiratory passages faithfully. Consequently, in an attempt to isolate only the pathogenic organisms from lower respiratory passages, bronchial washings and bronchoscopic material have been studied with better results compared to sputum. Naturally the yield of superinfecting organisms including the fungi from the sputum will depend on many factors which may be enumerated as under :

1. extent of pulmonary destruction,
2. chronicity of lesions,
3. duration of chemotherapy received,
4. administration of debilitating agents like corticosteroids and antimetabolites and x-ray therapy,
5. Type of specimen studied.

36.36 % of our cases with proved pulmonary tuberculosis yielded fungal organisms on sputum culture. We have been able to obtain only few references from amongst Indian studies relating to this aspect of the problem. Bansal (1973) obtained positive culture in 39.4% patients; a figure which is in close correlation with our observations. However, Shome *et al.* (1976) could demonstrate the fungal organisms only in 18% cases. The specimen used by Shome *et al.* comprised of sputum, bronchial aspirate and bronchoscopic material. For reasons pointed above (vide

supre), no doubt, the bronchial aspirate and bronchoscopic material are likely to be more specific for pulmonary pathology than sputum itself. This might explain difference between our results.

*Candida* was obtained in 26.36 % of our cases and *Aspergillus* in 10%. Our study is in agreement with that of those obtained by Lakshmi *et al.* (1972) and Ravindran *et al.* (1974). However, Geral in 1960 isolated *Candida* in 60% of cases of pulmonary tuberculosis. Sobti (1974) reported isolation of *Aspergillus* in 40% cases from U.S.A.

The variation in the percentages obtained by these workers relating to this finding could easily be explained by the difference in the country of origin of these reports. There is no doubt, the prevalence of fungal infection in the various countries will vary with the environmental and the soil factor. The history of previous treatment taken by the patient is another important contributory factor to the same problem.

62% of the *Candida* species isolated from our cases comprised of *Candida Albicans*. This is in conformity with the reports from Jha *et al.* (1974) and from Shome *et al.* (1976).

The age group which was most affected by the fungal organisms in our series was 20-40 years. Lakshmi *et al.* (1972) and Shome *et al.* (1976) have also reported similar findings. This could be due to many factors, the most important of which appears to be that this is the age group when tuberculosis is most prevalent and patients are most exposed to stress and strain of life which might tend to reduce the host resistance.

The history of previous treatment had a positive bearing on the isolation of fungal organism. 70% of our patients where the fungal organisms were isolated, had been treated in the past; only 30% of our patient had no history of any previous treatments. Antibiotics, corticosteroids, antimetabolites and radiation therapy predispose to primary fungal infection (Wood *et al.*, 1951; Browne, 1954; Sharp, 1954; Bartland and Halton, 1954, S. Eligman, 1957). Shome *et al.* (1976) have also had similar experience.

The longer, the duration of past treatment, the more was the isolation of fungal organism in pulmonary tuberculosis. This finding of our study is in correlation with the study of Wood *et al.* (1951); Browne (1954) and Sharp (1954).

12.96% of our cases who had cavities and 7.14% of cases who did not have cavities on X-ray yielded *Aspergillus* in their sputum.

Cavitation, specially due to tuberculosis, particularly after the tubercle bacilli have been completely exterminated, no doubt, is predisposed to infection to *Aspergillus* organism. This has been confirmed by Research Committee of British Tuberculosis Association (1968), Sandhu *et al.* (1966) and Misra (1971).

83.33% of our cases, who were diabetic, yielded fungal organisms in their sputum. The predominance of fungal infection in diabetic patient has also been stressed by Jha *et al.* (1974).

### Summary

Sputum from 110 admitted cases of pulmonary tuberculosis was examined by smear examination and by culture for the presence of mycotic organisms 36.36% of the cases had fungii in their sputum, 10% had *Aspergillus* and the remaining *Candida albicans* (26.36%). The isolation of the fungii was more common in patients with larger cavities, diabetics and in these who had had prolonged antituberculosis chemotherapy.

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## NEEDLE BIOPSY OF PLEURA

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Commonly employed methods in the diagnosis of pleural effusion, viz. bacteriological, radiological, cytological, biochemical and animal inoculation, are time consuming and even then fail to confirm the etiology in 40% of cases. Pleural biopsy can be the most useful diagnostic test (Read, 1968) in these cases. Percutaneous needle biopsy of the pleura is a safe, simple and rapid means of diagnosis with good results. Various needles have been used for this purpose namely Vim Silverman, Cope, Abrams, Franseen, Carpenter, and Ballestro needles. Abrams in 1958 devised a biopsy punch, that has been used for pleural biopsy (Mestitz *et al.* 1957; 1968; Thiruvengadam *et al.* 1962; Agarwal *et al.* 1970; Deshmukh *et al.* 1972; Mital *et al.* 1974) and also for the peritoneal biopsy (Sarin *et al.* 1961, 1962, 1964; Mehrotra *et al.* 1964).

### Material and Methods

Pleural biopsy was done by Abrams needle in 70 cases of pleural effusion in whom the diagnosis could not be established through the other routine procedures. All the cases were well interrogated and thoroughly examined. Skiagram chest P. A. and lateral view was done to localise the effusion. Bleeding time and coagulation time was done in all cases prior to the biopsy. Technique used was that employed and described by Abrams (1958). Only one attempt was made in each case.

### Observations

Table 1  
*Showing tissue positivity*

Total No. of cases	Adequate tissue		Failure	
	No. of cases	Percent-age	No. of cases	Percent-age
70	62	88.6	8	11.4

### Discussion

Abrams biopsy punch was used for doing the pleural biopsy in 70 cases of pleural effusion in which the etiological diagnosis could not be

Table 2

*Showing specific lesion in pleura! biopsv.*

Diagnosis tissue age	No. of Specific lesion			Non-specific lesion	
	No. of cases	Percent-cases	cases	No. of Per-cases	centage
Tuberculosis	38	25	65.8	13	34.2
Malignancy	15	9	60.0	6	40.0
Pyogenic empyema	8	6	75.0	2	25.0
Effusion secondary to hypoproteinaemia	1			1	100
Total	62	40	64.5	22	35.5

Table 3

*Showing the complications*

Complication cases	No. of	Percent-age
Major		5.7
Minor		
Small pneumothorax	4	
Subcutaneous emphysema	6	8.6
Total	10	14.3

established by other routine procedures. An adequate tissue for Histopathology was obtained in 62 out of 70 cases (88.6%). Mestitz *et al.* (1957), in their series of 116 cases, could get adequate tissue in 107 cases (92%). Thiruvengadam *et al.* (1962), Pagel *et al.* (1960), and Agarwal *et al.* (1970) have reported a tissue positivity of 95% (95 out of 100 cases), 100% (all 26 cases) and

84.5% (49 out of 58 cases) respectively. Deshmukh *et al* (1972), in the single specimen method could get adequate tissue in 23 out of 25 cases (92 %) and by the multiple specimen method it was 100 % in 25 cases. Thus the tissue positivity has varied between 74 % and 100 %. In our cases it was 88.6% by single specimen method which stands well in comparison. The positivity could have been appreciably higher had multiple specimens been taken during biopsy as described by Deshmukh *et al* (1972).

In the present study, a specific diagnosis could be established in 40 out of 62 tissue positive cases (64.5 %) or 57 % of the total of 70 cases. Mestitz *et al* (1957) diagnosed 62% of their 116 cases. Pagel *et al* (1960) could establish histological diagnosis in 13 out of 26 cases (50%). Thiruvengadam *et al* (1962), Raj Kondawar *et al* (1963), Agarwal *et al* (1970) and Deshmukh *et al* (1972) could make a specific diagnosis in 51 % (51 out of 100 cases), 32.3% (42 out of 130 cases), 20.6% (12 out of 58 cases), and 56 % (16 out of 25 cases) respectively. Hence the specific diagnosis in series of other workers has varied from 20.6% to 72%, which compares well with our results. Specific diagnosis of tuberculosis could be made in 25 out of 38 cases (65.8%), malignancy in 9 out of 15 cases (60 %) and pyogenic empyema in 6 out of 8 cases (75%). Rajkondawar *et al* (1963) diagnosed tuberculosis in 80% cases and malignancy in 75% cases. Agarwal *et al* (1970) found tuberculosis in 22 % and malignancy in 25% cases.

