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Editor:

Dr. P.K. Sen

Co-Editors:

Dr. M.D. Deshmukh

Dr. N.L. Bordia

Associate Editors:

Dr. H.B. Dingley Dr.

S.P. Pamra

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Abstracts

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SPUTUM NEGATIVE PATIENTS

For many years, there has been an increasing awareness of a need to study the fate of sputum negative patients who seek treatment at various treatment centres for symptoms suggestive of chest diseases. Some workers have recommended that such patients should be kept under observation while many clinicians prefer to put them on prolonged intensive anti-TB drug therapy. In 1974, this problem was discussed in the pages of this Journal*. The National Conference at Hyderabad in 1975 devoted a full session to this important problem. A study based on a long term follow-up by the New Delhi TB Centre showed most of such patients to be definitely active and tuberculous.

A study carried out in Hong Kong recently with the collaboration of the British Medical Research Council has shown that 34% of the patients whose five sputum specimens were negative by direct smear became sputum positive during the course of one year when left untreated. On the other hand, there was hardly any significant relapse at 12 months' assessment among those treated with standard therapy or short term chemotherapy.

A controlled study of such patients was undertaken recently by the National TB Institute, Bangalore also. The patients included in this study were freshly detected, previously untreated residents of Bangalore City, judged as active by two miniature X-ray film readers. Half of these were randomly allocated to a treatment regimen of Isoniazid and Thiacetazone and the other half were on a placebo. Thirty-one percent of those who were initially sputum negative by one smear examination were found to be sputum positive on second examination, 10.7% by smear and 20.8% by culture alone. This clearly brings out the importance of second or repeated sputum examination. Even among those patients who were not read as active by any of the two readers, 2.8% proved to be sputum positive on second sputum examination. A few more became sputum positive or showed radiological progress of disease during the course of follow-up with no treatment. Thus, about 40% out of the initially sputum negative continued to be sputum negative, while 60% of them proved definitely to be active tuberculous at some stage or other, more often within the first four months following detection. Among sputum negative treated group, regression of lesions was more frequent than in the untreated group.

*Indian Journal of Tuberculosis, July 1974.

The following observations appear pertinent to this problem in the present state of knowledge :

(1) There is no way of knowing who will break down and become sputum positive out of the patients judged as tuberculous, radiologically. Significance of a cavity as an index of future breakdown appears to be equivocal but the extent of disease does not seem to have any correlation with breakdown.

(2) In suspected radiological cases, repeated and careful sputum examination is highly desirable. Even under programme conditions, overnight specimen of sputum is more feasible than radiography. Today, most patients are being detected at TB Centres and urban health institutions. Even though most patients live in the rural areas, the number detected in P.H.Is. is small. Repeated sputum examination is essential for reliable diagnosis. The second specimen could be an overnight collection. In various centres spot specimen is not collected as carefully as under research conditions, and that is why the results in programme conditions are comparatively poor. When examination of spot specimens gives poor positive results, the workers in P.H.Is. feel depressed and avoid an unrewarding task. This is one of the reasons why the P.H.I. staff do not like to examine sputum.

(3) Miniature films should be read by experienced readers and carefully. Wherever possible, two readers should read the miniature films independently though a short period of follow-up is more rewarding than dual reading. Suppurative disease, allergic conditions, bacterial and viral pneumonias etc. can and must be excluded before diagnosis of tuberculosis is made.

(4) In rural areas where follow-up for observation is not easily possible and is hardly ever done, patients with highly suspicious x-ray shadows should be offered anti-TB drug therapy if clinical picture is also suggestive since tuberculosis continues to *be* the most common pathology. Mistakes in diagnosis, though few, will continue to occur even in the best of hands, the question is, which is less risky and likely to be more profitable generally—to treat or not to treat.

REVIEW ARTICLE

ABDOMINAL TUBERCULOSIS — A REVIEW

M.D. DESHMUKH*

Historical Aspects

Hippocrates had noted intestinal involvement in Pulmonary Tuberculosis as also its seriousness for he declared "Diarrhoea attacking a person with Phthisis is a mortal symptom" (Pimparkar 1977). Lubeck incident in 1930 furnished convincing proof that gastrointestinal tract can be the seat of primary tuberculosis by ingestion of tubercle bacilli. 251 new born infants were given 3 oral doses in 10 days of what was believed to be B.C.G. but was later proved to be Human strain of tubercle bacilli 72 of these 251 children died of tuberculosis. Primary lesions were found in the alimentary tract in all 100% including 15 cases where lesions were found in the lungs too, in addition to the alimentary tract (Rich 1951).

Definition

There seems to be some difference of opinion as to the various manifestation that could be included under the term abdominal tuberculosis. Armstrong (1952) stated that abdominal tuberculosis may be said to comprise infection by the Mycobacterium Tuberculosis of (i) the intestinal tract (ii) the mesenteric lymph glands (iii) the peritoneum.

Achar and Viswanathan (1972) appear to agree with the above definition but Wig (1972) is of the opinion that it should include disease of the stomach, duodenum, small bowel, large bowel, intra-abdominal lymph nodes, liver, spleen, pancreas and the peritoneum. Genito urinary tuberculosis is usually classified separately although kidneys and a major portion of the genital system in the female are in the abdomen. The term abdominal tuberculosis is, however, commonly applied by clinicians to denote intestinal, glandular and peritoneal tuberculosis which are far more common than tuberculous disease of other abdominal organs. Wig further suggests that for a bedside approach, it may, however, be useful to retain only two groups viz. (a) intestinal, (b) peritoneal. Glandular involvement is almost always present in both the groups.

Prevalence

In U.K. and U.S.A., primary abdominal

tuberculous infection by ingestion of infected milk from tuberculous cows (bovine infection was quite common at one time. Blacklock (1932) performed 1800 autopsies on children in Glasgow, of which 283 showed tuberculous lesions. In 101 (36%) the primary site was in the abdomen. In 18, there was naked eye evidence of intestinal ulceration and in the remaining 83 abdominal lymph glands were the seat of disease without any obvious lesion in the intestines. Bacilli might have produced only microscopic lesions or might have passed through the intestinal wall without invading it, borne by the Phagocytes through the lymphatic vessels to the lymph glands. The severity of the condition was also obvious by the fact that 25% of these children showed progressive disease such as generalised miliary tuberculosis, tuberculous peritonitis, tuberculosis of the brain and its meninges.

Energetic measures to control bovine infection in the cattle both in U.K. and U.S.A., resulted in abolition of bovine tuberculosis in cattle and man. A few years later, with the advent of specific anti-tuberculosis drugs, a remarkable degree of control of tuberculosis was achieved in advanced countries and with that, prevalence of both pulmonary and extra thoracic lesions came tumbling down. Warren & Sommers (1948), after examining 125 operation specimens, came to the conclusion that almost all cases of the once common hypertrophic tuberculosis consisted of cicatrizing enteritis. This, of course, does not apply to India. Wig and Bawa (1953) and later Wig et al (1954, 1956 and 1961) emphasised that in India, tuberculosis of the intestines was the commonest granulomatous lesion and Crohn's disease was quite rare.

In India, bovine tuberculosis has never been a problem as tuberculosis is rare among Indian cattle. Moreover, the Indian habit of boiling milk before consumption renders it safe, even if infected. But no appreciable degree of control of tuberculosis from human strain has been achieved in India even after the advent of specific anti-tuberculosis drugs and hence it is no wonder that most workers find that abdominal tuberculosis is quite common in India (Pimparkar 1977; Prakash 1978). All workers state that no exact figures can be given as there have been no surveys in India to determine the prevalence of

* Professor Emeritus in Tuberculosis, J.J. Group of Hospitals, Bombay.
Honorary Secretary, Maharashtra State Anti-Tuberculosis Association, Bombay.

abdominal tuberculosis. Surveys, in any case, would be difficult to carry out since the only convincing evidence is bacteriological or histopathological proof of the presence of tuberculosis in the biopsy specimens of intestine, abdominal lymph nodes or peritoneum and the biopsy specimens can be obtained only by punch biopsy, laparoscopy or laparotomy.

Hence, we fall back on data from hospital patients or autopsy material. Trivedi and Gupta (1941) and Bhansali et al (1968) found evidence of intestinal tuberculosis varying from 0.02 to 0.5 in unselected autopsy material. Ukil (1942) reported intestinal tuberculosis secondary to pulmonary lesions in 51% of the 1000 autopsies.

Pimparkar (1977) found evidence of tuberculosis in 1402 (12%) out of 746 autopsies carried out from 1964-1970 at the K.E.M. Hospital, Bombay. The tuberculous lesions in various organs were as follows: Lungs 7%, Liver 1.7%; Intestines 1.4%; Spleen 1.1%; Peritoneum 0.4%; Colon 0.22%; Stomach 0.008%; Pancreas 0.008% and others 0.008%.

Chuttani (1970) found the 0.8% of hospital admissions to be due to intestinal tuberculosis 3.4 to 11% of all small bowel obstructions and 5.7% of all perforation were reported to be due to intestinal tuberculosis (Bhansali and Desai 1958; Bhansali et al 1968; Bhansali 1968). Achar & Vishwanathan (1972) stated that primary abdominal tuberculosis was extremely rare. Most of the cases were due to haematogenous dissemination. There were 43 cases (3.5%) of tuberculous lesions in the abdomen among 1215 autopsies on children carried out in Christian Medical College, Vellore. of these 2 had perforation of ulcers in the intestines and 2 had intestinal obstruction.

Most of the workers agree that secondary abdominal tuberculosis is mostly seen in young adults in the 3rd and 4th decades of life and it is 3 to 4 times more common in females (Wig et al 1961; Sathe 1963; Pimparkar 1977). Peritonitis is more common in children and young adults especially females (Armstrong 1952).

Prem Mukherjee and Ravi Raj ore (1979) found 45% of female patients of abdominal tuberculosis to be in the 21 to 30 years age-group. The common lesion was hypertrophic ileocaecal disease (35%). There were only 5 cases of distant colon involvement and 2 cases of gastric involvement. Elhance (1979) found 25 cases of ileocaecal tuberculosis in his series of 98 cases (25.5%) and ulcerative lesions of the small bowel in 33 (33.6%).

Next to ileocaecal region, the intestinal lesions are seen in the following descending order of frequency - ileum, caecum, ascending colon, jejunum, appendix, sigmoid colon, rectum, duodenum, stomach and oesophagus (Bentley and Webster 1967; Gupta and Dube 1970; Paustian 1964).

Pathology

Armstrong (1952) describes the modes of infection and pathological changes in different types of lesions in abdominal tuberculosis as follows:

(1) Intestinal tuberculosis.

Method of infection

- (a) Primary — by ingestion
- (b) Secondary— (i) by ingestion
- (ii) by hematogenous spread
- (iii) by lymphatic spread

Types: (i) Ulcerative (ii) hyperplastic or hypertrophic.

(2) Mesenteric lymph gland tuberculosis.

Method of infection.

- (a) by spread from intestinal mucosa.
 - (i) in primary (ii) in secondary
- (b) by haematogenous spread.

(3) Tuberculous peritonitis.

Method of infection

- (a) Extension from caseating and breaking down glands, from loculated area of peritonitis, by direct continuity or by lymphatic spread.

- (b) Haematogenous spread

Types: (i) Caseating or ulcerative type
(ii) Ascitic; (iii) Fibroid.

Tubercular lesions especially in primary infections in young children are found mainly in the lower part of ileum because it is rich in lymphoid tissue and its peyer's patches are proportionately larger than in adults. The liability of infants to attacks of enteritis from other causes probably increases its vulnerability to infection by the tubercle bacilli.

In secondary intestinal infection also the site is more commonly in the lower part of ileum especially in the ileocaecal region (Anand 1956; Paustian 1964; Chandra and Basu 1967; Bhansali et al 1968; Pimparkar et al 1874). This is attri-

buted to (i) increase physiological stasis (ii) abundant lymphoid tissue, (iii) increased rate of absorption from the area, (iv) nearly complete absorption of the small bowel contents from the area (Paustian 1964; Anderson 1963; Boyd 1943; Pimparkai 1977).

The tubercular lesions start in the peyer's patches and solitary lymph follicles which show swelling, caseation, softening and ulceration. The ulcers spread transversely round the gut. The walls and edges are thickened and raised but not undermined. The floor is caseous and often shows miliary tubercles which may also be found on the peritoneal surface of the bowel. The mesenteric and para-aortic lymph glands are usually enlarged. Perforation is rare because of thickening of the peritoneum but local faecal abscess and fistulae may occur. Healing may cause stenosis but complete obstruction is rare because of the fluid contents of the small intestines. Obstruction is mostly from kinking through adhesions or through strangulation under a fibrous band. Severe haemorrhage is rare because of periarteritis and endarteritis but microscopic blood in stools is common and occult blood may occur in early cases even when microscopic blood is absent.

There is a rare hypertrophic or hyperplastic variety, characteristic of which is a great proliferation of the connective tissue involving all layers of the bowel and extending completely round the gut. Ulceration of the mucous surface may occur but it is late in onset and of little importance. The wall may be an inch thick and lumen greatly narrowed. The part usually affected is caecum and ascending colon but sometimes other parts of the colon may be affected. The affected segment forms an elongated, easily palpable mass usually hard and tender, often mistaken for carcinoma. Wig. (1972) feels that the hypertrophic variety depends upon haematogenous infection by an organism of low virulence in a person with high resistance. The infecting organism is always of the human type of tubercle bacilli.

It has been mentioned that involvement of the stomach is rare but isolated cases are described even in Indian literature. Bhargava & Sekhni (1973) Wani and Rashid (1977). Good (1941) stated various factors which protected the stomach from involvement in tuberculosis. (1969) suggested that tubercle bacilli may enter via a pre-existing ulcer. Abrams and Holden (1964) described haemorrhage, perforation, obstruction & fistulae formation as complications of the lesion. Massive haemorrhage was reported by Bhargava and Sekhni (1933).

Mesenteric lymph gland tuberculosis

This may be a part of the primary complex or the spread may be from intestinal lesions or the infection may come via the blood stream. The glandular component may be out of proportion to the intestinal lesion. The juxta intestinal glands may be small and intermediate group in the mesentery enlarged considerably, caseous and may break down. Spread may occur to the gland in the mesenteric root to the para aortic glands and on to receptaculum chyli and then to the thoracic duct resulting in miliary dissemination. Caseous glands may invade the peritoneum or may soften and breakdown forming a localised abscess. Intestinal obstruction may result from pressure of glands or kinking through adhesions. Healing is by fibrosis or calcification.

Tuberculous peritonitis

Apart from spread from caseating glands or intestinal lesions, it may also occur from salpingitis of haematogenous origin in young women. Fulminating type may be seen as a result of severe generalised infection of many organs. Peritoneum is studied with minute grey tubercles, particularly over the small intestines and the omentum.

Subacute type : This may be a part of polyserositis. The peritoneal tubercles are larger. Loops of intestines are inflamed and tumid. Meteorism may be a marked feature. It may resolve or may become chronic .

Chronic type of peritonitis: It is chronic but usually benign. It may be (i) caseating or ulcerative (ii) Ascitic (iii) Fibroid.