Complications were mild and self limiting and required no specific management. Four cases (5.7%) had small pneumothorax and 6 cases (8.6 %) had subcutaneous emphysema. No major complications were reported by other workers.

In conclusion pleural biopsy by Abrams needle was found to be the most effective rapid and safe diagnostic procedure in cases of pleural effusion where diagnosis is difficult to establish by other means.

#### Summary

Punch biopsy of pleura was performed by

Abrams needle in 70 cases of pleural effusion where diagnosis could not be established through other routine procedures. An adequate tissue for histological study was obtained in 62 cases (88.6%) and a histological diagnosis was established on 40 cases (64.5%). No major complications were encountered.

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## CASE REPORTS

### CAVITATING PULMONARY HODGKIN'S DISEASE

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#### Introduction

The incidence of involvement of lung parenchyma in Hodgkin's disease varies greatly. Verse (1931) found pulmonary lesions in 40% with generalised Hodgkin's disease at autopsy. O'Brien and O'Brien (1954) found the incidence only 3.5%. Bouslog and Wasson (1932) reported a case of cavity formation following radiotherapy. Cavitation without previous irradiation has been reported by Hardein (1939); Efskind & Wexels (1952), Wolpave *et al* (1944) found cavitation in 1 out of 22 with pulmonary disease, while Dickson and Smitham (1952) reported in 2 out of 27.

Because of rarity of condition the case report is being presented here.

#### Case Report

D.K., 29 years married Hindu female was referred with history of cough with scanty sputum and recurrent attacks of haemoptysis of one year duration. 4 years back patient had observed a small swelling of the size of a cherry in the right inguinal region which gradually increased to the size of a moderate sized potato. A provisional diagnosis of lymphosarcoma was made and she was given 50-60 vials of Inj. Endoxan i.v. The swelling burst out and healed within 3-4 months. She remained well for some time and then noticed a small swelling in the left inguinal region which gradually increased to the size of an orange. The swelling resolved itself but led to the development of multiple beaded swellings. Biopsy of the inguinal gland revealed the diagnosis of lymphosarcoma. 4 months later she developed cough with little sputum and recurrent scanty haemoptysis. She had been in good health except for slight feverishness in the evening hours and requiring no specific treatment. She had given birth to two children without any gynaecological problems.

Examination of the patient revealed a young lady of average built. Her general condition was fair and there was no evidence of dyspnoea, but had clubbing of fingers and pitting oedema over lower extremities. Liver was about 6 cm below the right subcostal margin, smooth and had slight tenderness. However, the spleen was not palpable. She had no ascites. Submandibular, posterior cervical and axillary lymphnodes on both the sides were palpable. The glands were of varying size and non-tender. Examination of the respira-

tory system showed only presence of fine crepitations in the right mid and the lower inter-scapular areas.

The sputum was persistently negative for acid fast bacilli and culture for pyogenic organism grew commensal organisms. Haematological examination showed leukocytosis and hypochromic anaemia, the total RBC count was 3 million/cumm, total leucocyte 18000/cumm with neutrophils 86%; lymphocytes 10%, eosinophils 3% and monocyte 1% and haemoglobin was 9.5 gm/100 ml (65%). Erythrocyte sedimentation rate was 40 mm after one hour (wastergreen). Bleeding time was 2 minutes 10 seconds and clotting time 3 mts. 20 seconds. Serum protein was 6.2 gm% with albumin 3.3 gms% and globulin 2.9 gms%. Aldehyde and chopra's tests were negative; urine analysis showed few epithelial cells and calcium oxalate crystals. Albumin was one plus. Biopsy of the axillary gland was suggestive of Hodgkins disease. Skiagram chest P.A.V. (Fig. 1) showed

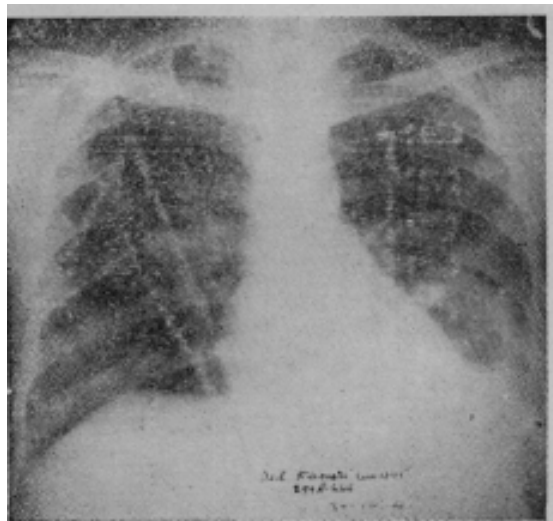


Fig. 1. Skiagram chest P.A.V. showing cavity in right parahilar region and left lower zone.

cavity on right parahilar region and left lower zone, and tomogram (fig. 2) at 11 cm from the table top showed cavity in the right upper zone-right parahilar area and left lower zone. Biopsy of the lung confirmed pulmonary Hodgkin's (fig. 3). Liver biopsy revealed portal cirrhosis.

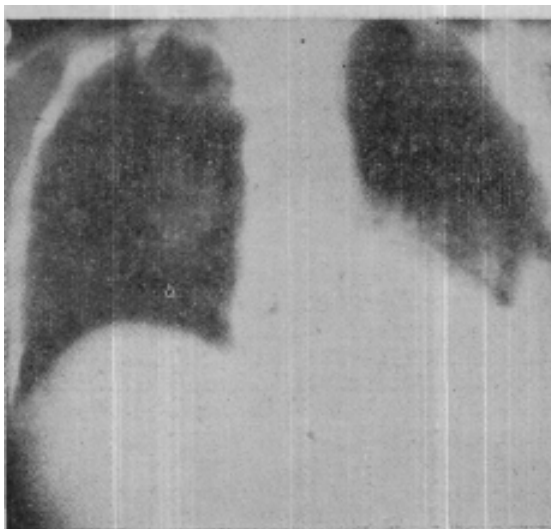


Fig. 2. Tomogram at 11 cm from table top showing multiple cavities on both the sides.

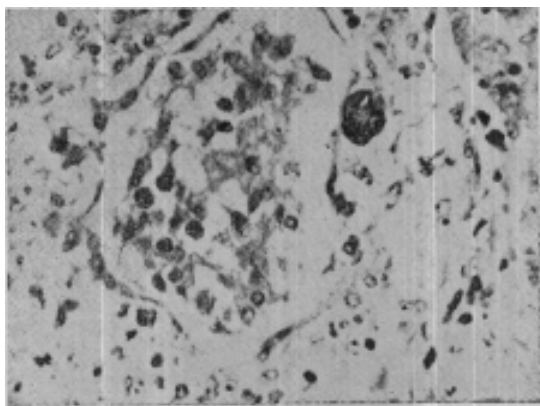


Fig. 3. Microphotograph of lung showing stereoburg reed-giant cells, reticulum and eosinophil cells.

### Discussion

Lymphomatous infiltration may rarely undergo cavernous disintegration of its own. Vieta & Graver (1941) reported cavitation in 3 out of 51 patients. Steel (1964) noted cavitation in 3 out of 14 patients prior to treatment while one more patient had cavities after radiotherapy.

Ellman & Boweller (1960) described radiological appearance in 6 cases with pulmonary lesions in Hodgkin's disease and one of which showed cavitation in terminal stages following treatment with radiotherapy and cytotoxic drugs. Holesh

(1953) reviewed 300 cases referred to radiotherapy clinic and stressed that cavitation might occur at the later stage. She described the tomographic appearance of cavities usually being irregular and craggy but occasionally smooth and thick walled.

### Summary

A case of cavitating pulmonary Hodgkin's confirmed on with history of recurrent scanty haemoptysis feverishness, hypochromic anaemia, and leucocytosis and biopsy is reported herewith.

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## ASSOCIATION OF MALIGNANCY OF OTHER ORGANS IN A CASE OF PULMONARY TUBERCULOSIS

K.C. MATHUR

(From S.P. Medical College, Bikaner).

Bayle in 1810 described 'Cavitation cancer-use' as one of the six types of tuberculosis and he is generally given credit for being the first to describe the coexistence of carcinoma and tuberculosis. Cases presenting coexistence of tuberculosis and cancer in the same organ namely, lungs are being reported in increasing number (Me Quarre *et al.*, 1968; Snider & Placik, 1969). But the case reports of malignancy of other organs associated with lung tuberculosis (Tarcenska, 1967 and Shorek, 1968) still seem to be very scanty in the literature.

### Case Report

J.D., male 50 years, was admitted on 4-5-1972 with the complaints of cough with moderate mucoid expectoration, exertional dyspnoea Grade II and anorexia of six months duration. For the last 40 years he smoked about 20 Bidiies a day and had been chewing Tobacco for about 9 months. Four years ago a small painless swelling, appeared at the angle of the right mandible, gradually increasing in size, most marked during last six months and becoming painful during the last 15 days. A pus discharging ulcer appeared at its upper and posterior aspect, followed by a number of small swellings which appeared in the right posterior triangle.

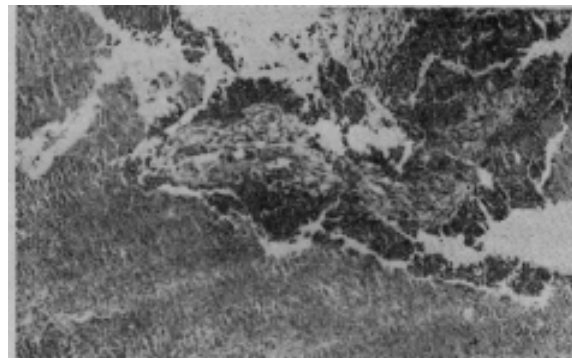
On examination, evidence of consolidation in upper half of right lung was noted. Local examination of the neck showed a diffuse lobulated, firm, tender swelling fixed to the skin and measuring 8 cm x 5 cm x 3 cm extending from the ear lobule to the anterior triangle of the right neck and was compressing the external auditory meatus so that only a slit was visible. There was an ulcer of 1 cm diameter on the upper and posterior aspect of the swelling and purulent discharge was coming out from this ulcer. In addition, several small swellings (lymphglands) measuring 1 cm to 4 cm in diameter were palpable in anterior as well as posterior triangles of the right side of the neck along the external jugular vein. These were discrete, firm, tender and mobile in the lower part but were matted and fixed to deeper structures and to the skin elsewhere. Adenopathy was not present in other part of the body.

Liver was palpable upto 2 cm below the right inferior costal margin. Its surface was smooth, firm and non-tender with rounded margins.

Otorhinological examination revealed no abnormality.

Skiagram of chest (PA view) revealed the presence of consolidation with a small cavity in the right upper zone of the lung. Sputum was found to be positive for AFB by direct smear. ESR was 36 mm at the end of first hour.

Histopathological examination of the biopsy specimen of lymphgland from lower part of right side of neck revealed evidence of malignant metastasis as well as of tuberculosis (caseation surrounded by epithelioid cells) seen in the same field under the oil immersion lens of the microscope (Plate 1).



Patient was treated with standard antitubercular chemotherapy. Deep X-ray therapy was given daily to the main swelling (Co-60-200r) till total of 5000 r was administered. With this swelling disappeared completely (leaving only tiny lymphnodes) and liver also regressed. Patient experienced marked symptomatic relief in his respiratory symptoms by 17-6-1972, when he was discharged from the hospital. Diagnosis of right sided pulmonary tuberculosis with tubercular cervical adenitis and carcinoma of parotid with metastasis in cervical lymphglands and liver was made.

### Discussion

Regarding the simultaneous occurrence of carcinoma and tuberculosis in the same organ,

Rokitansky (1885) and Pearl (1929) postulated antagonism between these two conditions. While Moak (1902) and Conlson and Bell (1929) did not support the theory of antagonism, patients with both diseases are now being seen (McQuarrie *et al*, 1968; Snider and Placik, 1969 and Snider, 1969). Cases with evidence of both tuberculosis and malignancy in lungs are being reported in increasing number (Schwartz, 1964; Greenberg *et al*, 1964; McQuarrie *et al*, 1968; Snider, 1969; Snider and Placik, 1969). Similar association in breast (Grege and Kienle, 1969; and Miller *et al*, 1971) and in tongue (Comoretto, 1968) have also been reported.

The present case report illustrated coexistence of tuberculosis and malignancy in different organs viz. lung and parotid gland and also in the same organ *i.e.* lymphgland. It was very interesting to note that the evidence, both of tuberculosis and malignant metastasis was present in the same lymphgland which could be seen simultaneously in the same field under the microscope (Plate 1). Miller *et al* (1971) reported a similar case of carcinoma and tuberculosis of breast. His first biopsy specimen from right supraclavicular lymphnode provided evidence of tuberculosis and second specimen taken after ten days revealed both adenocarcinoma and granulomatous inflammation. Grege and Kienle (1969) reported a case of ipsilateral axillary node tuberculosis and breast cancer.

With the present knowledge it may be concluded that coexistence of tuberculosis of lung and malignancy of other organs are merely coincidental. Such coexistence is likely to occur more frequently among elderly patients.

#### ACKNOWLEDGEMENT

Author is thankful to Prof. K.D. Gupta,

Principal and Controller, S.P. Medical College and Associated Group of Hospitals, Bikaner, for his kind permission to publish this case report.

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# POSTERIOR FOSSA SYNDROME CAUSED BY CYSTIC DILATATION OF THE THE CISTERNA MAGNA—A MANIFESTATION OF TUBERCULOUS MENINGITIS

I. DINAKAR

(From Kurnool Medical College,  
Kurnool)

The clinical manifestations of tuberculous meningitis are protean and many clinico-pathological varieties have been reported in the literature (Dastur, 1972; Udani *et al*, 1971). A midline cerebellar syndrome (Vermis syndrome) caused by a cystic dilatation of the cisterna magna due to posterior fossa meningitis has not been described hitherto. In the present report this entity is illustrated and the pathogenesis of the condition discussed.

## Case Report

A 5-year old boy was admitted for headache, vomiting and irregular fever of 2 months, duration. On examination the child was drowsy. He had bilateral papilloedema, trunkal ataxia and pyramidal signs in both lower limbs. Ventriculography revealed dilated lateral and third ventricles. The aqueduct and fourth ventricle were small and displaced forward (Fig. 1).



Fig. 1. Lateral view of ventriculogram, showing narrowing of fourth ventricle, displaced forwards.

During posterior fossa craniotomy the cisterna magna was enormously distended, its arachnoid thickened, opaque and studded with tubercles of pin head size. The wall (arachnoid) was excised. Further exploration did not reveal any space occupying lesion. The fourth ventricle was pushed anteriorly. The adjacent medial parts of the cerebellar lobes were compressed and separated. There were numerous adhesions on the basal aspect of both cerebellar hemispheres.

Histopathological study of the excised arachnoidal wall showed evidence of tuberculosis. The child did not fare well, and died on the eleventh post-operative day.

## Discussion

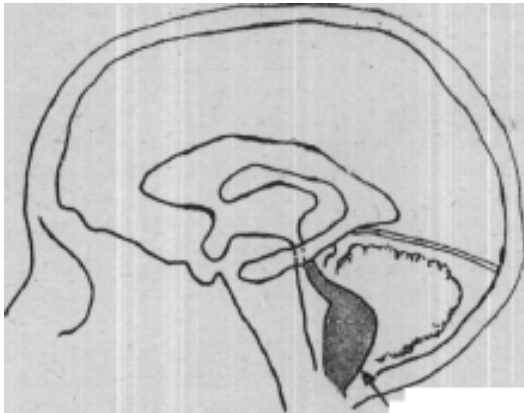
This case exemplifies yet another manifestation of tuberculous meningitis. In the common type of post meningitic hydrocephalus, the meningeal exudate and adhesions round the brain stem in the basal cisterns interfere with the circulation of CSF. A back pressure is thus built up resulting in a hydrocephalus in which the entire ventricular system including the fourth ventricle is distended (Dinakar, 1975). In extreme cases, a grossly dilated fourth ventricle acting as a midline cerebellar tumour is not a rare occurrence (Udani *et al*, 1971). In the case reported in this paper however, the fourth ventricle and aqueduct were compressed and pushed anteriorly, by a tense, distended cisterna magna acting like a space occupying lesion. The dense arachnoidal adhesions at the sites of the outlet of the cisterna magna is another feature.