(i) The caseating form is serious when it occurs in infants and young children where it is common. There is a caseation and breakdown of mesenteric glands, formation of caseous masses between loops of adherent bowels with caseation and thickening of the omentum. Rolled up omentum may resemble intussusception, kinking, angulation and even obstruction may occur. Effusion is scanty, sometime haemorrhagic, often encysted through formation of granulation tissue arising from and binding adjacent loops. Ulceration of the bowel may take place and may lead to faecal abscess or even faecal fistula.

(ii) Ascitic form: This is usually seen in children of school age, adolescents or young females. Fluid may be small or large in amount. It is usually straw coloured with specific gravity above 1.016, protein contents more than 3.5 gms. per 100 ml. and cell count over 100 cells per cm. predominantly lymphocytes. (Jain et al 1966;

Singh et al 1969). According to Jain et al (1966) a comparative study of blood and ascitic fluid sugar is of considerable diagnostic importance because a significant decrease (55 to 100 mgm. %) is observed in tuberculous peritonitis, as compared to the non-tuberculous cases where the values are usually 90 to 135 mgm. %.

(iii) Fibroid type: This appears to occur when the peritoneal resistance is high. It is analogous to dry pleurisy. Fluid may be abundant or small in amount. The tuberculous granulation tissue instead of caseating progresses to formation of fibrous tissue. Sometimes dense adhesions form between coils of intestines and obstruction may occur.

The histopathological picture in abdominal tuberculosis is apparently the result of the interaction between the virulence of the organism and the degree of immunity or allergic reaction of the host. Pathologically tuberculosis has been classified as (i) ulcerative type (ii) hyperplastic type and (iii) ulcero-hyperplastic type (Anderson 1953; Boyd 1943). The usual microscopic picture is that of a mononuclear cell infiltration with epithelioid reaction and foreign body giant cells. In tuberculous enteritis, the most significant histological feature is caseation necrosis but it may be present only in the mesenteric glands with non-specific changes in the bowel wall (Anand 1956). The effect of anti-tuberculosis drugs on the pathology has also to be remembered for, by the time the patient comes to the surgeon he has often had considerable drug therapy. Typical caseation may then not be present. This is particularly noticed when right hemicolectomy for ileocaecal tuberculosis is done in two stages. The biopsy specimen taken at the first operation will show typical caseation necrosis, but the tissue removed at the second operation after anti-tuberculosis treatment may not show any caseation (Wig. 1972; Mukerjee and Rajor 1979).

Habibullah et al (1977) described 25 cases of intestinal tuberculosis diagnosed at operation and confirmed by subsequent histopathological examination of the tissue removed at operation. Typical tubercular granulation tissue was seen in all cases but caseation necrosis was observed only in 15 intestinal and 14 lymph node lesions.

Symptoms and Signs

As the pathology of different types of abdominal tuberculosis is often coexisting, the clinical picture is a composite one. If one or other aspect predominates, symptoms of that particular involvement will be marked. Acute cases with

marked toxæmia usually occur in infants and young adults.

Subacute and chronic type

Primary infection is characterised by insidious onset of abdominal pain, diarrhoea, anaemia and loss of appetite. The clinical picture consists of simple enteritis and general toxæmia with colicky abdominal pains referred to the umbilicus. Bowel movements may be irregular with diarrhoea alternating with constipation. There is rapid loss of weight, raised temperature, often hectic or irregular, with flushing of cheeks and night sweats. Patient is anaemic. The skin may show an earthen colour or bronze pigmentation may be noticed on hands and face. Pulse is raised. Stools are loose with blood and pus. Abdomen is tender in the right iliac fossa, flat or retracted but sometimes tumid. Meteorism may be present. Stools may show tubercle bacilli. The clinical picture may simulate typhoid or paratyphoid.

Secondary intestinal tuberculosis

If this is a complication of pulmonary tuberculosis, clinical picture of the latter will predominate.

Hypertrophic type:

This is a rare but important manifestation of chronic type. It is most probably haematogenous in origin and usually occurs in adults between 20 and 40 years of age.

There is vague pain in the right iliac fossa of colicky type. There is constipation alternating with diarrhoea. Intestinal obstruction may recur. There is fever and wasting.

On examination, a hard fixed palpable tumour is felt in the right iliac fossa. It is usually vertical and sausage shaped but may be round and pyriform. The size is usually that of a hen's egg or even bigger or there may be general thickening in the region. Right inguinal glands may be enlarged. Sometimes the tumour is felt only by examination per rectum or per vagina. Barium meal or barium enema x-rays may show considerable narrowing of the lumen of the caecum. It has to be differentiated from ovarian or tubal tumours, hydronephrosis, floating kidney, tuberculous adenitis, localised peritonitis or carcinoma.

Mesenteric gland tuberculosis

This is a part of primary disease in infants, school going children or adolescents. If perito-

neum is involved it can be serious (Price and MacKauley 1942). Achar and Viswanathan (1972) state that symptoms in children may be absent or there may be colicky abdominal pains, general malaise, and steatorrhoea resembling tropical sprue and asthenia.

In ascitic form, the symptoms are usually insidious with low grade fever, asthenia, loss of weight and gradual enlargement of the abdomen. Pain is infrequent. Diarrhoea may be noted but vomiting does not occur till there is partial obstruction of the bowel. In the plastic variety, the clinical picture is often dominated by symptoms of partial intestinal obstruction—colicky abdominal pains, vomiting and constipation. There may or may not be fever and other constitutional disturbances but the general health is below par. On examination, the abdomen may reveal the characteristic 'doughy feel' or nodular masses especially in the umbilical region. A rolled up omentum may be felt in the epigastrium. The incidence of 'tabes mesenterica', once common in children, appears to have gone down considerably.

Large abdominal glands may compress the portal vein producing ascities and symptoms of portal obstruction. Pressure on the thoracic duct may cause Chylous ascities. I have recently seen a case where enlarged para aortic lymph glands had given rise to chylous fluid in the right pleural cavity. We had to be satisfied with diagnosis arrived at from tuberculous histopathology of a cervical lymphnode biopsy but whole body scan had revealed enlarged para aortic glands. Aspiration of the pleural fluid and prompt anti-tubercular treatment with rifampicin containing drug regimen gave satisfactory results. Subsequently a follow up 'whole body scan' showed marked reduction in the size of the abdominal glands.

Plain x-ray of the abdomen may occasionally reveal calcification in the lymph glands.

In the hyperplastic type, abdominal pain is present in 70 to 100% of the patients (Anand and Phatak 1961; Singh et al 1963) Exacerbation may occur from time to time in more than one third of the patients. Constipation is a common symptom but intermittent diarrhoea may occur in 25% of the patients. Wig (1972) noted alternating diarrhoea and constipation in only a few patients. He found flatulence to be a frequent symptom. There is a feeling of diffuse distension of the abdomen which is aggravated by meals and is relieved by passing flatus. It is associated with borborygmi in the majority of patients. Feeling of a persistent lump in the right iliac fossa or a 'ball of wind' or a 'gola' moving in the

abdomen are important symptoms, the latter highly suggestive of partial intestinal obstruction. Singh et al (1963) found toxæmia in about 25% of cases, and that, too, usually mild. They find that the thickened Caecum can be often emptied by pressure emitting a gurgling sound in this process. Pimparkar (1976), after a very painstaking study of 40 cases of abdominal tuberculosis, drew attention to the fact that to give a coherent clinical picture, one has to divide the cases into (a) those with intestinal obstruction, (b) those without intestinal obstruction. He found that in those with obstruction, abdominal pain, fever, anorexia and asthenia were present in 100%, while in the other group these symptoms were present only to the extent of 92, 69, 80 & 80% respectively.

Nocturnal sweats, borborygmi, nausea, vomiting and post-prandial discomfort were present in the obstructive group to the extent of 89, 89, 85, 81 & 67% respectively; in the other group these were 54, 30, 30, 23 & 38% respectively.

Diarrhoea was more common in non-obstructive group than in obstructive group (23% ; 14%) but constipation or diarrhoea alternating with constipation was more common in the obstructive group (constipation 7% and 0% ; alternating constipation and diarrhoea 67% and 23%). While loss of weight was equal, anaemia was slightly more in those with obstruction.

Intestinal function of absorption has been studied by means of standard glucose and lactose tolerance tests and by excretion of d-xylose, radioactive Co 58, B12 faecal chemical fat and by histological examination of intestinal biopsies obtained by oral route. Malabsorption of d-xylose Co57, B12 and steatorrhoea has been reported in about one third of cases. Malabsorption is much more common in those with strictures and obstruction than in those without strictures. Stagnant loop syndrome occurs commonly in those with ulcero-constrictive lesions and it has been found to be the commonest cause of malnutrition. Intestinal biopsies have revealed normal mucosa thus differentiating the stagnant loop syndrome from coeliac disease.

The following table shows the various values in the two groups of patients.

Mukerjee and Rajor (1979) reported 557 female patients of abdominal tuberculosis seen at Lady Hardinge Medical College and Hospital, New Delhi, between 1964 and 1975. They also found that the main symptoms like pain in the abdomen (dull ache to intermittent colicky pain) and distension were more common

Test	With obstruction (per cent)	Without obstruction (per cent)
Glucose Tolerance Test	28.00	0.00
Lactose Tolerance Test	22.00	0.00
D-xylose Excretion Test	57.00	8.00
Co 58 B12 f Excretion Test	63.00	30.00
Faecal fat Excretion Test	60.00	25.00

in stenotic and cicatrising variety. Vomiting was found to be an early symptom in small bowel lesions, intermittent diarrhoea and constipation was found in some while constipation was the predominant symptom in others. The majority of patients were thin and poorly nourished. The common finding was doughy abdomen. Lump in ileocaecal region was felt in 39.5% of cases. Visible Peristalsis and moving lump was found in a small percentage. All had varying degrees of anaemia and E.S.R. was raised above 15 m.m. (Westerner 1st hour) in 85% of cases.

Elhence (1979) found at Agra, that of the total 340 cases of subacute and acute intestinal obstruction, 236 had surgery and 104 received conservative treatment. Of those operated, 98 proved to be tuberculous. Findings among the 138 non tuberculous cases were volvulus of small intestine, bands and adhesions, mobile caecum and ascending colon and Meckel's diverticulum. Most of those cases had anti-tuberculous drug therapy under the mistaken diagnosis of tuberculosis. At times, automatic unlinking and relief of symptoms was taken as improvement because of drugs and anti-tuberculosis drugs were continued over long periods. Author divides his cases into 3 groups :

(1) Those with typical picture of tuberculous subacute obstruction with abdominal pain, difficulty in passing flatus and faeces, general ill health, toxæmia and pyrexia. These cases were either ileocaecal hyperplastic variety or small bowel strictures (58 cases).

(2) More acute symptoms (26 cases) — acute abdominal pain, tenderness, abdominal distension, signs of peritonitis and shock. These patients had acute symptoms superimposed on subacute or chronic obstructive symptoms. This type had miliary, plastic adhesive variety of

peritonitis or perforation above the ulcero-constrictive lesions.

(3) 14 cases had abdominal pain, anaemia, weakness and palpable mesenteric glands.

Duration of symptoms was as follows :

- (1) Subacute group — 1 month to 3 years
- (2) Acute group — 1 to 15 days.
- (3) Third group — 1 to 3 months.

Tuberculosis of the Stomach

The clinical picture of gastric tuberculosis simulates chronic gastritis, peptic ulcer or carcinoma of the stomach. There is post-prandial pain in the stomach, distension and discomfort. Vomiting and loss of weight may be the presenting features (Habibullah & others 1977; Hoonet al 1950). In 50% of the cases there may be a palpable mass and in about half of them there is evidence of tuberculosis elsewhere in the body (Chazan and Aitchison 1960; Gains 1952). Abrams and Holden (1964) described haemorrhage, perforation, obstruction and fistula formation as the complications of this lesion. Massive haemorrhage has been reported by Chazan and Aitchison (1960); Bhargava and Sefchon (1973).

Laboratory Investigations

It will be obvious from the variety of symptoms and signs in different types of cases that it will not be possible to diagnose abdominal tuberculosis with any certainty by clinical findings alone and that further investigations would be necessary. The only convincing evidence in abdominal tuberculosis, as in any other tuberculous lesion, will be to demonstrate tubercle bacilli or the typical histopathological picture in a biopsy specimen obtained by peritoneal biopsy or in a lymph gland or a piece of omentum or gut obtained by exploratory laparotomy or at the time of the abdominal operation. X'Ray investigations, while not as convincing as the bacteriological proof, do help in the diagnosis in many cases.

Wig (1972) suggests the following approaches to diagnosis :-

1. Plain x'ray of the abdomen may reveal calcified mesenteric glands in some cases as also the multiple fluid levels in patients with subacute intestinal obstruction. X'Ray of the chest may show co-existing active or old healed pulmonary lesions.

Barium enema studies are essential to diag-

nose intestinal tuberculosis of the hyperplastic variety. Caecum will be seen contacted and pulled up from its usual place in the pelvis. The lumen will be seen narrowed. The terminal part of the ileum is usually dilated but may be irregularly contracted. There will be irregular and persistent filling defect in the caecum and ascending colon. Barium meal studies should be undertaken only if there is no clinical evidence of intestinal obstruction. Stricture and dilatation of the small bowel can be well studied by Barium meal x-rays. When ileum is involved, co-existing lesion in caecum is a pointer to diagnosis of abdominal tuberculosis.

2. Examination of ascitic fluid : The fluid will have all characteristics of an inflammatory exudate. Tubercle bacilli can sometimes be demonstrated by direct smear examination of the centrifuged deposit but culture examination will reveal them more often.

3. Peritonoscopy : Visualization of the peritoneum by a laparoscope, biopsy and photographic record have been great advances in laboratory diagnosis.

Pimparkar (1977) feels that Peritonoscopy is safer than laparotomy for visualising the lesion and also taking a biopsy. Miliary tubercles can be seen particularly on the undersurface of the anterior abdominal wall. In a typical case, the peritoneum and small bowel appear studded with white nodules and loops of small intestines are dilated and matted together. The transverse colon is adherent to the omentum or to the anterior abdominal wall, creating numerous pockets in the Peritoneal cavity. The omentum is usually contracted and thickened as a result of infiltration with caseous material or may appear as a tumour mass. Occasionally, a thickened and contracted mesentery and the intestines may be bound down to the posterior abdominal wall. Pelvic organs and liver can also be inspected.

4. Punch biopsy of the Peritoneum -

Sarin et al (1962) as also Mehrotra and others (1966) found punch biopsy of Peritoneum to be a safe and valuable procedure for the diagnosis of abdominal tuberculosis. It is safe when there is Peritoneal effusion but is risky in the absence of fluid.

Biopsy with Peritonoscope as mentioned in (3) above is a safer method.

Wig (1972) further believed that, at times it became necessary to confirm the clinical diagnosis by bacteriological or histological examination of excised lymph gland or portion of the gut

obtained by laparotomy. This procedure is of course routinely adopted when abdominal operations are done in two stages as originally introduced in India by Anand and his colleagues at Amritsar (Anand 1956; Anand and Phatak 1961). Mukerjee and Rajor (1979) stated that out of the total 557 patients they had operated upon, diagnosis was confirmed at operation and by subsequent histopathological examination of lymph gland obtained at the time of operation in 380.