Based on the ventriculographic and operative findings in this case it is inferred that the meningeal adhesions in this type of cases originate or predominate at the periphery of the cisterna magna, where the cisternal arachnoid becomes continuous with that covering the cerebellum, thus interfering with the drainage of the spinal fluid from the cisterna magna. The latter therefore becomes gradually distended and tense (Fig. 2). This severely distended cyst like cistern pushes the fourth ventricle forwards and compresses it and also causes kinking and forward displacement of the aqueduct simulating a vermis tumour.

There is no way of recognizing this condition pre-operatively. Surgical exploration alone delineates the condition. However, evacuation of the cyst does not appear to be an adequate measure. The adhesions distal to the cisterna magna remain unaffected. Hence a drainage (shunt) procedure is indicated in these patients.

## Summary

Cystic dilatation of the cisterna magna, and



Cyst  
ANATOMICAL RELATIONS  
(DIAGRAMATIC)

Fig. 2. Schematic diagram showing the position of the distended cisterna magna in relation to the fourth ventricle.

consequent vermis syndrome is yet another manifestation of the sequelae of tuberculous

meningitis. A case is reported to illustrate the clinical operative features of this entity and the possible pathogenesis of this condition has been discussed.

#### ACKNOWLEDGEMENT

I thank the Superintendent, Government General Hospital, Kurnool for permission to use the hospital records. My thanks are due to Mr. G. Prahlada Rao, Stenographer for secretarial assistance and Mr. P. Haricharanapathi, Photographer for the photography.

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## PULMONARY CANDIDIASIS

V. THIAGARAJAN, S. RADHAKRISHNAN, V. SIVARAJAN, R. RAJASEKAR and K.R. SRINIVASAN  
(From Thanjavur Medical College, Thanjavur)

This case is presented for its association with cell mediated immunodeficiency manifesting in the form of pulmonary candidiasis, lepromatous leprosy and primary myxoedema. Available reports associate candidiasis in altered hosts. A detailed clinical account of this disease with positive, laboratory findings is given. Pulmonary candidiasis as reported in the literature is analysed against the background of clinical manifestations as found in this case. A brief description of the literature relevant to this disease is also mentioned.

### Report of a Case

A 55 year old female was first seen on 20th September 1976 with low grade fever, cough with expectoration and progressive dyspnoea since 2 years.

### Past History

In 1946, she had hypothyroidism; B.M.R. was low; blood cholesterol was 352 mg%. E.C.G. showed low voltage complexes in all leads with flattening of T waves; rate 60 /mt. She was given 0.1 mg. of thyroxine which she took irregularly. In 1964, she developed depressive psychosis and was treated with anti-depressants (Imipramine & ECT). In 1966 she relapsed again and was treated accordingly. In 1974, she developed cough with expectoration, low grade fever with malaise. Sputum for AFB on consecutive examinations was negative. Chest roentgenography revealed diffuse infiltrative ill defined patchy opacities in the right mid and lower zones. She was on streptomycin, INH and Thiacetazone for a period of 3 months and later maintained on INH and Thiacetazone. Her symptoms persisted, inspite of regular drug therapy. In 1976 April, her respiratory symptoms increased markedly. She brought out substantial quantities of mupoid sputum tinged with blood. She had persistent low grade fever and marked deterioration of general health. Chest X-ray showed involvement of both the lungs including apices with diffuse infiltrative opacities and with patchy pneumonitis. She was started on Ethambutol, Rifampicin along with INH.

### Present Condition

On examination she was well oriented, cooperative, ill nourished, anaemic with no

significant lymphadenopathy. Patient had skin lesions consistent with lepromatous leprosy of 5 years duration proved later by histopathological examination. The nails were pale and hard. In the intertriginous areas, there were few itchy eczematous lesions. There was evidence of glossitis.

On examination respiratory system revealed prolonged expiratory phase with medium rales throughout the lung fields associated with expiratory wheeze heard well near the bases. The pulmonary second sound was loud and split normally. Other systems were clinically normal.

### Laboratory Investigations

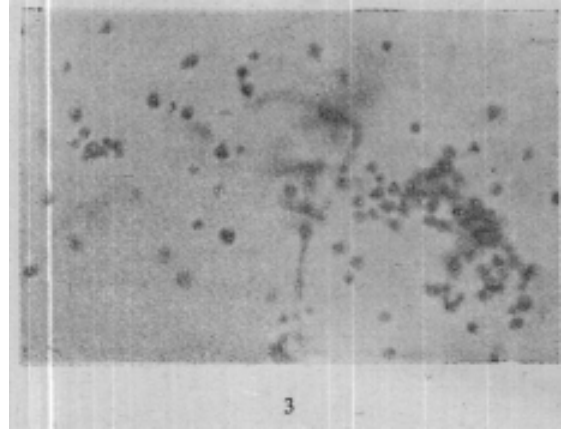
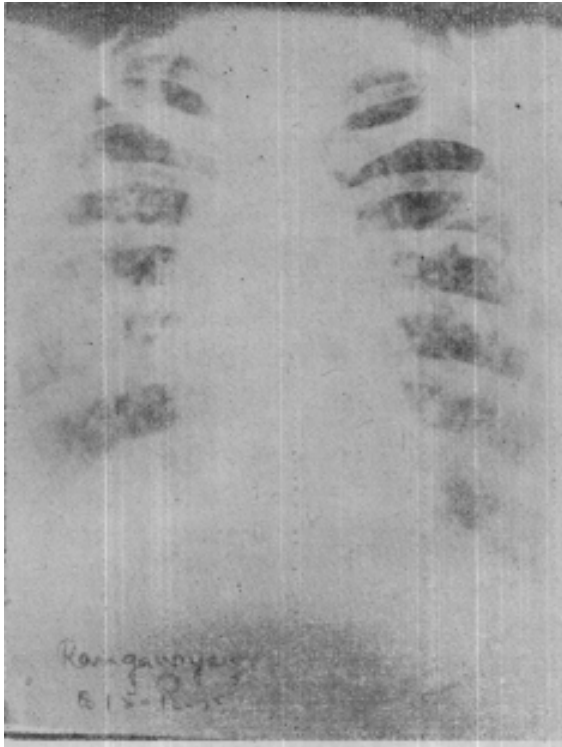
Blood Total Count	9400 cells/cumm.
Differential count	P <sub>70</sub> L <sub>24</sub> E <sub>6</sub>
R.B.C.	3 Million/cmm.
Hb	10gms%
Blood Cholesterol	325mgs%
Random Blood Sugar	100mgs%
Blood Urea	27mgs%
Protein Bound Iodine	3.10mcgms%
Urine : Albumin	Nil.
Sugar	
Mantoux	Negative to P.P.D. R.T. 23.
E.C.G.	Rate 55/mt. Low voltage complexes in all leads with prolongation of P-R. interval and flattening of T waves. Direct smear, concentration method and culture negative. Nothing contributory.
Sputum for AFB	
Culture for pyogenic organisms Agarase electro-phoresis	Showed a mild depression of albumin and slight elevation of globulin, globulins were markedly elevated.

### Fungus

- Direct smear of sputum with KOH showed budding yeast like cells.
- Bronchial lavage.
  - On direct smear with KOH showed the presence of pseudohyphae along with budding yeast like cell.

2. Culture on blood sugar, sabourauds, nutrient agar and corn meal agar *Candida* was grown and was proved to be *Candida albicans* by colonial morphology and biochemical reactions.

showed prominent yellow speckling of the kidneys. Culture of renal micro abscesses showed pure forms of *Candida albicans* confirmed from colonial morphology and biochemical studies.



#### *Histopathological examinations*

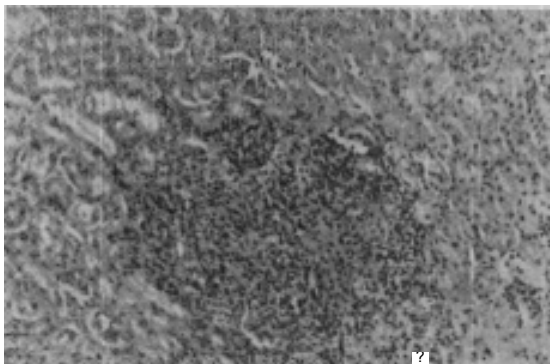
Cut section of the kidneys also showed tiny yellow dots in the renal cortex. Microscopically there were multiple micro-abscesses in the kidney and heart. Sections stained with Gram's stain revealed pseudohyphae forms of *Candida albicans* in these abscesses.

#### **Discussion**

Pulmonary candidiasis was suspected in this case by the presentation in the chest x-ray as diffuse infiltrative opacities<sup>6,19</sup> starting in the lower and mid zones of one lung, gradually progressing to involve other areas of both the lungs and mediastinum without calcification or cavitation in a course of two years, inspite of specific antituberculous drugs. Other evidences confirming the diagnosis are

1. absence of AFB in the sputum from the beginning.
2. repeated isolation of *Candida albicans*
  - a. in fresh specimens of sputum, b. bronchoscopic aspiration C. culture,<sup>6</sup> d. biochemical analysis,<sup>3,20</sup> e. animal pathogenicity<sup>8,9,20</sup> and
3. absence of bacterial infection (smear and culture).

Animal inoculation done in a healthy rabbit caused slow deterioration of health and activities and death on the 6th day. On autopsy the rabbit



To start with the patient developed hypothyroid state for which she had irregular and inadequate treatment and the condition being still persistent. Impairment of cell mediated hyper

sensitivity has been demonstrated in hypothyroid patients.<sup>14, 15, 18</sup> (Buchaner w.w. Anderson J.R.). Later she developed lepromatous leprosy proved by histopathological examination. Usually lepromatous leprosy is seen in patients with cell mediated immune-deficiency state (Ridley D.S., 1966; Bullock, 1968; WHO Report, 1969, Hassenclerger 1971; Lionel Fry P.P. Seah(1974)<sup>1, VW</sup>,<sup>22</sup> Hassenclerger and Buck(1963)demon-strated that the percentage of positive skin test to antigens of *Candida albicans* was significantly low among lepromatou's leprosy patients.<sup>1</sup>

In 1968, Bullock Jr. and coworkers confirmed that selective anergy to tuberculin and non myco-bacterial proteins does exist in lepromatous patients.<sup>1</sup> Candidiasis has been described as a common complication of long term antibiotics, antimetotics, cortico steroid therapy and diabetes mellitus (Crofton)<sup>11</sup> which were absent in this case. Hence manifestation of candidiasis in this case could also be accounted as has occurred in a state of cell mediated immunodeficiency caused by a lack of T. lymphocytes.<sup>21</sup> The electro-phoretic pattern of this patient's serum rules out a pathology of immunodeficiency disease mediated through B-lymphocytes, as evidenced by the presence of markedly raised and globulin fraction.

T lymphocytes are responsible for the cell mediated immunity. B lymphocytes are responsible for humoral immunity. This has been proved by Good R.A. and subsequently by Richard A. Chilgren *Etal.* He has clarified the cellular immune defect in chronic mucocutaneous candidiasis. (Lancet I: 1286-88, 1969) by means of skin tests using *Candida albicans* antigen (Hollister-Stier) and P.P.D. in patients suffering from mucocutaneous candidiasis. They injected these antigens to the patients suffering from mucocutaneous candidiasis. There were no reactions. Subsequently, these patients were injected  $2 \times 10^8$  eluted cells (97% Lymphocytes) into multiple sites in both arms, intracutaneously and subcuta-neously. The tests were repeated and turned out to be positive. This is a proof to say that T. lymphocytes which were responsible for cell mediated immunity, were deficient in patients with candidiasis.<sup>13</sup>

In our case also, the skin tests with P.P.D. repeated on two occasions were negative. This could be accounted, probably as due to deficiency of cell mediated immunity caused by the lack of T lymphocytes.

### Conclusion

As seen in world literature, cases of mucocutaneous candidiasis have been reported in combina-

tion with defective cell mediated immunity. Some of them were also associated with endocrinopathies like hypoparathyroidism, hypothyroidism, hypoadrenalism and diabetes mellitus. (Richard A. Chilgren *et al*)<sup>2, 6, 12</sup>. So far to our knowledge no case has been reported with a combination of cell mediated immuno deficiency with pulmonary candidiasis, lepromatous leprosy and primary myxoedema. Mediastinal involvement in candidiasis even though infrequent<sup>6</sup> is present in our case. This case is presented for its rare combinations.

### ACKNOWLEDGEMENT

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BOOK REVIEW

**SWEDISH TOBACCO POLICY; FRIDE ANTONI; CURRENT SWEDEN; 168; JULY 1977.**

The Swedish National Board of Health & Welfare appointed a Commission in December, 1971 to review the evidence concerning harmful effects of smoking and to suggest measures for reducing the health hazards involved. The salient recommendations of the Tobacco Commission are as follows:

1. The goal of current tobacco policy should be to bring down the cigarette consumption to the level of 1920.

2. People coming in close contact with children must be informed collectively and individually about the harmful effects of smoking.

3. A restriction of the right of employers to engage smokers for certain tasks or for work on premises where the risk of disease was exceptionally high.

4. A reduction in the maximum allowable concentration of all constituent substances of tobacco smoke.

5. Smoking should be prohibited on working premises and smoking should be gradually excluded from public premises.

6. Consideration should be given to the introduction of higher premiums for life insurance of smokers.

7. Tobacco products should be less readily available. Advertisements for such products should be done away with.

8. The prices of tobacco products should be systematically increased annually.

9. Sale of tobacco products through slot machines be discontinued by 1979.

10. A total ban on the sale of tobacco products to persons under 16 years of age.

11. Discontinuation of sales of tobacco products in food stores and other places selling other commodities primarily.

12. A compulsory health warning on all packages. A repertoire of warnings should be decided and these warnings should be frequently changed to attract consumers attention.

13. Rules should be framed against scenes in films and television programmes liable to encourage cigarette consumption.

14. Popular lectures by educational and welfare organisations on the hazards of smoking.

*N.B.* Cinema advertisements, outdoor advertising and direct advertising have been stopped as a result of an agreement between tobacco industry and the Consumer Ombudsman.

**S.P. Pamra**

## NEWS AND NOTES

### NATIONAL CONFERENCE

The Governor of Kerala will be inaugurating the 32nd National Conference on Tuberculosis and Chest Diseases on the 23rd November at 5.30 P.M. at Trivandrum, Kerala. Dr. K.V. Ksishnaswami, Director, Government Chest Institute and TB Training and Demonstration Centre, Madras, is the President of the Conference.

The main subjects for discussion at the Conference include: Epidemiology of Tuberculosis in India, National TB Control Programme, Panel Discussion on Role of Surgery in the Management of Respiratory Diseases, Chemotherapy including short-term regimens, Tuberculosis in Industry, Immunology of Tuberculosis etc.

Dr. C.W.L. Jeanes, Special Adviser, Health and Population. Canadian International Development Agency, Ottawa (Canada) will be one of the guest speakers. He will speak on "National TB Control Programme, including the role of TB Associations and other voluntary organisations in the working of the programme". Prof. G. Daddi, former Director of the Forlanini Institute, Rome (Italy) and Prof. B. Mariani, Prof. Daddi's successor, will be presenting papers on "Refampicin revisited after 10 years" and "Recent trends in TB therapy" respectively. Dr K.S. Sanjivi, Prof. Emeritus in Medicine, Medical College, Madras, will give a popular lecture on the opening day of the Scientific Sessions.