Pimparkar (1977) deals at length with the question of diagnosis. Some of the main points are noted below :-

The vagueness of symptoms as also the variation of signs make the diagnosis of abdominal tuberculosis difficult in early stages. The main aids are E.S.R., Tuberculin Test, examination of ascitic fluid and gastric contents, radiology, peritoneal or liver biopsy, peritonoscopy, examination of thoracic duct lymph and exploratory laparotomy.

A raised E.S.R. with symptoms of abdominal tuberculosis with or without a positive chest x-ray and with positive tuberculin test is suggestive of abdominal tuberculosis.

The radiological signs are — Slierlin's sign, string sign, dilatation, delay in emptying, irregularity of bowel silhouette filling defects of caecum, segmentation and fragmentation.

Pimparkar further states that majority of the patients are anaemic but the severity of anaemia may vary. As in other forms of tuberculosis, in abdominal tuberculosis also there may be lowering of total serum proteins and serum albumin and a rise of globulins, the increase being most marked in alpha 2 globulin. Determination of albumin alpha 2 globulin has been found to be a sensitive index of activity of tuberculosis by other workers also (Kulkarni et al 1960).

Hepatic involvement is not uncommon. Liver function tests are reported to be disturbed in Pulmonary and abdominal tuberculosis by other workers also (Sen et al 1972). Total and esterified cholesterol, plasma fibrinogen and prothrombin are low, whereas serum alkaline phosphatase, glutamic oxalacetic transaminase and thymol turbidity are high. Increased bromsulphthalein (B.S.P.) retention has been reported. Serum bilirubin and S.G.P.T. activity usually are normal. Liver biopsy is useful when abdominal tuberculosis is associated with miliary tuberculosis.

Pimparkar (1974) showed that tropical sprue

can be differentiated by the normal histological appearance of the jejunal mucosa in biopsy specimen obtained by per oral method in patients of abdominal tuberculosis with Steatorrhoea.

Pimparkar (1974) also points out that abdominal tuberculosis is the second commonest cause of malnutrition. He further argues that as the sites of D-xylose, glucose, lactose, Vit. B₁₂ and fats are different, the site of the lesion may be determined by doing all these tests. However, he observes, that this is not conclusive.

Pimparkar (1977) says that abdominal lymph gland biopsy after laparotomy is necessary in many cases to clinch the diagnosis. Lymph gland should be examined not only for typical caseating tuberculous lesions but also sent for a smear and culture for tubercle bacilli.

Steinback (1976) showed abnormal Gallicum⁶⁷ Citrate scan of the abdomen in tuberculous Peritonitis and suggests that this method may be used routinely. Use of whole body scan for demonstration of enlarged abdominal lymph glands has been mentioned before.

Witte and others (1963) suggest thoracic duct canulisation for getting lymph for examination as a simple means of diagnosing abdominal tuberculosis.

Management

All agree that the drug treatment of abdominal tuberculosis is similar to that of tuberculosis in the chest or any where else. As soon as the diagnosis is established the patient should be put on anti-tuberculosis drugs. These can be first line drugs in fresh cases but in those with history of previous anti-tuberculosis drug treatment, newer drugs have to be used. Intensity of the drug treatment will depend upon the severity of the condition. The duration is also the same as that for pulmonary lesions -- 1 year to 1^{1/2} years. In very severe cases especially if the patient can afford it, Rifampicin containing regimen may be given. With Rifampicin containing regimens the duration of treatment can often be reduced to 6 or 9 months.

Wig (1972) felt that PAS should be used with care and should preferably be avoided in cases with flatulence and diarrhoea. He also advocated corticosteroids under cover of potent specific anti-tuberculosis drugs in seriously ill patients. He also recommended general supportive treatment with rest, nutritious food, high protein, high calorie diet, supplementary vitamins and iron. The diet should also be a low residue one

and even this may be further modified in patients with diarrhoea.

Pimparkar (1977) recommended that every patient should be observed for long periods for evidence of cicatrisation and obstruction which will develop in some patients inspite of good chemotherapy. Some of the patients will need surgical treatment. Surgery is also indicated when perforation, blind loop syndrome or bleeding have occurred or when response to medical treatment is inadequate. Patients with hypertrophic ileocaecal tuberculosis and those with stricture, obstruction, and blind loop syndrome will require surgery more often than those with only peritonitis. Medical treatment before and after surgery is very necessary.

In the series described by Elhence (1979), the details of the 91 abdominal operations were as follows:-

Ileocaecal anastomosis (14); right hemicolectomy (17); limited ileocaecal resection (12); resection of ileal strictures (15); by-pass of ileal stenosis (12); laparotomy and separation of adhesions (6); laparotomy and lymph gland biopsy (14); stricture plasty for multiple stenosis (1). 11 patients died due to various complications which included faecal fistulae, persistent shock, fluid electrolyte imbalance, wound infection and dehiscence. Author believes that all cases presenting in subacute or acute stage of intestinal obstruction as also all with enlarged abdominal glands associated with general weakness and low grade fever should have laparotomy to confirm the diagnosis and to relieve obstruction. He feels that starting anti-tuberculosis drugs in a patient with only abdominal signs and no other evidence of tuberculosis elsewhere is not justified unless the diagnosis of abdominal tuberculosis is confirmed. Otherwise many non-tuberculosis cases may be unnecessarily treated for a long time with anti-tuberculosis drugs.

Mukerjee and Rajor (1979), with their immense experience of 557 cases of abdominal tuberculosis state that out of 557 operated cases, the diagnosis was established in 380 cases after laparotomy. Right hemicolectomy in one or two stages was done for ileocaecal type of lesions in 142; hemicolectomy with resection in 36; resection of small bowel in 90; resection of large bowel in 9; mesenteric lymph adenitis in 27; miliary tuberculosis in 59; ascites in 17; peritonitis in 4; gastro jejunostomy (for tuberculosis lesions of the stomach) in 2; ileo-transverse colon anastomosis.

Nazir and others (1977) described a case of

primary gastric tuberculosis which probably meant that the patient (an adult 35 years old) did not have any other tuberculous lesion. Clinically, it was diagnosed as a case of pyloric obstruction. Even at the time of operation when an indurated mass in the pyloric region was seen with marked adhesions between the mass and the pancreas colon and the gall bladder as also numerous enlarged lymph glands along the caeline plexins and greater and lesser omentum of the stomach, tentative diagnosis was inoperable carcinoma of the stomach. A palliative gastroenterostomy was performed after excising a lymph gland from greater curvature of the stomach. Histopathological examination of the lymph gland showed typical tuberculous lymphadenitis. Patient was then put on anti-tuberculosis drugs and was reported to have improved much after the anti-tuberculosis drug therapy.

Strik (1968) recommends partial gastrectomy followed by chemotherapy for tuberculosis of the stomach.

Summary

It is very important to remember that as long as Tuberculosis remains uncontrolled, abdominal tuberculosis like other forms of tuberculosis will be commonly met with.

About 70 to 100% of all granulomatous lesions, according to different workers, are of tuberculous aetiology in India. 15% of all fistulas-in-ano are of tuberculous origin. 3.4 to 11% of all intestinal obstructions and 5.7% of all intestinal perforations are due to tuberculosis.

Primary abdominal tuberculosis is very rare. Secondary lesions can be of haematogenous origin or direct spread from Pulmonary tuberculosis lesion by means of swallowed sputum.

Primary lesions, if seen at all, will be in infants and small children for they can pick up objects soiled with sputum of a tuberculous patient and put them in their mouth.

Abdominal tuberculosis (secondary intestinal) is common in the 3rd and 4th decades of life and females predominate. Tuberculous Peritonitis is common in school going children, adolescents and young persons especially females. Hypertrophic ilio-caecal tuberculosis is common in young adult males.

In the intestinal lesions, there are two common types — (1) Ileo-caecal hypertrophic lesions, and (2) Ulcero-constrictive lesions in the ileum.

Although any part of the alimentary canal can be affected, lesions of the oesophagus, stomach and rectum are very rare.

Symptoms and signs are very variable and clinical diagnosis almost impossible. The only convincing way to prove the diagnosis is to do bacteriological and histopathological examination of the biopsy specimens obtained by Peritonoscopy or laparotomy.

Anti-tuberculosis drugs, along with surgical intervention which is required quite often, give satisfactory results.

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SARCOIDOSIS IN INDIA

S.C. CHAKRAVARTY*

It has been reported that Sarcoidosis with erythema nodosum as acute disease has good prognosis. (Lofgren, 1953, James, 1961).

In India, there is no record of erythema nodosum with Sarcoidosis (Chakravarty 1971), Chakravarty and Damodaran 1978). Most of the patients referred to our clinic are first treated as tuberculosis for two to three years by some clinicians and when they are not improved, they are referred to us for investigation of Sarcoidosis. As such most of the patients come to our investigation at chronic stage and invariably with clinical findings. The important criterion for determination of evolution and prognosis of Sarcoidosis is the exact time of appearance of the disease. Unfortunately the exact time of appearance of the disease is difficult to locate except in a few cases.

With this limitation, this study was undertaken to note the evolution and Prognosis of Sarcoidosis in India.

Materials and Methods

24 Patients of proved diagnosis of Sarcoidosis were followed up from 3-15 years. 21 of these patients were positive to Kveim test and 3 were diagnosed by lung biopsy (Chakravarty and Damodaran 1977). of the 24 patients 23 were tuberculin (100 Tu) negative and one patient was tuberculin (5 Tu) positive. There were 14 males and 10 females in this group. Age group was 20-60 years but mostly between 20-40 years. Most of the patients had symptoms like cough, pain in the Chest, retrosternal pain or easy fatigability. So all of them were treated with corticosteroids for a varying period.

Radiologically, 8 patients had bilateral hilar adenopathy (BHL) *i.e.* first stage of Sarcoidosis. 6 patients had BHL + mottling of lung *i.e.* second stage of Sarcoidosis. 10 patients had mottling of lung with pulmonary fibrosis *i.e.* third stage of sarcoidosis. Of the last group 3 had Emphysema. 5 patients had enlarged liver and 4 Patients had enlarged spleen. Two patients had enlarged parotid glands. One patient had bundle-branch block in E.C.G. Hypercalcemia (with calcium more than 11.6 mg/100 ml) was present in 10 patients (41.6 per cent).

No patient had erythema nodosum nor ocular lesions (Hosoda, Chakravarty *et al* 1976) Chakravarty & Damodaran (1978).

Observation

Of 8 BHL group of patients, in 6. hilar adenopathy disappeared or reduced in size of 6 BHL + mottling group, 3 improved of 10 pulmonary mottling and fibrosis group, 4 improved radiologically and clinically. Three patients had emphysema of lung which improved clinically with corticosteroid. Two patients had progressive fibrosis of lung and died of pulmonary insufficiency.

Three patients had pulmonary tuberculosis (sputum positive for AFB) and one patient had tuberculous cervical lymphadenitis after 3 years of diagnosis of sarcoidosis. This was proved by cervical lymph gland biopsy.

Secondary candidiasis of lung (fungal infection) was present in one patient. 6 months after the diagnosis of Sarcoidosis, which was cured after treatment with aerosol of Nystalin in 40% propylene glycol in normal saline (Chakravarty 1967). One patient developed diabetes mellitus after treatment with corticosteroids. After treatment with corticosteroids out of 5 hepatomegaly patients liver came to normal size in 3, and one patient out of 3, enlarged spleen came to normal size.

Right bundle branch block in ECG in one patient was not observed after treatment with corticosteroid. Bell's palsy in two patients and Sarcoidosis of skin in two patients were cured after treatment with corticosteroid.

Illustrative case reports

A 50 year old Woman came to this clinic in 1971 with complaints of heaviness of chest and evening rise of temperature off and on since 1957. In the last 6 months she lost 6 kg of body weight and had exertional dyspnea. From 1957 to 1971 she had been treated for pulmonary tuberculosis. For two years with streptomycin, INH and PAS. (from 1957-1959).

After this whenever she had rise of temperature she used to get antituberculous treatment for two months every year. In 1967, She had popular lesion of skin before her right ear as also on forehead. It was biopsied and non-caseating granuloma was found. But since she was treated for tuberculosis, the attending doctors thought that this was also tuberculosis of skin. However, skin lesions disappeared spontaneously.

*North Delhi Clinic, 26-UA, Jawahar Nagar, Delhi-7.

If X-ray pictures of lung of this patient are studied carefully, (Fig. 1-4) it is observed that it took three years to progress from DHL (Bilateral hilar adenopathy) (Stage I) to BHL -f mottling stage (Stage II) and took four years to progress from BHL -f- mottling stage (Stage II) to pulmonary mottling + pulmonary fibrosis stage (Stage III). But at this stage also hilar adenopathy was present though reduced in size than before. But in nine years time from BHL stage, hilar glands disappeared and pulmonary fibrosis increased and later the patient became dyspneic.

(At our clinic in 1971, the patient was negative to tuberculin (100 Tu) and was positive to Kveim test. Serum calcium was 12.5 mg/100 ml. Sputum of the patient was negative for acid fast bacilli and fungal flora. She was diagnosed as a case of Sarcoidosis and was treated with corticosteroid for 6 months. She improved clinically. She had neither rise of temperature nor dyspnea and gained weight and started doing her usual work.

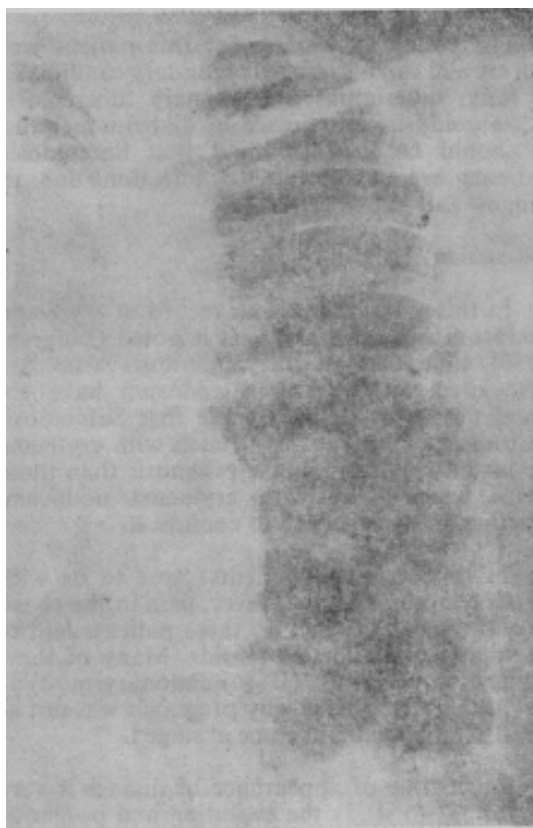


Fig. 1. X-ray of the chest of the patient shows BHL + pulmonary mottling (specially on left upper zone).