### TB SEAL CAMPAIGN

Prime Minister Morarji Desai inaugurated the 28th TB Seal Campaign at his residence on 2nd October, 1977 by making a token purchase of TB Seals for Rs. 101/-. The function was organised by the Delhi TB Association.

### Andhra Pradesh

The Campaign was inaugurated on 2nd October by Shri M. Manik Rao, Hon'ble Minister for Commercial Taxes, Information and Public Relation and Cinematography, Andhra Pradesh. Shri B.C. Gangopadhyay, I.A.S., Secretary to Government, Medical and Health Department, presided. Dr. S.N. Mathur, Director of Medical and Health Services welcomed the gathering. Dr. D. Umopathy Rao, Honorary Secretary, TB Association of Andhra Pradesh, read his report. Dr. C. Sreenivasa

Rao, Director, State TB Centre, Irramnuma, Hyderabad, read the messages received from the Vice-President of India, Governor of Andhra Pradesh, Chief Minister of Andhra Pradesh and President of the TB Association of India. Shri B.C. Gangopadhyay distributed Prizes and merit certificates. Dr. B. Narasimha Rao, Honorary Secretary, TB Association, City Branch, proposed a vote of thanks.

### Goa, Daman and Diu

Shri S.K. Benerji, Lt. Governor and Patron of the TB Association of Goa, Daman and Diu inaugurated the Campaign at Vasco-da-Gama. Minister for Health. Sri Shanker Laad and President of the Association presided. The Lt. Governor announced a personal donation of Rs. 500/- towards the Seal Campaign. Mrs. Gauri Banerji distributed the awards and certificates. Welcoming the guests Smt. Sulekhabai Y. Chowgule, Chairman of the Care and After Care Committee, Vasco-da-Gama explained the activities of the Care and After Care Committee, Vasco.

### Maharashtra

The 28th TB Seal Campaign was inaugurated in Bombay by Shri Vasantharao Patil, Chief Minister, at his residence on 2nd October. Dr. M.D. Deshmukh, Honorary Secretary of Association, welcomed the guests and read message from Vice-President of India Mr. B.D. Jatti. Mr. Homi H.H. Taleyarkhan, Vice-President of the Association spoke about the activities of the Association. Dr. N.C. Puri, Chairman of the Fund Raising Committee, proposed the vote of thanks.

### WANDER-TAI ORATION

The Tuberculosis Association of India has selected Dr. G.D. Gothi, Epidemiologist, National TB Institute, Bangalore for the Wander-TAI Oration this year. Dr. Gothi will give the Oration at the time of the 32nd National Conference on TB and Chest Diseases in Trivandrum. The subject he has selected for this Oration is "Natural History of TB".

### SHRI LACHMI LAL BORDIA MEMORIAL AWARD

Dr. N.L. Bordia, former TB Adviser to Government of India, has donated a sum of Rs. 10,000/- to the Tuberculosis Association of India for instituting an annual cash award of Rs. 1,000/-, a certificate and a Silver Medal to

a non-medical tuberculosis worker in Madhya Pradesh. This award will be known as "Shri Lachmi Lai Bordia Memorial Award".

## STATE CONFERENCES

### Karnataka

The 7th Karnataka State TB Conference will be held on 29th and 30th October, 1977 at Madikeri, (Coorg), Karnataka. This will be inaugurated by Sri H.M. Channabasappa, Health Minister of Karnataka.

### Orissa

The first TB and Chest Diseases Workers' Conference of the Orissa State TB Association will be held on 5th November, 1977 at Bhubaneswar. The Conference will be inaugurated by the Health Minister of Orissa.

### SHIBIRS

The Maharashtra State Anti-TB Association organised a multi-diagnostic Shibir at Blind School, Jogeshwari (West) on 14th August, 1977.

Dr. R.B. Billimoria Centre of the Maharashtra State Anti-TB Association was inaugurated by Lion Surendra Mody, President, Lions Club of Khetwadi, on 2nd October.

Dr. M.D. Deshmukh announced that Mrs. Billimoria, apart from donating the room gave a further donation of Rs. 5,000/- for initial expenses. Lion President Surendra Mody declared the donation of a Steel cupboard and a steriliser.

Shri Homi J.H. Taleyarkhan who presided over the function said that he was glad that work was being extended in the area. He thanked Mrs. Billimoria for the generous donation and assured her that the name of her late husband would be kept up. Dr. T.B. Master proposed a vote of thanks.

The Association also organised an anti-tuberculosis Shibir in cooperation with the Rotary Club of Khopoli and Yusuf Meharalli Centre on 9th October, 1977.

### TB WEEK IN A.P.

TB Week celebrations was organised by the TB Association of Andhra Pradesh and its City Branch from 3.10.1977. Inaugurating the week

Shri B.C. Gangopadhyay, I.A.S., requested that the centres should cover hundred per cent by BCG programmes between the age group of 0-18 years. Dr. D. Umapathy Rao, Honorary Secretary, TB Association of Andhra Pradesh, gave a short report on the TB Week organised by the Association. Dr. S.N. Mathur, Director of Medical and Health Services, advised the patients to direct all people suffering from cough and fever to TB Clinics to take treatment regularly for more than 18 months. Dr. C. Sreenivasa Rao, Director of State TB Centre, proposed vote of thanks.

### REFRESHER IN A.P.

A Refresher Course under the auspices of the Andhra Pradesh TB Association, Anantapur District and its branch, I.M.A. Guntakal branch and the College of General Practitioners, Hyderabad was inaugurated by Dr. C.C. Mukhopadhyay, M.D., Head of the Department Laboratories Arogyavaram Medical Centre. Shri N.K. Parthasarathy, Divisional Superintendent, Southern Railway, Guntakal presided. The Scientific session on 10th was chaired by Dr. C.C. Mukhopadhyay and on 11th by Dr. S.C. Kapoor, M.D., T.D.D., F.C.C.P., Additional Chief Medical Officer, Southern Railway Hospital, Madras. Doctors from Anantapur, Dharmavaram, Gooty, Guntakal and other places attended the course.

### COMMUNITY PROJECTS

The TB Centre, Patiala has started a Community Project in some villages in Patiala District.

The Karnataka Association intends to start two new Pilot Projects in the districts of Gulbarga and Raichur this month.

### I.A.M.S. AWARD

The Indian Academy of Medical Sciences will award the "Dr. S.S. Misra Memorial Bronze Medal plus cash prize". Further particulars can be had from the Administrative Officer, Indian Academy of Medical Sciences, C-11/16, Ansari Nagar, New Delhi-110 016.

### OBITUARY

We regret to announce that Dr. J.B. Shrivastav, former Director General of Health Services and in that capacity, Chairman of our Association, from 23.7.1970 to 3.3.1976 passed away on 6th September, 1977. The Association offers its deepest condolences to the bereaved family.

# THE INDIAN JOURNAL OF TUBERCULOSIS

## ABSTRACTS

Vol. XXIV

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Abst. No. 4

### **Sarcoidosis: A Study in Eastern India**

*Samir K. Gupta, J. Ind. Med. Asso.; 1977, 68,245*

Seven cases of sarcoidosis diagnosed between 1972 and 1976 are reported from a hospital in Calcutta. The age range was 21 to 66 years (mean age 42.3 years). Two were females and 5 males. Most of the patients had some symptoms such as fever, cough, loss of weight, pain in the chest, gastro-intestinal disturbances etc. In 2 cases the symptoms were very vague and mild. Erythema nodosum was seen in one woman aged 55 years. Liver and spleen enlargement was common. The provisional diagnosis was sarcoidosis in 3 cases, lymphoma in 2, carcinoma of the liver in 1 and 1 progressive miliary tuberculosis. Four cases had bilateral hilar adenopathy, three of these along with some mottling of the lung field. Two had mottling and fibrosis without hilar adenopathy and in 1 x-ray chest was normal. In 4 the tuberculin reaction to 1 or 10 TU was 10 mm or more at 72 hours. Sedimentation rate was between 25 mm and 40 mm in most of them. The total serum proteins were raised only in 2 out of the 6 who were tested. The albumin/globulin ratio was reversed in 4 out of these 6. Total daily urinary calcium was raised in 3 out of 5 cases whereas serum calcium was raised only in one out of 5. Alkaline phosphates was raised in 1 and SGPT in 2 out of the 5 cases. Kveim test was positive in 6 out of 7 cases. In the Kveim negative case the liver and spleen were enlarged and the diagnosis was confirmed by both liver and scalene node biopsy.