Comment: BHL -f- mottling of lung appeared (Stage II) after 3 years of appearance of BHL (Stage I)

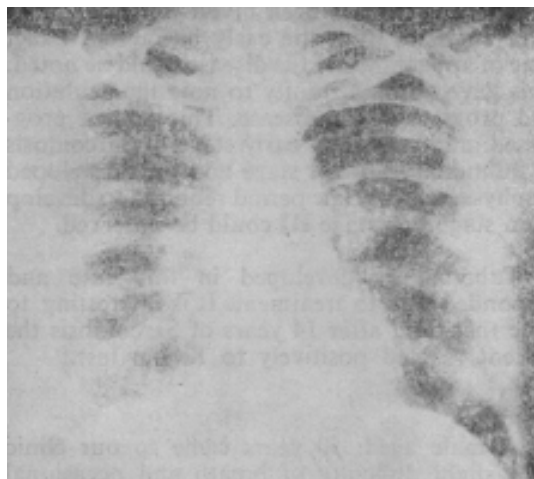


Fig. 2. X-ray of the chest shows BHL + mottling of lung on both sides of chest.

Comment : Note mottling of lung of both sides presents with BHL four years after appearance of BHL (Stage I.)

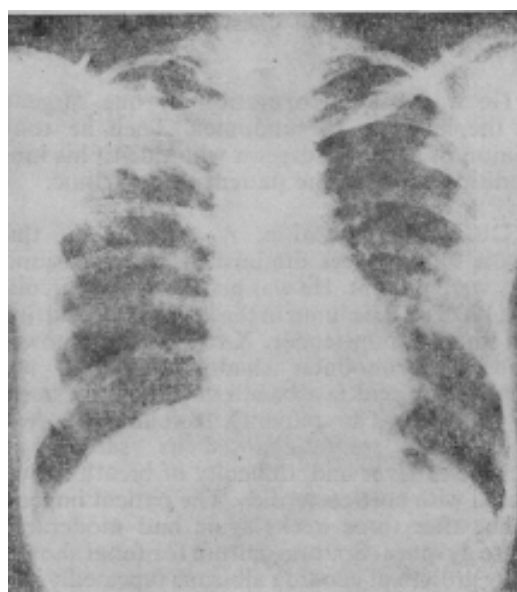


Fig. 3. X-ray of chest shows bilateral pulmonary fibrosis.

Comment : Note BHL has disappeared. This picture is fourteen years after first appearance of BHL.

She has been doing well for three and half years but got rise of temperature and cough with expectoration in 1974. Her sputum became positive for acid fast bacilli by smear and by culture. She had tuberculosis on top of sarcoidosis. She was treated with antituberculosis drugs and has been doing well.

Comment

This patient has been investigated for more than 16 years from the early beginning. Exact time of appearance of the disease could be noted. This gave an opportunity to note the evolution and progress of the disease. This patient progressed gradually from early stage of Sarcoidosis to pulmonary fibrosis stage and then developed emphysema. The time period required to develop from stage I to stage III could be observed.

Tuberculosis developed in this case and responded well to treatment. It is interesting to note that even after 14 years of Sarcoidosis the patient reacted positively to Kveim test.

Case 2

A male aged 50 years came to our clinic with slight difficulty of breath and occasional fever for five years and for the last five months there was a lump in the abdomen.

At a routine check up, five years back he had fibro-nodular shadows in x-ray of the chest. He has been medically observed for the five years and x-ray shadows of chest in x-ray remained almost the same.

He was advised operation by one surgeon for the lump in the abdomen. Then he took opinion of another surgeon who due to his lung condition referred the patient to our clinic.

During investigation, it was noted that breath sounds were diminished and occasional rales were present. He was negative to tuberculin test (100 Tu). The lump in the left hypochondrium was smooth, non-tender. X-ray of chest showed bilateral fibronodular shadows. Sputum was negative for acid fast bacilli and fungi by smear and culture. The patient's Kveim test was positive. He was diagnosed as Sarcoidosis. Because of fever and difficulty of breath he was treated with corticosteroids. The patient improved but after three weeks again had moderately severe dyspnea. Sputum culture for fungi showed heavy growth of *Candida albicans* repeatedly.

This was diagnosed as secondary candidiasis. The patient was treated with aerosol of Nystatin 40% propylene glycol in normal saline (Chakravariy 1967) for two weeks. Dyspnea disappeared and patient felt well. Repeated sputum cultures showed them to be negative for *C. albicans*. The patient had secondary candidiasis of lung which was cured with Nystatin aerosol.

After treatment with corticosteroid for six months the lump of the abdomen disappeared.

The patient was kept on corticosteroid and after 9 months (from the beginning) started fever and hacking cough with expectoration. Sputum examination showed it was positive for acid fast bacilli. The patient had pulmonary tuberculosis on top of Sarcoidosis. On being treated with antituberculosis drugs the patient was cured. The patient's dose of corticosteroid was reduced but was not completely stopped. After two years (from the beginning) the patient developed thirst and nocturnal urination repeatedly. Blood sugar was high and sugar was present in urine also. It was diagnosed as diabetes mellitus. The patient was treated with anti-diabetic drugs and blood sugar is normal now. The patient is doing well.

Comment

This patient's lump in the abdomen was considered as a tumour by a surgeon and was going to be operated. This lump seems to be a distorted spleen affected with Sarcoidosis which came to normal size and shape after being treated with corticosteroid.

The course of disease for this patient was stormy and ran a gamut of secondary candidiasis of lung, superimposed pulmonary tuberculosis on Sarcoidosis complicated by diabetes mellitus. It should be kept in mind that Sarcoidosis patients are susceptible to infection due to fungus and tubercle bacilli.

Discussion

In this study there is no record of erythema nodosum nor eye lesions. It is noted (Lofgren, 1953) that patients of carcooidosis who are associated with erythema nodosum have got good prognosis. It is possible that Sarcoidosis in India which is not associated with erythema nodosum may have worse prognosis than those cases which start with erythema nodosum. Further study is needed to confirm it.

In this series all patients came to us with some symptoms such as fever, pain in the chest, easy fatigueability etc. So these patients had to be treated with corticosteroids. Many of these patients came with BHL + Pulmonary mottling (*e.g.* Stage II). This is why prognosis was not as good as when patients came at stage I.

Exact time of appearance of disease is very important to study the evolution and prognosis of sarcoidosis. But unfortunately in India, the patient is treated in most cases as tuberculosis and treated for two to three years before they come for investigation for Sarcoidosis. Moreover, the routine check up is alAost non-existent

in India. As such exact time of appearance of the disease cannot be noted. Fortunately in case no. 1 and in a few more cases in this study, we could locate almost the exact date of appearance of the disease and could follow it up for more than fifteen years. So that an opportunity to note the evolution of the disease was obtained.

From follow up of 15 years of a few cases it was noted that from BHL (stage I) to reach BHL + pulmonary mottling stage (Stage II) it took 3-5 years and from BHL -f- pulmonary mottling stage (Stage II) it takes four years to reach the stage of pulmonary fibrosis (Stage III). It took 9 years for BHL to completely disappear and fibrosis of lung to set in. When lung biopsy of such patients of pulmonary fibrosis is taken, it is found that epithelioid granuloma pattern has been replaced by hyaline tissue.

Mikami *et al* (1971) representing Japan Sarcoidosis Committee reported that it took three years to reach stage III from stage II of the disease. 10% of the cases showed hilar and mediastinal lymphadenopathy for several years. 6% of these cases developed pulmonary fibrosis. In case 1, the fibrosis progressed further and emphysema set in. It is possible that, if the patient was treated at an early stage with corticosteroid further progress of fibrosis could be prevented. (Douglas 1964).

It appears that most important changes in Sarcoidosis are fibrosis of lung and pulmonary insufficiency. In this series two patients had progressive fibrosis of lung and died of insufficiency of lung. Three patients had also emphysema of lung. Regarding progress of the disease it should be remembered that Sarcoidosis patients are susceptible to tuberculosis and fungus diseases, Aspergilloma and secondary candidiasis are common. In this series, there are patients of tuberculosis followed by Sarcoidosis as also Sarcoidosis followed by tuberculosis. There was a time it was suspected that perhaps Sarcoidosis was an aberrant form of tuberculosis. The present concept does not agree with this. However, in cases of tuberculosis followed by Sarcoidosis group it is noted that high tuberculin reactor has become tuberculin non-reactor and in Sarcoidosis patients getting tuberculosis, the reverse reaction to tuberculin occurs.

Sarcoidosis patients with family history of diabetes mellitus, when treated with corticosteroid, may get diabetes mellitus. In two patients of this series there was sarcoidosis of lung with sarcoidosis of skin. Sarcoidosis of skin was cured with corticosteroid. Patients with hypercalcaemia with hypercalciuria in this series improved with corticosteroid. No case of kidney

affection nor nephrocalcinosis nor renal failure due to Sarcoidosis was noted in this series.

Summary

1. A total of 24 patients of sarcoidosis (21 proved by positive Kveim test and 3 by lung biopsy) were followed for 3-15 years.

2. 8 Patients had bilateral hilar adenopathy (BHL) *i.e.* Stage I of the disease. Hilar adenopathy disappeared or reduced in size in 6. (75%).

3. 6 Patients had BHL and mottling of lung (Stage II) of these 3 improved *i.e.* BHL reduced in size and mottling disappeared.

4. 10 Patients had pulmonary mottling with pulmonary fibrosis (Stage III). If these, 4 improved radiologically and two had progressive fibrosis of lung.

5. It took about 3-5 years to progress from BHL (Stage I) to BHL + mottling stage (Stage II) and about four years from BHL + mottling stage (Stage II) to pulmonary mottling + pulmonary fibrosis (Stage III) stage. It took about nine years for BHL to disappear completely in pulmonary fibrosis group of cases.

6. Three patients had pulmonary tuberculosis. One patient had tuberculous cervical lymphadenitis (proved by lymph gland biopsy) and one patient had secondary candidiasis of lung.

7. Of 5 hepatomegaly patients, liver came to normal size in 3 patients and in one of three patients with splenomegaly, spleen became normal in size with corticosteroid treatment. One patient with right bundle branch block in E.C.G. after treatment with corticosteroid did not show any abnormality in E.C.G. Two patients with sarcoidosis of skin were cured after treatment with corticosteroid.

One patient developed diabetes mellitus after corticosteroid therapy.

10 patients with hypercalcaemia showed serum calcium within normal limits after treatment with corticosteroids.

8. None of the patients had erythema nodosum or ocular lesions.

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INFLUENCE OF NON-SPECIFIC SENSITIVITY ON THE EFFECTIVENESS OF B.C.G. VACCINATION

V. SIVARAMAN,* V. UMASANKAR,** B.R. RAMALINGAM,*** B. SUGUNA****

The effectiveness of B.C.G. Vaccination might be expressed in terms of the number of cases prevented. Styblo and Meijer (1976) and Rouillon and Waaler (1976) have attempted a theoretical evaluation of the effectiveness of B.C.G. under various epidemiological assumptions. However they have not taken into consideration the influence of non-specific sensitivity. Such non-specific sensitivity is widely prevalent in our country and is believed to have an impact on the effectiveness of B.C.G. The aim of the present paper is to suggest a methodology to estimate the effectiveness of B.C.G. in a community where non-specific sensitivity is prevalent and to analyse its relation to cost benefit balance.

A. Impact of Non-Specific Sensitivity

Persons with small reactions to low dose of human type of tuberculin, but in whom strong reactions are elicited to a large dose are assumed to be infected with mycobacteria other than mycobacterium tuberculosis or other organisms. It is termed non-specific sensitivity and is reported to afford certain degree of protection against tuberculosis in animal experiments (Palmer and Long 1976). There is controversy regarding the efficacy of B.C.G. 'Vaccination given to individuals with non-specific sensitivity. Ferebee and Palmer (quoted by Hart 1967) felt that in hot countries where non-specific sensitivity is fairly common B.C.G. might have very little protective effect. The W.H.O. expert committee on tuberculosis in its ninth report (1974) was of the opinion that B.C.G. affords additional protection over and above that conferred by non-specific sensitivity. It is now proposed to analyse the influence of non-specific sensitivity on the effectiveness of B.C.G. Vaccination under two assumptions:-

Assumption-1

B.C.G. has no protective effect in a sensitized population.

Assumption-2

B.C.G. Vaccination increases the protective effect of sensitisation upto the level induced by

B.C.G. Vaccination alone. With this assumption and under epidemiological conditions obtaining in India the protective effect of B.C.G. in a sensitized population may be calculated in two stages as follows:

Stage-I : Protective Effect of Atypical Mycobacteria in India

By analogy with the protective effect of B.C.G. the protective effect of infection with atypical mycobacteria may be defined as the difference between disease incidence rates in the sensitized and non-sensitized as a percentage of the rate in the non-sensitized.

Gothi et al (loc. cit.) have found out the incidence rates among sensitized and non-sensitized. Applying their figures the following protective effects are derived.

Against culture positive disease:

$$(2.8-0.7) \times 100$$

$$2.8$$

Against culture positive and suspect diseases :

$$(5.9-2.3) \times 100$$

$$= 61\%$$

Stage-II: Protective Effect of B.C.G. in a sensitized population

(a) *Against culture positive disease* : Following the reasoning of Ten Dam et al (1976) if it were assumed that the protective effect of B.C.C. is 80% and that of atypical mycobacteria 75% then each 100 cases that would have occurred in a population free from any sensitivity would be reduced to 25 cases if this population were sensitized by atypical mycobacteria and to 20 cases if it were B.C.G. Vaccinated. In a sensitized population B.C.G. Vaccination could reduce each 25 cases to 20 the protection would be

$$\frac{25-20}{25} = 0.20 \text{ or } 20\%$$

From T.B. Sanatorium, Pondicherry.

* Medical Superintendent.

** Assistant Surgeon.

*** Assistant Surgeon.

**** Assistant Surgeon.

(b) *Against culture positive and suspect disease:* By the same method as (a) above the protection against culture positive and suspect disease would be

$$\frac{39-20}{39} = 0.49 \text{ or } 49\%$$

Material and Methods

Two cohorts of 100000 each aged 5 years and 10 years will be considered. All of them are presumed to be non-reactors to I.T.U. initially; H will be presumed further that the protection by B.C.G. is expected to last only 5 years (Frimodt Moller 1973). Cases arising from non-reactors to I.T.U. over a period of 5 years and cases prevented by B.C.G. will be estimated.

The calculation of the number of cases prevented involves the following steps:

1. Division of 100,000 non-infected children

(referred hereafter as NI) into 20 TU reactors and non-reactors. The abbreviations NI R20 and NI N20 respectively will be used hereafter for these categories. It is presumed that the group NI R20 is infected with atypical mycobacteria (Gothi et al 1977) and the group NI N20 is not infected with any mycobacteria.

2. Estimation of the cases likely to arise from the above: This is done by applying the incidence figures of Gothi et al (1976).
3. The number of cases prevented by B.C.G. is obtained under the two assumptions by adopting the different efficacies obtained earlier. Three categories of prevented cases are considered namely infectious INH sensitive, infectious INH resistant and suspect (abacillary).

Table I shows the epidemiological parameters used in the calculations. Steps (1) and (2) are shown in Table II.