S.P.P.

### **Experience with Kveim test in sarcoidosis in India**

*S.C. Chakravarty, V.N. Damodaran. J. Ind. Med. Asso.; 1977, 68, 247*

Kveim antigen is most commonly prepared from human spleen and lymph glands. Spleen is preferred as it yields a large amount of antigen at one time. All sarcoid tissues are not antigenic and cannot produce the typical Kveim reaction. Active principle of the antigen is particulate and in the membrane fraction of the tissue. It is kept

in suspension and gives rise to Kveim reaction only when bound to a macromolecular carrier. 0.1 to 0.2 mm of Kveim antigen is given intradermally on the volar surface of the forearm. Corticosteroids if being given must be stopped 2 weeks before the test and should not be administered till biopsy has been completed 6 weeks after the injection. A patient who gives a positive reaction generally gets a papule at the site of injection after about 3 weeks. The size of the papule is a reliable measure of the level of Kveim re-activity. Skin biopsy at the Kveim test site shows epithelioid granuloma with or without giant cells in positive cases. In negative patients perivascular lymphocyte infiltration is generally seen in the biopsy of the test site.

Positivity of Kveim reaction depends on duration of the disease. If the test is done within 2 years of the onset of disease, there is a greater chance of getting positive result. In the author's series 17 out of 28 were positive when the duration of disease was less than 2 years and 4 out of 18 when the duration was over 2 years.

S.P.P.

### **Sex ratio in erythema nodosum**

*Hans. J. Ustvedt. J. Oslo City Hasp.; 1977, 27, 9*

The association of erythema nodosum (EN) with primary tuberculosis is very well known. It is also seen in cases of sarcoidosis. Of particular interest is a liability of EN to be precipitated by a series of drugs and specific antigens. The latter include hemolytic streptococci, coccidioidomycosis, histoplasmosis, gonorrhoea, diphtheria, meningococcal infections, staphylococcal infection, syphilis, brucellosis etc. Rarely it is associated with conditions such as ulcerative colitis, Crohn's disease, Hodgkin's disease etc.

It is characterized clinically by nodules of a bright red or bluish colour, localized predominantly to the extensor surfaces of the legs, sometimes also to the arms and other parts of the body but rarely the face. The nodules never break down but resolve within a few days to a couple of weeks with a series of colour changes similar to those

seen after a contusion, often accompanied by fever, joint pains, increased ESR and exceptionally by episcleritis. The time interval between the exposure to the causative factor and appearance of nodules varies from 1 to 6 days. It is believed to be a manifestation of immune reaction of the delayed hypersensitivity type *i.e.* a cell-mediated immune response with T lymphocytes as the initiator of the phenomenon. Circulating antibodies are not involved nor is the complement. When cutaneous sensitivity to tuberculin reappears after temporary suppression as in measles and other acute diseases, re-appearance of allergy may also be followed by a fresh crop of EN nodules. EN seen in sarcoidosis and other conditions does not seem to differ clinically or histologically from the typical EN following primary tuberculous infection. Occurrence of EN seems to vary in different geographical regions and ethnic groups. Scandinavians and Negroes in USA seem to be involved much more than others.

EN is seldom seen in children under one year of age. Till puberty there is only slight preponderance in females but thereafter the picture changes completely and 80 to 90 % of the cases are amongst women. This female preponderance seems to hold good for all causes of EN. This difference is not due to exogenous factors but is believed to be immunological and/or hormonal.

The results of 10 years follow up of patients with EN showed a higher morbidity and a slightly higher mortality from tuberculosis than in the general population of corresponding age and sex.

S.P.P.

#### **Escherichia Coli Meningitis and Congenital Tuberculosis in the same infant**

*O.G. Brooke, Jean Dow and T.K. Hand. St. George's Hospital, London. Lancet; 1977, i, 599*

A girl weighing 2.46 kg., the first child of Kenyan Asians who had been living in U.K. for the last 12 years was delivered by forceps at 38 weeks gestation. Her mother had been unwell with a low grade fever in the week before delivery and continued to be febrile in the puerperium. The cause of this illness was not apparent at that time but x-ray chest was normal. The infant was in fair condition at birth and needed no special resuscitation. She became jaundiced on the sixth day but the bilirubin level fell quickly thereafter and no transfusion was given. No cause for jaundice could be found. On the 14th day the infant was discharged, apparently well and gaining weight, only to be re-admitted two days later with a 24 hour history of pyrexia, listlessness and

poor feeding. WBC count was 11,900 with 55% neutrophils. CSF contained 6 polymorphs, 110 protein and 33 glucose. Culture of CSF yielded a pure growth of *Escherichia coli*. She was treated with gentamicin and ampicillin and subsequently chloramphenicol. Shortly after admission the infant developed tachypnoea and some respiratory difficulty. X-ray chest showed diffuse miliary mottling suggestive of miliary tuberculosis. This diagnosis was rejected at that time in favour of septicaemia with haematogenous spread to the lungs on the grounds that mother's x-ray chest was clear. CSF culture was positive for *E. Coli* and the infant was very young. X-ray chest repeated a few days later showed further deterioration with bilateral confluent pneumonia. The infant died on the 23rd day and necropsy confirmed the diagnosis of miliary tuberculosis with large number of acid fast bacilli in the lung tissue. The infant's mother was subsequently investigated and uterine curettings were found to contain tubercle bacilli.

The case is a reminder that genital tuberculosis in women may not be associated with infertility and coincidental infections with different pathogenic bacteria may occur congenitally or otherwise in immunologically incompetent individuals including neonates.

S.P.P.

#### **Diffuse pulmonary disease after therapy with Nitrogen Mustard, Vincristine, Procarbazine and Prednisone.**

*Robert J. Farney et al. Amer. Rev. Resp. 1977, 115, 135*

Pulmonary reactions may follow treatment with Nitrogen Mustard, Vincristine, Procarbazine and Prednisone. Two patients with Hodgkin's disease are described who were treated with these drugs and developed diffuse lung disease. Although non-specific, the following features are suggestive of hyper sensitivity reactions:

- (a) Extensive eosinophil reactions
- (b) Heavy plasma cells infiltration
- (c) Angitis and
- (d) Non-caseating granulomas like those of sarcoidosis

The disease processes were evaluated with the help of serial pulmonary function studies, chest radiography and open lung biopsy. The reaction responded favourably to treatment with corticosteroids. Procarbazine may have been the incriminating agent.

S.P.P.

Persistence of *Mycobacterium tuberculosis* in sputum without chest roentagenographic evidence of active disease.

*Stephen J. Jay et al. Amer. Rev. Resp. Dis.; 1977, 115, 147*

Tubercle bacilli were isolated consistently from the sputum of a 64 year old man over a period of 11 years. The patient was admitted to a hospital because of haemoptysis. There were no other symptoms. He was non-smoker and physical examination of the chest was completely negative. Chest skiagram showed evidence of healed primary tuberculous lesion at the hilum without any evidence of parenchymal disease. The source of bacilli was proved to be a hilar lymph node with a bronchial fistula.

S.P.P.

#### **Depressed lymphocytes function after bereavement**

*R.W. Bartrop et. al. The Lancet, 1977, I, 834*

During 1975 twenty-six bereaved spouses took part in a detailed prospective investigation of the effects of severe stress on the immune system. T and B cell numbers and function and hormone concentrations were studied approximately 2 weeks after bereavement and 6 weeks thereafter. The response to phytohaemagglutinin was significantly depressed in the bereaved group on the second occasion, as was the response to concanavalin A at 6 weeks. There was no difference in T and B cell numbers, protein concentrations, the presence of auto-antibodies and delayed hypersensitivity, and in cortisol, prolactin, growth hormone, and thyroid hormone assays between the bereaved group and the controls. This is the first time severe psychological stress has been shown to produce a measurable abnormality in immune function which is not obviously caused by hormonal changes.

S.P.P.

#### **Defence mechanisms of the Respiratory Membrane**

*Gareth M. Green et al. Amer. Rev. Resp. Dis.; 1977, 115, 479*

The respiratory membrane is the most extensive of all tissues that interface directly between man and his environment. The success or failure of pulmonary defence mechanisms largely determines the appearance of clinical lung disease. The lung is protected by inter-locking systems of non-specific and specific defences. Inhaled substances can be isolated by mechanical barriers or can be physically removed from the lung either

by transport up the bronchial mucociliary escalator or by transport through interstitial and lymphatic channels leading to lymph nodes. Substances can be locally detoxified within the lung by interaction with secretory proteins, such as antibodies, or by neutralization and dissolution within phagocytic cells.

The pulmonary alveolar macrophage is the central figure in the protection of the respiratory membrane operating in all 3 of the non-specific modes of defence and augmented by specific immunologic mechanisms as well. Alterations in macrophage function and physiology may be crucial in determining the effectiveness of pulmonary defence. Recent advances in the cell biology of the alveolar macrophage have led to a greater understanding of its complex function. The multiple origins of macrophages from local and circulating cell pools and the variability in their fate and life span reflect the multi-faceted role of this cell type. The importance of the interactions between macrophages, other lung cells and other defence mechanisms has become increasingly clear. As well as functioning as resident defender of the alveolus, the macrophage is an important effector of the pulmonary immune response and plays a key role in the pathogenesis of a wide variety of inflammatory, destructive and fibrotic lung diseases.

Humoral and cell-mediated immune responses amplify and direct lung defences against infection and may also participate in protection against other agents. Immunoglobulin A and G, microbial neutralizing and opsonizing antibodies and macrophage-stimulating T lymphocytes are the major immunospecific forms of lung defence. Infectious agents cigarette smoke, air pollutants, industrial dusts, and a spectrum of co-existent disease states may impair pulmonary defence mechanisms and increase susceptibility to acute and chronic respiratory diseases. A thorough understanding of the ways in which the lung protects itself against the daily assault of infectious, toxic and immunogenic materials should lead to a better understanding of the pathogenesis and consequences of lung disease and to better clinical care of the patient with respiratory disease.

S.P.P.

#### **Ultrasound in examination of Pleural and Parenchymal Diseases**

*F. V. Adams and V. Gulati, Amer. Rev. Resp. Dis.; 1977, 115, (Suppl. April) 83*

In order to determine the usefulness of ultrasound in the evaluation and differentiation of

pleural and parenchymal disease, 50 patients were examined in whom physical and radiologic examinations were non-diagnostic or who were judged at high risk for exploratory thoracentesis. Negative echograms for pleural fluid, which confirmed equivocal physical and radiologic examinations, were recorded in 13 patients. In 34 patients the characteristic M-mode display of a central echo-free space, indicative of pleural fluid, was recorded. Aspiration yielded fluid as localized by echogram in 30 (88 %). Of the 30 patients, 13 (43 %) had negative lateral decubitus views and 10 (30%), had been aspirated unsuccessfully before ultrasound localized the fluid loculation. The remaining 7 patients, including 3 on mechanical ventilation, who were believed to have increased risk for thoracentesis had successful initial tap based on echographic localization of fluid. Several effusions less than 100 ml in size, including one 10 ml loculation, were detected by echogram. It is concluded that ultrasound allows detection and localization of pleural fluid when radiologic and physical diagnostic means are not helpful.

Three patients with fluid-containing pulmonary cysts were also examined by ultrasound. An echo-free space which corresponded to intracystic fluid noted on x-ray was demonstrated in all three. Comparison of these recordings with those from patients with pleural effusion suggest echo characteristics that may distinguish between intra and extra-pulmonary fluid accumulations.

S.P.P.

#### **Lung Cancer survival and mediastinal glands: Reassessment**

*G.L. Baum et. al.- Amer. Rev. Resp. Dis.; 1977, 115 (Suppl. April), 87*

Follow up of 202 patients operated upon for lung cancer between 1-1-66 and 31-12-70 was obtained for all and survival related to cell type and presence of hilar glands; mediastinal glands with or without hilar glands or no positive glands. Of the 202 cases operated upon, 151 had primary resections. Of the resected cases five year survival of the entire group was 27.8 % with 40.8 % of the 44 cases of large cell tumor surviving this period and 19.2% of the adenocarcinomas. These represented the extremes of the survival for the common type of lung cancer encountered. A surprising fact was that adenocarcinoma was the commonest type of lung cancer encountered in Israel. When presence or absence of mediastinal glands was considered in each cell type it became clear that for all cell types in the operated group survival at five years was down to 11.1 % when

mediastinal glands were present at operation compared to 31% and 36% when either only hilar glands or no glands were presented. For large cell tumor and adenocarcinoma presence of mediastinal glands clearly compromised survival when compared to no or only hilar glands. For epidermoid cancer the separation was not so clear. The group of small cell tumours was too small to judge. On the other hand a total of 5 of 45 cases with mediastinal glands did survive five years which raises some question as to the exclusion of resection of carcinoma when the presence of mediastinal glands is the only clinical factor against resection. In large cell tumours, however, it is significant that inspite of 52 % survival of 25 cases without glands only 1 of 10 with mediastinal glands survived 5 years. 3 of 12 cases of epidermoid cancer with mediastinal glands survived this period.

S.P.P.

#### **Nasal Polyps and Bronchial Asthma**

*J.R. Moloney and J. Collins. BJ. Dis. Chest. (1977) 71. 1*

Association of nasal polyps and asthma is not infrequent. The association of two is more in non atopic patients particularly who develop sensitivity to salicylates. The histopathology of nasal polyps is similar in both atopic and non atopic subjects. The relationship between the onset of asthma and the appearance of nasal polyps or the removal of nasal polyps is uncertain. In patients with late onset non atopic asthma it is advisable to warn of the possible dangers of salicylates.

H.B.D.

#### **B.C.G. and vole bacillus in the prevention of tuberculosis in adolescence and early adult life.**

*P D' Arcy Hart, IAN Sutherland. Brit. Med. Jour. 30 July, 77*

A 20 years follow up trial of B.C.G. and vole bacillus vaccines in the prevention of tuberculosis in 54,239 participants showed protective effect of each vaccine in 84 % during the first five years. This gradually decreased to 77% on an average for each vaccine over the whole period. There was decline in the incidence of tuberculosis in all groups during the trial.

Of the total 610 cases of tuberculosis, only 27 developed tuberculosis between 15 and 20 years.,

The prevalence and incidence of tuberculosis

ABSTRACTS

in Great Britain have decreased radically during the trial period. The expected benefit from large scale B.C.G. vaccination of children is now far less and may decrease further, if incidence of tuberculosis continues to decline.

H.B.D.

**Asthmatic Pulmonary Eosinophilia—A Review of 65 cases.**

*W.G. Middleton, I.C. Patterson, I. W.B. Grant and A.C. Douglas. Br. J. Dis. Chest (1977) 71, 115*

Out of 65 patients with asthma and eosinophilia, 54 were investigated and in 32 (59%) allergic aspergillosis was present.

Asthma with pulmonary eosinophilia carries a relatively poor prognosis as regards permanent symptoms, impairment of pulmonary function and residual radiographic abnormality. Association of allergic aspergillosis with long standing asthma, the prognosis was least favourable.

Patients receiving long term daily corticosteroids are even less likely to develop further radiographic opacities than with those given intermittent corticosteroid therapy. For prevention of recurrent pulmonary infiltrates, it is necessary to give a daily dose of 10 mgm prednisolone.

H.B.D.