Table I
Input data used in the calculations A. Epidemiological Parameters

	5 years	10 years	Reference
1. Prevalence of non-specific sensitivity (20 TU reactors)	48.7%	79.3%	Chakraborti et al (1976)
2. Prevalence of infection	4.9%	11.4%	
3. 5 year incidence of culture positive disease 20 TU reactors 20 TU non-reactors	0.07% 0.28%	0.07% 0.28%	Gothi et al (1976)
4. 5 Year incidence of cultures positive and suspect disease 20 TU reactors 20 TU non-reactors	0.23% 0.59%	0.23% 0.59%	Gothi et al (1976)
5. Incidence of Primary INH resistance (As a percentage of incidence of infectious cases)	6.5%	6.5%	NTI(1974)

B. Cost assumptions

	Value
	Rs.
1. Cost of non-infectious cases	226.89
2. Cost of infectious INH sensitive cases	325
3. Cost of infectious INH resistant cases	554
4. Cost of 10000 vaccinations	51100

Note: The values are adopted from Naganna K (1975)—for details see text.

Technical Parameters (Efficacy of B.C.G.)

Type of case	20 TU Reactors	20 TU non-reactors
(1) Against culture positive disease	20% 49%	80% 80%
(2) Against culture positive disease suspect disease		

Note : Mode of derivation explained in text.

Table II

Number of cases likely to arise from 100,000 non-infected in the absence of vaccination

	Non reactors to ITU among 5 Years old		Non reactors to ITU among 10 years old	
	20 TU reactors	20 TU non- reactors	20 TU reactors	20 TU non- reactors
1. Tuberculin status of cohort	48700	51300	79300	20700
2. 5 year incidence of C-f- and suspect disease	1 12.0a	302. 7b	182. 4a	122.0
3. 5 year incidence of C -f- disease	34. 1c	143. 6d	55. 5c	58.0
4. 5 year incidence of suspect disease	77 . 9	159.1	126.9	64.1

(a) @ 0.23 % of 48700 and 79300

(b) @ 0.59% of 51300 and 20700

(c) @ 0.07 % of 48700 and 79300

(d) @ 0.28% of 51300 and 20700

Note: The figures are shown upto 1 decimal for precision and also because otherwise totals may not add up.

Table III and IV give the number of cases prevented under assumption 1 and 2 respectively. A perusal of these tables shows that:

1. B.C.G. Vaccination of 100,000 children aged 5 years and not infected with mycobacterium tuberculosis, prevents 242 cases under assumption I and 297 cases under assumption 2.
2. B.C.G. Vaccination of 100,000 children aged 10 years and not infected with mycobacterium tuberculosis prevents 98 cases under assumption I and 187 cases under assumption 2.

It might therefore be logical to conclude that even if B.C.G. affords additional protection over and above that of non-specific sensitivity, the vaccination of younger age group yields higher benefit.

B. Cost Benefit Analysis

The adoption of a control measure depends not only on its effectiveness but also on its cost. B.C.G. Vaccination might be considered beneficial if it involves less expenditure than that likely to be incurred in finding and treating cases that might otherwise arise. The cost assumptions are shown in Table 1A and are based on the study of Naganna (1975).

1. Cost of Vaccination

The cost of vaccination is computed by first estimating the number of direct vaccinations required to cover 100,000 non-infected (this depends upon the prevalence of infection in different age groups). The cost of the total vaccination is then obtained since it is known that Rs. 51,100/- are required for 100,000 direct

Table III

Number of cases prevented by B.C.G. under Assumption-I.

	Non-reactors to ITU among 5 years old	Non-reactors to ITU among 10 years old	Non-reactors to ITU among 10 years old	
	20 TU reactors	20 TU non-reactors	20 TU reactors	20 TU non-reactors
1. Culture positive and suspect cases prevented	0	242	0	98
2. Culture positive INH sensitive cases prevented	0	107	0	46
3. Culture positive INH resistant cases prevented	0	7	0	3
4. Suspect cases prevented		128	0	49

Table IV

Number of cases prevented by B.C.G. under Assumption-II

	Non-reactors to ITU among 5 years old		Non-reactors to ITU among 10 years old	
	20 TU reactors	20 TU non-reactors	20 TU reactors	20 TU non-reactors
1. Culture positive and suspect cases prevented	556	242	89	98
2. Culture positive INH sensitive cases prevented		107	10	46
3. Culture positive INH resistant cases prevented	0	7	1	3
4. Suspect cases prevented	49	128	78	49

Prox. effect against C + 20%
C-f- and suspect 49%

vaccinations (Naganna 1975). The steps involved are shown in Table V.

Table V *Cost of**Vaccination*

	5 years	10 years
Number of direct vaccinations required benefit 1 00,000 non reactors	105152	112867
Cost of vaccinations	53733	57675

1. Cost of finding and treating cases:

For estimating the cost of finding and treating cases different costs have been attributed to different types of cases thus:

(a) *Cost of suspect case:* It will be assumed that these cases are given single drug therapy. Naganna (loc. cit.) has estimated that the total cost of finding a treatment case, cost of actual treatment with a single drug (INH) and loss of economic productivity works out to be Rs. 226.89. This figure will be applied to active non-infectious or suspect cases.

(b) *Cost of INH sensitive cases :* If active infectious INH sensitive cases are treated with single drug the conversion rate is 70 per cent.

Considering this the cost of INH sensitive cases works out to be about Rs. 325.

(c) *Cost of INH resistant cases* refers to primary INH resistant cases. In such cases single drug therapy has a very high failure rate and two drugs at least have to be given in which case the cost will be Rs. 554.

The cost of each prevented case is the same under both assumptions but as the total number of cases prevented under assumption 2 is higher the total cost of prevented cases is higher under this assumption.

By going through tables VI and VII one can appreciate that it is cheaper to vaccinate them to find and treat cases in 5 years old.

Table VI

Cost of cases pi-vented by B.C.G. under assumption-1

	5 Years	10 Years
Cost of culture positive INH sensitive cases prevented	34,775	14,950
Cost of culture positive INH resistant cases prevented	3,878	1,662
Cost of suspect cases preventec	29,042	11,118
Total cost of prevented cases	67,695	27,730
Cost of vaccination	53,733	57,675

Table VII

Cost of cases prevented by B.C.G. under Assumption-2

	5 Years	10 Years
Cost of culture positive INH sensisitive cases prevented	36,725	18,200
Cost of culture positive INH resistant cases prevented	3,878	2,216
Cost of suspect cases prevented	40,160	28,815
Total Cost of prevented cases	80,763	49,231
Cost of vaccination	53,733	57,675

C. Relation of Prevalence of Non-Specific Sensitivity and Infection to Cost Benefit Balance

It may be seen that the cost benefit balance is dependent upon the prevalence of infection with M. tuberculosis. It is possible to examine how far these prevalences influence the cost benefit balance under the two assumptions using simple algebraic methods:

Cost of vaccination:

Let y% be the prevalence of NI. The cost of vaccination of 100000 NI is $\frac{5110000}{\lambda}$

Cost of prevented cases:

Let x% be the prevalence of NI R20. The total cost of prevented cases is 1324 x under assumption 1 and 1324 x + 225 (100 -- x) under assumption 2 (The method of derivation of these expressions is shown in Appendix). B.C.G. Vaccination is a cheaper strategy if the cost of prevented cases is more than the cost of vaccination. This condition is mathematically expressed thus:

$$1. \quad 1324 x > \frac{5110000}{\lambda} \quad (\text{under assumption 1})$$

$$2. \quad 1324 x + 225 (100 - x) > \frac{5110000}{\lambda} \quad (\text{under assumption 2})$$

conditions may be graphically visualised by plotting the curves representing the equations:

$$1324 x = \frac{5110000}{\lambda}$$

and

$$1324 x + 225 (100 - x) = \frac{5110000}{\lambda}$$

Fig. 1 shows these curves and the prevalences under which it is cheaper to vaccinate than to find and treat cases. It may be observed that there is a fairly good range of prevalences of infection and non-specific sensitivity over which vaccination is cheaper.

It is also seen that vaccination even if it affords additional protection to sensitized population is a costlier strategy in the following extreme eventualities:

1. When the prevalence of NI is less than 38% or in other words when the preval-

ence of infection with *M. tuberculosis* is more than 62% , whatever may be prevalence of non-specific sensitivity.

2. When the prevalence of NI R20 is less than 20% or in other words when the prevalence of non-specific sensitivity is more than 80%, whatever may be prevalence of infection with *M. tuberculosis*.

Discussion

Chakraborty et al (1976) felt that the proper assessment of the effectiveness of B.C.G. Vaccination will be difficult in a population having non-specific sensitivity, thereby leaving the question open as to the most suitable age at which B.C.G. vaccination is to be given to the community with maximum effectiveness. The present paper attempts to answer the question through theoretical calculations based upon available data. The study confirms the impression of Chakraborty et al (loc. cit.) that vaccinations may have to be restricted to the population in earlier age groups say 0-9 years.

The cost of finding and treating cases applied in the present paper might be underestimates because the cost of radiology, cost of health visiting for irregular cases, cost of second line drugs and or hospitalisation in drug resistant cases have not been included. The indirect benefit brought about by B.C.G. by breaking the chain of transmission has not been taken into consideration; on the whole the data used put B.C.G. at a disadvantage yet it is seen that it does prove cheaper in many circumstances.

It might therefore be concluded that B.C.G. is a cheap and effective tool in the fight against tuberculosis even in areas with non-specific sensitivity, within certain prevalence limits of infection and non-specific sensitivity.

Summary

The effectiveness of B.C.G. Vaccination may be measured in terms of number of cases prevented. The number of cases prevented by

The results indicate that B.C.G. might be a cheap and effective tool in the fight against tuberculosis even in areas with non-specific sensitivity within certain prevalence limits of infection and non-specific sensitivity.

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APPENDIX

Cost of Vaccination

To benefit "Y" number of NI, 100 direct vaccinations have to be given.

To benefit 100000 NI 100 x 100000 vaccinations have to be given. ----- ●●●

The cost of 100000 vaccinations is Rs. 51100.

The cost of 100 X 100000 vaccination is

$$\frac{51100 \times 100 \times 100000}{100000 \times Y} = \frac{5110000}{Y}$$

Cost of prevented cases: Assumption 1

Let x% be the prevalence of NI N20.

NI per 100000 total children = 1000 x

Number of C + cases arising from the above

$$\frac{1000 \times X \times 0.28}{100} = 2.8 x$$

Number of INH sensitive cases amongst the above:

$$\frac{2.8 \times X \times 93.5}{100} = 2.618 x$$

Number of INH sensitive cases prevented:

$$\frac{2.618 \times X \times 80}{100} = 2.0944 x$$

Total cost of INH sensitive cases prevented:
 2.0944 x X 325 = 680.68 x (1)

Number of INH resistant cases :

$$\frac{2.8 \times X \times 6.5}{100} = 0.182 x$$

Number of INH resistant cases prevented:

$$\frac{0.182 \times X \times 80}{100} = 0.1456 x$$

Total cost of INH resistant cases prevented:

$$0.1456 \times X \times 554 = 80.6624 x \quad (2)$$

Number of C + and suspect cases prevented:

$$\frac{1000 \times X \times 0.59}{100} = 5.9 x$$

Number of suspect cases = 5.9 x — 2.8 x = 3.1x

Number of suspect cases prevented:

$$\frac{3.1 \times X \times 80}{100} = 2.48 x$$

Cost of suspect cases prevented:

$$2.48 \times X \times 226.89 = 562.6872$$

The total cost of prevented cases is the sum of (1), (2) and (3) above.

Therefore 1324x (by rounding up)

The equation of cost benefit balance is:

$$1324x = 5110000 \quad \text{i.e. cost of prevented cases is}$$

the cost of vaccination. The equation is represented by an hyperbola passing through the points:

$$x = 100 \quad x = 40 \quad x = 50 \quad x = 60 \quad x = 38.60 y$$

$$= 38.60 \quad y = 96.49 \quad y = 77.19 \quad y = 64.33 \quad y = 100$$

Assumption 2

Under assumption 2, in addition to cases among NI N20, BCG is able to prevent a proportion of cases arising from N2 R20.

$$\text{Number of NI R20 per 100000} = 100000 - 1000x = 1000(100-x)$$

C+ cases arising among the above

$$\frac{1000(100-x) \times 0.07}{100} = 0.7(100-x)$$

Number of INH sensitive cases :

$$\frac{0.7(100-x) \times 93.5}{100} = 0.6545(100-x)$$

Number of INH sensitive cases prevented (20% protection)

$$\frac{0.6545(100-x) \times 20}{100} = 0.1309(100-x)$$

$$\text{Cost of INH sensitive cases prevented: } 0.1309(100-x) \times 325 = 42.5425(100-x) \quad (1)$$

Number of INH resistant cases :

$$\frac{0.7(100-x) \times 6.5}{100} = 0.0455(100-x)$$

Number of INH resistant cases prevented:

$$\frac{0.0455(100-x) \times 20}{100} = 0.0091(100-x)$$

Cost of INH resistant cases prevented:

$$0.0091(100-x) \times 554 = 5.0414(100-x) \quad (2)$$

C -f- and suspect cases arising among NI R20

$$1000(100 - x) \times 0.23 = 2.3(100 - x)$$

Suspect cases arising among NI R20

$$2.3(100 - x) - 0.7(100 - x) = 1.6(100 - x)$$

Cases prevented among the above (Protection 49%)

$$\frac{1.6(100 - x) \times 49}{100} = 0.784(100 - x)$$

Cost of suspect cases prevented:

$$0.784(100 - x) \times 226.89 = 177.88176(100 - x) \quad (3)$$

Cost of cases prevented among NI R20 is the sum of (1), (2) and (3) viz. $225(100 - x)$ (By rounding up).

Under assumption (2)

Total cases prevented=cases prevented among NI N20

+cases prevented among NI N20

The total cost of prevented cases is therefore:

$$1324x + 225(100 - x)$$

The equation of cost benefit balance is

$$1324x + 225(100 - x) = \frac{5110000}{}$$

It is represented by an hyperbola passing through the points:

X=100	x = 30	x = 30
y = 92.12	y = 76.89	y = 92.12
x = 50	x = 50	y = 100
y = 65.98	y = 57.78	x = 26.02

CASE REPORTS

CEREBRAL ANGIOGRAPHY IN TUBERCULOUS MENINGITIS

HEM LATA GUPTA,* RANI DHEMAN** and ARATI MITRA***

Baumgarten for the first time described arterial changes in acute tubercular meningitis (1881) and these changes were demonstrated radiologically by Grietz (1964). Lehrer (1966) described the triad comprising of wide sweeping pericallosal artery and narrowing of the supraclino-oid portion of the carotid artery and narrowing or occlusion of smaller or medium sized arteries with scanty collaterals. The resultant narrowing and occlusion cause infarction of localised areas of the brain and account for some neurological complications like hemiplegia. There are very few reports where the angiographic findings have been correlated with the clinical findings (3-5) The aim of the present study is to find out the cerebral arterial involvement in cases of tubercular meningitis and its correlation with the neurological involvement and prognosis.

Material and Method

50 cases of tuberculous meningitis with or without neurological deficit admitted to this hospital from 1970-1974, were selected for the above study. All the cases were females above the age of 12 years. Diagnosis of tuberculous meningitis was based on strong clinical grounds and on evidence of tuberculosis elsewhere in the body, biochemical, cytological examination of CSF. In 10 cases AFB was demonstrated from CSF either on direct examination or culture. Response to antitubercular treatment further supported the diagnosis. In all the cases cerebral angiography was done. 50% of the cases with arterial involvement were put on antitubercular drugs and corticosteroids while remaining 50% (12 cases) were put on antitubercular treatment only.

Results

All the patients were females between the age group of 12-50 years (Table 1). 76% of the cases had systemic tuberculosis; out of this pulmonary tuberculosis was the commonest 44% (Table 2). Out of 50 cases 24 cases were quite seriously ill and had either major neurological deficit or confusional state with or without cranial nerve palsies. The commonest neurological deficit was confusional state with cranial nerve palsies.

Sixth, 7th and third nerves were the commonest to be involved. Ten patients had hemiplegia and 3 of them had Parkinsonian symptoms during the course of the disease, and one had loss of vision on the contralateral side. Decerebrate state was present in 4 cases, while 5 cases had

Table 1

Age Distribution

	Number of cases
12—20	15
21—30	26
31—40	7
41—50	2
Total :	50

Table 2

Associated Systemic Tuberculosis

	No. of cases
Pulmonary Tuberculosis Abdominal	22
Tuberculosis Spinal Tuberculosis	2
Lymphadenitis	4
No evidence of other systemic tuberculosis	10
Total:	50

From Lady Hardinge Medical College & Hospital, New Delhi.

* Lecture in Medicine.

** Senior Residents.

***Senior Residents.

cerebellar ataxia. 2 cases had signs of raised intracranial tension and 12 cases had only signs of meningeal involvement. Examination of the fundus revealed choroid tubercles in 6 cases, papilloedema in 4 cases and optic atrophy in one case.



Left carotid angiogram showing block of the left middle cerebral artery.

Cerebral angiography revealed arterial narrowing in 24 cases (48%). 2 cases had narrowing of the internal carotid artery while in rest the middle or anterior cerebral arteries were narrowed. All the cases of hemiplegia and cranial nerve palsies had narrowing. One case with internal hydrocephalus had hydrocephalic pattern of blood vessels. Good correlation between the clinical findings and arterial narrowing was observed (Table 5).

In another 24 patients normal arterial vasculature was demonstrated, 50% of the cases in this group were with no neurological deficit and with flacid paraplegia. There was no correlation between the arterial narrowing and the rise of CSF proteins and cells. (Table 3, 4)

Prognosis was poorest in patients with decerebrate state (3/4 died), 3 patients with hemiplegia and cranial nerve palsy each expired (Table 5). The total deaths were 10 (20%), 26 patients recovered completely with no neurological deficit, most of these patients had no deficit initially. 4 cases with hemiplegia and 3 hemiplegia with Parkinsonian features recovered (Table 7).

On analysing the angiographic findings it was found that out of 24 patients having arterial

Table 3
CSF Protein — Arterial Narrowing

Protein Level in C.S.F.	Arterial narrowing	Normal	Doubtful narrowing and spasm
Upto 100 mgm	7	7	
101—150	7	8	1
151—200	3	2	—
201—300	2	4	—
301—400	2	—	1
401—500	1	2	—
501—600	1	—	—
601—700	1	1	—
Total	24	24	2

Table 4
CSF Cells and Arterial Narrowing

Cellular response in CSF	Arterial narrowing	Normal	Doubtful narrowing/ spasm
5—50	5	7	1
51—100	6	2	—
101—150	3	2	—
151—200	—	4	—
201—300	1	—	1
301—500	5	4	—
401—500	—	2	—
501—600	—	1	—
601—1000	1	1	—
Total	24	24	2

Table 5

Correlation between the neurological state and arterial narrowing

Neurological State	Arterial Narrowing				
	Total Cases	Present	Absent	Doubtful	Spasm
Hemiplegia	7)	10	—	—	—
Hemiplegia with Parkinsonism	3)				
Spinal flaccid paraplegia	7	—	7	—	—
Confusional state with cranial nerve palsies	10	10	—	—	—
Decerebrate state	4	3	—	1	—
Cerebellar ataxia	5	1	3	—	1
Internal Hydrocephalous	2	—	2	—	—
No Neurological deficit	12		12		
Total	50	24	24	4	1



Fig. 3.

Left carotid angiogram showing the narrowing of the left middle cerebral artery in its middle portion.

Table 6

Neurological state and prognosis

	Dead	Residual Deficit	Recovered
		1	3
sonism		—	3
		3	3
cranial to nerve palsies		3	4
		1	—
		4	1
		0	2
		—	12
		12	28

Table 7

Arterial Narrowing and Prognosis

	Dead	Residual deficit or Improving	Recovered or
Arterial narrowing	9	7	8
No arterial narrowing	1	5	18
Doubtful narrowing			1
Spasm			1
Total:	10	12	28

narrowing 9 expired, while 7 had residual damage and 8 recovered. In cases where no arterial lesion could be demonstrated only one expired and 5 had residual damage (18 recovered. Table 7). No significant difference was observed between the two groups of patients who were put on antitubercular treatment alone and another group who had anti-TB drugs along with cortico steroids.

Discussion

Vascular changes consisting of arterial narrowing of large and small vessels with or without occlusion are frequently seen at autopsy in tubercular meningitis. Lot of literature is available on this, but the literature on cerebral arterial involvement in life in tuberculous meningitis is meagre. In 1964 Greitz demonstrated narrowing of the arteries in life in three cases. In the present study the angiographic findings revealed occlusion or narrowing of either middle or anterior cerebral arteries in 22 (44%) cases while 2 patients had internal carotid artery narrowing. One of the patients had clinical evidence of blockage of internal carotid artery in the form of contralateral diminished vision. Most of these patients had either hemiplegia (7), hemiplegia with Parkinsonian symptom (3), or confusional state with cranial nerve palsies (10) In 24 cases no arterial lesion could be demonstrated and most of the cases did not have any neurological deficit clinically also. 3 cases had cerebral ataxia and 7 had flaccid paraplegia.

It has been suggested that vertebral angiography is more helpful in patients with cerebellar signs.

In 1967 Wadia found arterial narrowing in 17 cases out of 33 patients. Three of the cases with arterial narrowing expired. In 1970 Misra could demonstrate a very high incidence of hydrocephalus (12 out of 13 cases) while occlusions of anterior or middle cerebral arteries were present in 10 cases. The high incidence of hydrocephalus is due to the fact that he studied children, while the present study, comprised of adults only. Mathew 1970 found that the commonest sites of occlusion were the supraclinoid portion of the carotid artery and proximal portion of anterior and middle cerebral arteries.

There is controversy about the collaterals in T.B.M. Lehrer (1966) described scanty collateral. On the other hand Mathews (loc cit) described net like clusters of the vessels in the feigion of basal ganglion, base of the brain and transdural external internal carotid anastomoses. In addition, there was altered architecture of cortical vasculature and early veins could be seen, early veins were attributed to associated infarction. In the present series all the 3 cases who developed Parkinsonian features during the course of the diseases recovered completely which supports the concept of development of collaterals in the region of the basal ganglion.

In the present series there was overall mortality rate of 22%. while same mortality rate had been reported by Misra 1970 also. In cases where arterial lesions were severe the prognosis was poor. This has been shown to be due to resultant infarction of the area supplied by the occluded artery (Thomas 1977).

The corticosteroid did not seem to help much in arterial involvement in these cases as there was no difference between the mortality and recovery in the two groups.

Summary

(1) Cerebral angiography was done in SOcases of T.B.M. at various stages, either unilateral or bilateral. Depending on neurological involvement. All the cases were female between the age groups of 12 to 50 years.

(2) No significant correlation was observed between the CSF findings and the cerebral arterial involvement. (Table 3, 4)

(3) There was definite correlation between the arterial involvement and neurological deficits, (Table 5). All the cases of hemiplegia had involvement of middle cerebral artery and other 10 cases of confusional state and cranial nerve palsies also had arterial involvement.

(4) It was observed that the cases who had arterial involvement had poor prognosis as 9 patients expired out of 24 cases whereas only one patient expired from the cases (24) in which no arterial involvement could be demonstrated.

(5) Angiography can be useful in differentiating between the space occupying lesions and arterial involvement.

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ISOLATED GASTRIC TUBERCULOSIS

ARUN KAKAR,* R.C. ARANYA,** and S.K. NAIR***

Tuberculosis of the stomach is a rare clinical entity especially in the western countries. Though, it is generally associated with an advanced pulmonary lesion, primary involvement is known to occur. The first description of gastric tuberculosis, according to Walters³ was given by Barkhausen in 1824. A number of isolated case reports of gastric tuberculosis have appeared in the English literature mainly from Africa and India^{2,3,*,5,6}. Only two cases have been reported from the western countries during the last 10 years^{7,8}.

Case Report No. 1

A.K., a 35 years old tailor, was admitted with a 7 months history of epigastric pain with vomiting. In addition patient gave history of a ball of wind moving in the central abdomen and had lost 8 lbs. in weight. There was no history of haematemesis, melena jaundice or pulmonary tuberculosis.

Physical examination revealed a young emaciated male with marked pallor. The positive findings on abdominal examination were: tenderness in epigastrium and a left to right visible peristalsis. No mass was palpable. There was no hepato-splenomegaly or free fluid in the peritoneal cavity.

Laboratory data demonstrated Hb. 10.5 gms%; white cell count of 6800/cml; erythrocyte sedimentation rate 35 mm/hour and a haematocrit value of 40%. Urinalysis was normal with a specific gravity of 1032. Serum electrolytes estimation revealed Na 136 mEq/litre, K 5mEq/litre and chloride-86 mEq/litre. Stool test for occult blood was negative.

The skiagrams of the chest and abdomen were normal. Barium meal study of upper gastrointestinal tract showed a filling defect in the pyloric antrum with an evidence of gastric stasis.

The patient was operated upon after an initial preparation by gastric lavage with normal saline and administration of fluids intravenously.

Operative findings revealed a firm mass in relation to distal 1/3 of the stomach. The mass

was adherent to pancreas. There were multiple nodes in the greater and lesser omentum. All the other intra abdominal structures were normal. A partial gastrectomy with gastro-jejunostomy was done.

Histopath report — Tubercular granuloma with Langhan's type of giant cells and epithelioid cells in the wall of the stomach (Fig. 1).

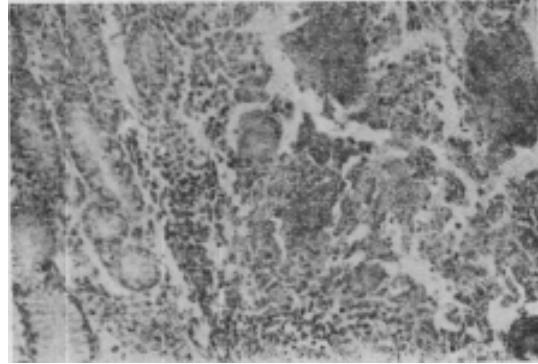


Fig. 1

Tuberculoma of the Stomach wall showing Langhan's Giant cells and Epithelioid cells HE x 225.

Case Report No. 2

A 30-year old lady was admitted with 3 years history of vomiting off and on, pain in upper abdomen and weight loss. There was no history of haematemesis, melena or jaundice.

The positive finding on physical examination were pallor, left to right visible peristalsis in upper abdomen with tenderness and a succussion splash. The gynaecological examination was normal.

Routine blood examination revealed a Hb. of 8.0 gms% with total leucocyte count of 7200/cml. The blood sedimentation rate was 65mm/Lst hour. Urine analysis was normal.

The skiagram of the chest did not show any active or healed Tuberculosis. Barium study of

From M.A.M. College, New Delhi

*Demonstrator in Surgery

**Asstt. Prof. in Surgery

***Prof. of Surgery

dilatation. The duodenal bulb could not be demonstrated in this study.

After a preoperative preparation consisting of gastric lavage with normal saline and administration of electrolyte solution and blood, an exploratory laparotomy was done. At operation a nodular mass was found in the wall of the stomach in the pyloric region. The stomach proximal to this mass showed gross dilatation and hypertrophy of the muscular coat with enlarged regional lymph nodes. Rest of the abdomen was normal. A partial gastrectomy with gastro-jejunostomy was done.

Histopath report — Non caseating tubercular granuloma of the stomach.

Comments

Primary gastric tuberculosis is very rare. It is generally secondary to pulmonary tuberculosis. The incidence of involvement of stomach in patients with pulmonary disease varies from 0.02 to 0.5%. * Though tuberculosis infection of the small bowel is not an uncommon condition stomach is rarely involved. The factors which protect the stomach against invasion by tubercle bacilli are bactericidal property of the hydrochloric acid, an intact gastric mucosa, scarcity of the lymph follicle in the wall of the stomach and rapid emptying of its contents.¹⁰

Gastric tuberculosis is known to occur in patients with pernicious anaemia and malignancy of the stomach.¹¹ The possible explanation is the anacidity and chronic gastritis which render the Stomach more susceptible to tuberculous infection. Similarly, superimposed infection by tubercle bacilli is known to occur in patients with peptic ulcer diathesis of stomach and duodenum.

Pathologically 80% of the gastric lesions are ulcerative in nature and occur in the pyloric region. Miliary tubercles, tuberculous tumor, pyloric stenosis and tuberculous lymphangitis are rare.

The clinical picture of gastric tuberculosis mimics gastritis, peptic ulcer and carcinoma. Pain in abdomen especially after meals, vomiting and weight loss are the salient clinical features seen in the reported cases. Bleeding from ulcerative lesions is not uncommon. The complications of perforation is exceedingly rare as these ulcers seldom penetrate the muscular coat of the stomach. Both cases reported here presented with clinical features of pyloric stenosis.

There is no pathognomonic roentgenographic

appearance of tuberculous lesions of the stomach. Radiologically it is difficult to differentiate them from gastric neoplasms. Both the cases reported here were also initially diagnosed as malignant tumors of stomach.

A correct pre-operative diagnosis is seldom made in these cases. The various features that may help in arriving at a correct diagnosis are (a) high suspicion index in young patients in developing countries (b) clinical or radiological evidence of tuberculosis elsewhere in the body (c) Radiological evidence of contiguity between the gastric and duodenal lesions.

Since a correct diagnosis is established only on histopathological examination a partial gastrectomy followed by antitubercular chemotherapy is advised as the treatment of choice.

Summary

Gastric tuberculosis is a rare condition. The reported incidence is 0.02-0.5% of cases of pulmonary tuberculosis. The rarity of gastric involvement is due to the bactericidal property of gastric acid and paucity of lymphoid follicles in the stomach. This is suggested by its more frequent association with pernicious anaemia and chronic gastritis.

The lesion is located in the pyloric antrum in 80% of cases and symptoms resemble those of gastric carcinoma or pyloric stenosis.

Two cases treated during the last ten years have been reported. The difficulty of pre-operative diagnosis has been emphasised and literature reviewed.

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TUBERCULOSIS OF STOMACH AND DUODENUM

C.C. MOHAN REDDY,* P. SARADA,** G. SUVARNA KUMARI,*** M. SIKANDER HAYATH,****
and M. JAWADALI KHAN*****

Tuberculosis of the stomach is a rare disease. The mucous membrane may be involved without producing symptoms in miliary tuberculosis. In advanced pulmonary tuberculosis a tuberculous ulcer may form near the pylorus. Hurst (cited by Hint, 1956) had recorded one case of chronic gastric ulcer and one of carcinoma where histopathology of the specimen after operation showed secondary tuberculous infection. Boyd (1961) stated that tuberculosis of the stomach is a great rarity. The lesion takes the usual form and may give rise to single or multiple ulcers.

At the Mayo Clinic only 7 tuberculous cases were found in 20,000 gastric lesions recorded (Aird, 1956). Jain and Agarwal (1965) having reviewed the literature cited a case where a big area of the stomach was affected without involving the regional lymphnodes and not associated with pulmonary tuberculosis.

Tuberculous ulcer of the stomach was first mentioned by Barkhausen in 1824 and fully described by Litter in 1876. Tuberculosis of the stomach is a rare condition, it was found to be present in 0.16% of 7,000 routine postmortems (Palmer, 1950).

Duodenal tuberculosis, even as a part of advanced generalised tuberculosis is an unusual finding. In most of the reports, the duodenal lesions were found incidental to advanced terminal tuberculosis, diagnosed usually on postmortem examination. Duodenal tuberculosis is said to be thirty times rarer than intestinal tuberculosis (Aird, 1957).

Being rare it was considered worth while to report two postmortem cases of tuberculosis of stomach and duodenum, which were recorded among 1736 routine postmortems conducted in the Department of Pathology, Kurnool Medical College, Kurnool (Andhra Pradesh) in a period of 10 years (1967-1977).

Case No. 1

A Hindu female aged about 25 years was

admitted in Government General Hospital, Kurnool on 13-9-1969 with the complaints of cough and breathlessness since one year and pain in the epigastric region for the last 4 months. Epigastric pain used to increase after taking meals. No history of fever or any vomiting. On physical examination she was found to be poorly built and nourished. She was anaemic and clubbing of fingers present.



Fig. 1

Gross Photo showing a solitary tuberculous ulcer of the stomach and duodenum (Case No. 1).

Deep tenderness was present in the epigastric region but no lump was palpable. Liver was just palpable below the right costal margin.

Investigations:

Hb. 6 gms%. T.C. 6050/c.m.m. D.C. P₄₆
L₂₈ E₆ M₂, E.S.R. 40 m.m./hr.

Urine and stool examinations were normal. Sputum for A.F.B. was negative. The patient became bad and expired on 16-9-1969 and the postmortem was conducted on 20-9-1969.

Autopsy Findings (Autopsy No. 159/69);

Body of a female aged 25 years grossly

From Kurnool Medical College, Kurnool, (Andhra Pradesh)

*Asstt. Prof, of Pathology

** Asstt. Professor of Pathology

***Professor of Pathology

****Asstt. Prof, of Pathology

*****Asstt. Prof, of Pathology

anaemic with mild pitting edema of lower limbs and clubbing of the fingers.

des were enlarged which showed caseation on cut section.

Both lungs showed fibro-caseous tuberculosis and pulmonary edema.

G.I. Tract

Stomach : Cardiac end of the stomach showed an ulcer of 2x3 cm. size with a sinus in the base of the ulcer leading to the lymphnodes of the stomach.

Duodenum : Second part of the duodenum shows an irregular ulcer of 4x3 cm. size with a sinus in the base leading to lymphnodes. Jejunum showed two transverse ulcers of size 2x0.5 cm. Ileum also revealed few ulcers of the players patches. Caecum shows an ulcer of 5x3 cm. and is adherent to surrounding lymphnodes.

Mesenteric and omental lymphnodes are enlarged, firm and matted. Cut section showed extensive caseation. Histopathological examination of the stomach, duodenum, jejunum, ileum lungs and lymphnodes showed typical tuberculous granulomatous reaction. Rest of the organs were normal.

Case No. 2

A Hindu male aged about 31 years was admitted in the Government General Hospital, Kurnool on 24-5-1972 with the complaints of epigastric pain and distension of abdomen after meals for the last one year.

There was swelling in the neck with discharging sinuses of 3 months duration.

Physical examination revealed an emaciated and anemic male who had slight tenderness in the epigastrium. No investigations were undertaken because patient expired immediately after admission. The postmortem was done on 28-5-1977.

Autopsy Findings (Autopsy No. 42/77) :-

Body of a male aged about 30 years, emaciated, grossly anemic. Multiple ulcers of varying sizes from 2x2 cm. to 5x5 cm. present on both sides of the neck. Clubbing of the fingers present. Cyanosis present. On reflecting the skin over the ulcers revealed caseous cheesy material. On left side of the neck the lymphnodes were enlarged and matted, measuring 2x1 cm. which on cut section showed caseation.

Abdomen : Peritoneal cavity contained 50 c.c. of seropurulent fluid. Mesenteric lymphno-



Fig. 2

Gross photo showing multiple small tuberculous ulcers near the cardiac end of the stomach (Case No. 2)

Stomach : Near the lesser curvature over the mucosal aspect there were four ulcers from the middle to the cardiac end (Fig. 1). One of the ulcer was perforated. Each ulcer measured 2x2 cm with thickened and irregular ragged walls.

Duodenum : Over the posterior wall close to the anterior surface on the first part of duodenum, there was a chronic ulcer of size 2x1.5 cm. with thickened and overhanging edges (Fig. 1). The lower edge is smooth and it was perforated.

Lungs showed bilateral pulmonary fibro-caseous tuberculosis. Rest of the organs were normal grossly.

Histopathological examination of the stomach and duodenum revealed typical tubercle formation with caseation, Langhan's giant cells and epitheloid cells. A.F.B. stain was negative.

The sections of the cervical, mesentric and tracheobronchial lymphnodes, liver, spleen, kidneys and both testis revealed miliary tuberculosis.

Discussion

The incidence of the disease varies from 0.36 to 2.3% in patients with pulmonary tuberculosis

although the cases have been reported without any clinical Koch's lesion elsewhere in the body (Chatterjee and Dutt, 1955, Jain and Agarwal, 1965 and Sharma, 1967). Isolated cases of gastric tuberculosis have been recorded in Russian (Kuzionov and Polinkova 1973) and Indian (Bhargava and Sekhon 1973) literature.

56 per cent of the patients with pulmonary tuberculosis have been found to have associated tuberculosis of some part of the gastrointestinal tract (Abrams and Holden, 1950). Novis et al (1973) recorded one case out of 59 patients with gastrointestinal tuberculosis to have involved the stomach.

The clinical picture of gastric tuberculosis simulates chronic gastritis, peptic ulcer or carcinoma. Post prandial pain in the abdomen, distention and discomfort, vomiting and loss of weight may be the presenting features of these patients (Hoon et al 1950). In 50 per cent of the cases there is a palpable mass and in about half of these, there is evidence of tuberculosis elsewhere in the body (Chazan and Aitchison, 1960 and Gaines 1952).

Pathologically Broders (1917) have described six types of lesions -

- I. Tubercular ulcer of stomach, which may be single or multiple, larger or small.
- II. Miliary tuberculosis of stomach
- III. Hyperplastic or infiltrative type
- IV. Solitary tuberculoma
- V. Tuberculosis pyloric stenosis VI. Tubercular lymphangitis of the stomach.

Gastric analysis to differentiate tuberculosis from Carcinoma of the stomach has also been of little help. Palmer (1950) reported achlorhydria in 25 per cent and hypochlorhydria in 57 per cent of the patients. Bhargava and Sekhon (1973) found gastric acidity within normal limits in their cases.

Roentgenographic studies are of little help in arriving at diagnosis (Gaines et al 1952) and there are no characteristic features of gastric tuberculosis (Kanekerkh 1959) and the picture may be similar to that of a gastric neoplasm. Partial gastrectomy followed by antitubercular chemotherapy has been advised as the treatment of choice (Stirk 1968).

Duodenal tuberculosis even as a part of advanced generalised tuberculosis is an unusual finding. Feldmen (1957) found that duodenal lesions occurred in 0.5% of tuberculous autopsied cases. The reasons for this rarity of gastro-duodenal tuberculosis as given by Ostnim and

Seaber (1948) are — Rapid expulsion of bacilli from stomach by peristaltic waves, gastric acidity relative scarcity of lymphatics in the wall, presence of intact mucosa and inherent resistance. Kossick (1969) however suggested that the tubercle bacilli enter via an preexisting ulcer to give rise to a lesion in the stomach.

Ferreira and Silvano Filho (1966) and Gupta (1971) have described four forms of Duodenal tuberculosis:- Ulcerative, hyperplastic, infiltrative and obstructive. Ulcerative and infiltrative forms remain asymptomatic for a long time.

Abrams and Holden (1964) described haemorrhage, perforation, obstruction and fistula formation as the complications of this lesion. Massive haemorrhage has been reported by Chazan and Aitchison (1960) and Bhargava and Sekhon (1973). In the present series two cases of tuberculous ulcers of the stomach and duodenum were on record among 1736 autopsies conducted in a period of 10 years.

The two cases showed associated pulmonary and intestinal tuberculosis.

In case No. 1 there was associated bilateral fibrocaceous pulmonary tuberculosis with tuberculosis of jejunum, ileum, caecum and mesenteric lymphnodes.

In case No. 2 there was associated bilateral pulmonary tuberculosis with involvement of cervical mesenteric and tracheobronchial lymphnodes and miliary tuberculosis of liver, spleen, kidneys and testis.

The first case showed a single small tuberculous ulcer at the cardiac end of the stomach and the second case showed multiple (4) small tuberculous ulcers near the cardiac end of the stomach. In the present series gastric analysis was not done as the patients lived for a shorter time after admission.

The two cases in the present series showed tuberculous ulcers of the stomach and duodenum. In one case the first part of duodenum showed a chronic ulcer of 2x1.5 cm. on the posterior wall with over hanging edges. The lower edge is smooth and perforated with recent peritonitis. In the second case second part of duodenum (post part) showed an irregular ulcer of 4x3 cm. with a sinus in the base leading to lymphnodes.

Summary

Two Autopsy cases with tuberculous ulcers of the stomach and duodenum were presented

in one case the ulcer showed sinus formation and in other case the ulcer showed perforation with peritonitis. Both cases showed associated tuberculosis in lungs and in other organs.

Acknowledgements

We wish to convey our thanks to Sri M. Venugopal Raju, Stenographer, Kurnool Medical College, Kurnool for typing this article and to Sri Haricharnapathi, Photographer, Kurnool Medical College, Kurnool for the photographic work.

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BOOK REVIEW

TUBERCULOSIS — CASE FINDING AND CHEMOTHERAPY by K. TOMAN

World Health Organisation, Geneva, 1979.

Tuberculosis control is based essentially on three activities *viz.* case finding, treatment and immunization. Of these three, the first two are undoubtedly more important and this book deals with these two components of a control programme.

The book is written in an unorthodox style in the form of questions and answers, as against the usual continuous style of a text book. This style obviously has its advantages and disadvantages. The main advantage, as very rightly pointed out by the author, is that the reader can easily get an answer to a problem. The disadvantage is the large number of to and fro references without which the answer is not complete and which break the continuity.

The book is not exhaustive. It skips over many details and therefore may not appeal to the specialist. However, it contains most of the essential information which is likely to be needed by an undergraduate medical student, a general practitioner or a 'generalist' in the peripheral health institution who has to implement an integrated health programme. It is good that controversies have been avoided.

There are a few omissions which detract from its merit. For example, whereas passive case finding which aims at providing an efficient diagnostic service for patients who attend the health facility voluntarily has been well covered, active case finding dealing with an active search in the community for unknown cases, who, because the symptoms are not pressing enough do not attend the diagnostic centres voluntarily has not been given the consideration it deserves. Similarly, a case has been taken as one where diagnosis is bacteriologically confirmed. While

this definition is adequate for epidemiological purpose, it is totally inadequate for clinical purposes. There is practically no country in the world where abacillary cases are not being diagnosed as active and tuberculous and treated as such. Therefore, under passive case finding, procedures and criteria for diagnosing such cases with negative sputum should have been dealt with in some detail. Impracticability of miniature radiography for active case finding has been rightly stressed. A search for active cases through selective symptomatic surveys should have been stressed a little more.

Some statements may also not be entirely acceptable. For example, the author says that case finding is easy but treatment is difficult. On the other hand, it is the finding of an unknown case which is extremely difficult and defeats control measures; for, unless and until you find a case you cannot treat it. Secondly, treatment of cases is not so difficult if certain organisational problems are effectively dealt with.

The author has wide experience of tuberculosis problems not only in the developed countries of the West, but also in developing countries and the problems of the latter, therefore, have been given due consideration. He has spent many years in India as a WHO consultant and many of the procedures recommended for developing countries are based, more or less, on the national tuberculosis problem of this country.

On the whole, the book is well produced, well written, in an easy and readable style. It is a useful addition to the books already available on the subject and is safely and strongly recommended for those busy practitioners and 'generalists' who are looking for concise and authentic information on problems of diagnosis and treatment of pulmonary tuberculosis.

S.P.P.

Obituary

SHRI B. M. CARIAPPA

Shri B.M. Cariappa, former Secretary-General, Tuberculosis Association of India, passed away, after a heart attack, on 1 st October 1979 at his daughter's residence in Madras.

Born in Parane Village, Coorg, Karnataka State, Shri Cariappa had his early education at Triuchirapalli which was cut short by



implementation of the National Control in our country. Programme

Shri Cariappa was closely associated with the formation of the Eastern Region of the International Union Against Tuberculosis in 1957 and was its Secretary-General and Honorary Treasurer until 1964. In 1974 he was elected as its Vice-President which post he held until he retired in June last year.

Shri Cariappa was the recipient of numerous awards including the "Commonwealth Award of Honour" by the Chest & Heart Association, London, in 1968 and the "Honorary Diploma" by the TB Association of Italy in 1978. The Tuberculosis Association of India awarded to him its Gold Medal in 1976. The Association cannot be adequately grateful to him for all that he has done for its development and places on record its high appreciation of his outstanding contribution to the anti-TB movement as a whole.

NEWS & NOTES

30TH SEAL CAMPAIGN

The 30th TB Seal Campaign commenced on 2nd October 1979, Gandhi Jayanti Day. Commending the Campaign to the Nation Shri Neelam Sanjeeva Reddy, President of India, in a special message, said:

“The Tuberculosis Association of India and its affiliates in the States will be inaugurating their Thirtieth TB Seal Campaign on the 2nd of October, Gandhi Jayanti Day.

The TB Seal Campaign provides a valuable opportunity to enlighten our people about the seriousness of the tuberculosis problem in our country and to enlist their cooperation in controlling this fell disease. I understand that this campaign, which is being organised in India every year since 1950, has brought in handsome dividends.

I appeal to our people to give generously and make the TB Seal Campaign a success”.

NATIONAL CONFERENCE

The 34th National Conference on TB and Chest Diseases will be held in Jaipur (Rajasthan) from the 28th to 31st October, 1979. The Conference will be inaugurated by Shri Bhairon Singh Shekhawat, Chief Minister of Rajasthan. Balu Sankaran, Director General of Health Services and Chairman TAI will inaugurate its scientific sessions. The programme includes a Symposium on Organisational and Administrative problems (including the place of primary health care programme) in the implementation of the National TB Control Programme, Panel Discussion on Present-day management of Pulmonary TB, Sessions on Chronic Obstructive Pulmonary Diseases, Chemotherapy, Prevalence and Incidence of TB and Chest Diseases in Industries and other aspects of tuberculosis, etc. For further details about the Conference and registration form please contact the Secretary-General, Tuberculosis Association of India, 3-Red Cross Road, New Delhi-110 001.

EASTERN REGION CONFERENCE-COLOMBO

The XIth Eastern Region Conference of the International Union Against Tuberculosis under the joint auspices of (1) the Ceylon National Association for the Prevention of Tuberculosis, (2) the Ministry of Health, Sri Lanka and (3) the Eastern Region of the International Union

Against Tuberculosis, will be held in Colombo (Sri Lanka) from the 14th to 19th October, 1979. The Programme Committee of the Conference has selected some of our senior Tuberculosis Specialists to present papers at the Scientific Sessions of the Conference. About a dozen delegates from India are expected to attend this Conference. Prof. R. Viswanathan, Emeritus Scientist, V.P. Chest Institute, Delhi will be the leader of the Indian delegation. Prof. Viswanathan and Shri P.N. Raman, Secretary General, Tuberculosis Association of India will represent India at the Council Meeting of the Eastern Region of the IUAT.

NEW FILM ON TB

The Tuberculosis Association of India has brought out a new educational film on Tuberculosis with commentary in Hindi. The film was produced by Messrs Karashree of Bombay under the guidance of Shri M.S. Sathyu, the noted film maker. The film is being dubbed in various regional languages according to the needs of the State TB Associations. Those wishing to buy prints of this film may kindly contact the Secretary-General, TB Association of India, 3, Red Cross Road, New Delhi.

SATELLITE SYMPOSIA ON LUNG CANCER AND CHRONIC OBSTRUCTIVE LUNG DISEASE

Under the auspices of the Northern India Chapter of the International Academy of Chest Physicians and Surgeons, Indian Association for Chest Diseases, the Asthma and Bronchitis Foundation of India., the Indian Chapter of Interasma and National Academy of Medical Sciences (India), it is proposed to hold two International Symposia on Lung Cancer and Chronic Obstructive Lung Diseases on November 15-16, 1979 in New Delhi. A large number of international and national experts will be participating in the Symposia. For further details kindly contact Dr. O.P. Jaggi, Secretary, V.P. Chest Institute, University of Delhi, Delhi-7.

FELLOWSHIP FOR STUDIES IN INDIA AND ABROAD

The Bombay Hospital Trust and its Medical Research Centre have set up the Rameshwardas Birla Smarak Kosh (Endowment) to assist, *inter alia*, further studies, Research and specialised training in India and abroad in medical and related fields. The value of Fellowship/Stipend is upto Rs. 1000/- per month if in India and upto

S500/- per month if in foreign country. For further details please write to the Director, R.D. Birla Smarak Kosh, Medical Research Centre, 2nd Floor, Bombay Hospital Avenue, Bombay-400 020.

INDIAN ACADEMY OF PEDIATRICS

The XVI National Conference of the Indian Academy of Pediatrics will be held at the Oberoi Hotel, Calcutta, from the 3rd to the 6th January 1980. A programme of continuity in Pediatric education will be held on the 2nd January for post-graduate and others. For further details contact Dr. G.K. Mehrotra, Organising Secretary, XVI National Conference of the Indian Academy of Pediatrics Institute of Child Health, 11, Dr. Biresh Guha Street, Calcutta-17.

NEWS FROM STATES

Andhra Pradesh

The TB Association of Andhra Pradesh organised a Refresher Course in Tuberculosis on 14th and 15th July 1979 at the Arogyavaram Medical Centre for the benefit of general practitioners. The course was inaugurated by Dr. D. Umapathy Rao, Honorary General Secretary, TB Association of Andhra Pradesh. A large number of general practitioners participated in it.

The State Association has decided to inaugurate the 30th TB Seal Campaign and organise an intensive Refresher Course for Medical Officer of primary health centres and government hospitals and general medical practitioners at government TB Sanatorium, Vikarabad, in the first week of October 1979. The Krishna District TB Association has also decided to organise an intensive Refresher Course on TB & Chest Diseases at Machilipatnam in November, 1979.

The 8th Andhra Pradesh TB & Chest Diseases Workers' Conference will be held at Hyderabad on the 8th and 9th December 1979. The 17th Annual General Meeting of the Association and a Conference of Seal Sale Organisers will also be held at that time. The State Association has selected Dr. S. Sivaram, Director, TB Centre, Trivandrum, to deliver the Dr. P.V. Benjamin Memorial Oration and Dr. O.A. Sarma, Superintendent, TB Hospital, Mangalagiri, for delivering the first Wander T.A. A.P. Oration at the 8th Andhra Pradesh TB & Chest Diseases Workers' Conference.

At the General Body Meeting of the Association held in Hyderabad on 16.7.1979 a Dist. TB Association was formed for the Dist. of

Ranga Raddy. Dr. C. Srinivasa Rao, Retd. Director of State TB Centre, Irramnuma, Hyderabad has been elected as its Honorary Secretary.

Goa, Daman & Diu

The TB Association of Goa, Daman & Diu organised a health education drive which included publication of special articles on Tuberculosis, distribution of pamphlets, leaflets and posters dealing with various aspects of tuberculosis, organisation of public meetings, etc. The functions were held in the Primary Health Centres in Valpoi and Aldona. TB detection camps and BCG Scar Surveys were conducted at Urban Health Centre at Mapuca, and Primary Health Centres at Curchorem. Diu and Ponda.

Maharashtra

The Maharashtra Anti-TB Association in co-operation with the local Lions Club, Zilla Parishad and Indian Medical Association, Bombay branch organised a Multi-Diagnostic Shibir at Primary Health Centre, Navgar, Taluka Vasai, Dist. Thana on 3rd July, 1979. A team of specialists from Bombay examined 370 patients.

Punjab

As part of the TB Control project the Punjab TB Association launched an extensive BCG vaccination campaign in Patiala district under the Chairmanship of Shri Tejinder Singh, Additional Deputy Commissioner, Patiala and attended by the Chief Medical Officer, District Education Officer and Revenue Officers. The vaccinators were provided with Kits, Syringe, etc. and also proforma registers for recording the work. A special pamphlet on BCG vaccination was distributed to all participants.

Tamil Nadu

The Tamil Nadu TB Association has decided to organise a State Conference on TB & Chest Diseases during the last week of November this year. The Association also proposes to have a conference of Secretaries of District TB Associations and a conference of D.T.O.s in the State along with this Conference.

Uttar Pradesh

The 3rd Uttar Pradesh State Conference on TB & Chest Diseases will be held at Nainital in the second week of November 1979. The Scientific Sessions will include papers on (1) Problems of TB in hills in UP (2) chemotherapy and management of resistant cases (3) Childhood TB (4) Extra

Pulmonary TB (5) Non-Tubercular chest diseases and (6) Role of Surgery in chest diseases. For further details please write to the Secretary, UP TB Association, 1-A.P. Sen Road, Lucknow.

The State Association launched an intensive health education drive early this year. The main function at Lucknow was presided over by Shri C.B. Gupta, Chairman UP TB Association and

addressed by Di M.M.S. Siddhu, M.P. and Dr. S.N. Gupta, Special Secretary to Government, Medical and Health Department, Uttar Pradesh. The closing functions were organised at District headquarters at Allahabad, Banda and Hardoi. The Association also participated in the Medical check-up camp organised by the Bharat Sewak Samaj held in Chandrawal Village, Dist. Lucknow.

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ABSTRACTS

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Abst. No. 4

Malignant Pleural Effusions and Their Treatment by Intercostal Talc Pleurodesis.

H.R.S. Barley, Brit. J. Dis. Chest (1979) 73, 173.

1. Cancer of the lung is much more likely to give rise to a malignant pleural effusion in women than in men.
2. Adenocarcinoma of the lungs are more liable to cause malignant pleural effusion.
3. Adenocarcinoma appear to have a tendency to disseminate their cells into pleural effusions whereas squamous cell tumours and sarcomas other than lympho-leukaenic group have little tendency to do so. Oatcell tumours are intermediate.
4. The large majority of malignant pleural effusions in men are caused by cancer of the lung whereas in women the cause may be extra thoracic cancers.
5. Bilateral malignant pleural effusions are more often caused by extra thoracic cancer than by cancer of the lung.
6. Pleural effusion in cancer may or may not be due to directly to the tumour and may or may not be due to malignant changes in the pleura. Non malignant effusion may be caused by obstructive pneumonitis, pulmonary infarction or radiation injury of the lymphatic changes.
7. Malignant effusions may or may not be blood stained and they may or may not contain malignant cells, according to their cause and to the nature of the tumour.
8. Diagnosis can be made in large number of patients by exfoliative cytology and tissue biopsy.
9. Most of the malignant pleural effusions are due to lymphatic obstruction, a few are done to direct or metastatic involvement of pleura and few may be due to superior vena caval obstruction. Pleural metasis may occur without effusion and effusion without metastasis. The presence of

pleural metasis does not exclude mediastinal lymphatic obstruction as the cause of effusion.

10. Before treating pleural effusion in a cancer patient non malignant causes should be corrected.

11. Malignant effusions which reaccumulate rapidly and embarrass respiration should be treated by intercostal talc pleurodesis.

H.B.D.

A Comparison of Oral Choline Theophyllinate and Beclomethasone in Severe Perennial Asthma in Children.

A.T. Edmunds, Sheila Mckenze, Eivor Baillie, Marian Tooley and S. Godfrey, Brit. J. Dis. Chest (1979) 27, 73, 149.

Fourteen children with perennial asthma previously treated with beclomethasone dipropionate were studied from four months in a cross over trial to compare the efficacy of choline theophyllinate and beclomethasone dipropionate aerosol in the control of their symptoms.

Beclomethasone and oral choline theophyllinate together improved control of asthma in comparison with beclomethasone alone. Reduction of the dose of beclomethasone from 400 ug/day caused worsening of asthma. Oral choline theophyllinate did not prevent deterioration and therefore cannot replace beclomethasone for long term treatment of such asthmatic patients.

H.B.D.

Observations on the Management of Acute Bronchial Asthma.

N.J. Cooke, G.K. Crompton and I.W.B. Grant. Brit. J. Dis. Chest (1979) 73, 157.

1. Complications are more with increasing pulse rate and in hospitals, but mortality is low with intensive management.

2. Young patients tend to have high pulse rate and greater risk of complications.

3. Patients with sudden onset of attack carry greater risk.

H.B.D.

Inhaled Corticosteroid Aerosol and Candidiasis

J.N. Sahay, S.S. Chatterjee and T.N. Stcmbridge, Brit. J. Dis. Chest (1979) 73, 164.

A three-month controlled study was done to assess the cumulative incidence of oral *Candida* carriage and thrush in patients having betamethasone valerate aerosol (800 ug/day) for control of asthma. Four of 41 patients on the corticosteroid aerosol developed thrush compared with none of 40 in the control group. The number of cumulative saliva culture positive for *C. albicans* was (approximately 20%) similar in each group oral candidiasis was not clinically important. A simple mouth wash had no prophylactic benefit in aerosol group.

H.B.D.

Ipratropium Bromide Salbutamol and Prednisolone in Bronchial Asthma and Chronic Bronchitis

Lightbody, I.M., Ingram, C.G. Legge, J.S., and Johnston, R.N. Br. J. Dis. Chest (1978) 72, 181.

Eleven patients with bronchial asthma and 10 with chronic bronchitis were treated over four consecutive 3-day periods firstly with aerosole either of ipratropium bromide (40 ug four times a day) or of salbutamol (200 ug four times daily) by random allocation, then the alternate drug, next by both drugs together and finally with prednisolone (10 mgm three times daily) in addition to both drugs.

Ipratropium bromide and salbutamol produced approximately equal improvement in both diseases, with salbutamol showing a marginal advantage in patients with asthma. The combination of both drugs doubled the FEV₁ changes in both groups of patients. The addition of prednisolone to both drugs produced a marginal advantage in asthma patients.

H.B.D.

Bronchial Carcinoma and Long Term Survival

Freise, G., Gahler, E, Leibig. S. (1978). Thorax, 33, 228-234.

Retrospective study of 433 patients who

underwent resection. Of the 471 patients who underwent thoracotomy, the tumour could not be resected in 38 (8%). Sixty three (13.4%) died within the first four weeks. 125 (28.9%) survived more than five years. A high percentage developed either late metastasis, late recurrence, or a primary lung carcinoma or secondaries in lung. The results of surgical resection for bronchial carcinoma cannot be considered satisfactory, although resection remain the best treatment even in those patients with an apparently unfavourable prognosis.

H.B.D.

Use of B.C.G. as an Immunostimulant in the Surgical Treatment of Carcinoma of a Lung—A Five Year Follow-up Report

Edward, F.R. Whitewell, F. (1978) Thorax, 33, 250-252.

Sixty consecutive surviving patients treated with subdermal (5×10^6 organism) have been followed up for five years after resection of lung carcinoma. A control group of the previous 60 consecutive surviving patients not treated with B.C.G. was similarly studied. Administration of B.C.G. neither influenced the survival rate nor has any action upon involved lymph nodes or small metastasis.

H.B.D.

Pulmonary Function Studies in Healthy Pakistani Adults

William, D.E., Drew Giller, K., and Taylor, W.F. (1978) Thorax, 33, 243-249.

Predicted normal spirometric values have been shown to have significant geographical and ethnic variations. These variations are of epidemiological significance undetermining the prevalence of disease and of clinical importance in measuring the effects on pulmonary functions of various diseases.

In 559 men and 94 women forced vital capacity (F.V.C.) was recorded from three satisfactory efforts and F.V.C. one second Torced expiratory volume (F.E.V.₁) and maximal mid-expiratory flow (M.M.F. or F.E.F.) were calculated from the best F.V.C. effort. The F.V.C. and F.E.V.₁ in men were similar in Emigrant Pakistani and North West Indian (Delhi) population but higher than population in South and Eastern India. Pakistani women had values similar to those of women in Northern India F.V.C. and F.E.V.₁ values did not differ between smokers and non-smokers, there was significant

difference in M.M.F. (F.E.F.) in two groups and the later determination is more sensitive test of subtle, asymptomatic changes in pulmonary functions.

H.B.D.

Lung Involvement in Hodgkins Disease

J.B. Macdonald. Thorax, 1977, 32, 644-667.

Lung involvement is commoner than pleural

hilar or mediastinal node involvement. It was observed in 43% of 284 patients. The commonest radiographic type, peribronchial infiltration, tended to occur early in the course of the disease while less common types, homogenous or pneumonic infiltrates and nodules occurred later. Modern chemotherapy is very effective in the treatment of pulmonary Hodgkin's disease.

H.B.D.