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IMPORTANT NOTICE

It is imperative that these quarterly Medical Bulletins are published regularly as they are in great demand by the Medical Profession all round, even outside this State. For this purpose sufficient number of articles of Scientific interest are absolutely necessary. You are requested to kindly co-operate by regularly contributing any articles of scientific interest. Such articles may kindly be sent in duplicate.

CONTENTS

TUBERCULOSIS IN CHILDREN.

Infection in children with the tubercle bacillus takes place through the respiratory tract. In INDIA the human type of tubercle bacillus infects the children, usually from an infectious tuberculous parent. In the Western countries, the bovine tubercle bacillus accounts for a fair percentage of tuberculous infection in children. But in INDIA infections of the children with bovine tubercle bacillus is exception rather than the rule, due to the universal habit of boiling the milk. In some children the source of infection with the tubercle bacillus remains obscure while in some children, the infection is contracted at the school from a tuberculous teacher.

Primary Tuberculosis.—When the tubercle bacillus invades the human body for the first time, the pathological changes that take place in the human body are called primary tuberculosis. The majority of primary tuberculous infections occur in infancy, childhood and adolescence, though no age-group is exempt from primary tuberculous infection. The respiratory tract being the commonest portal of entry of the tubercle bacillus, primary tuberculosis develops in the lungs frequently. The right upper lobe is the site of election for primary tuberculosis though the lesion can develop in any part of the lung. For primary *Tuberculosis* to develop it is essential that the tubercle bacillus lodges in the lung alveolus. Bacilli lodging in the bronchial tract are ejected by cough, by the action of the ciliated epithelium of the bronchial mucous membrane. A single bacillus lodging in the lung alveolus can give rise to primary tuberculosis. At the site of inoculation of the bacillus an alveolitis develops due to multiplication of the bacilli. There is an infiltration of the alveoli with inflammatory exudate and mononuclear cells derived from the blood and the fixed tissue his-tio-cytes. An area of tuberculous broncho-pneumonia develops. The focus that develops in the lung is called the primary focus or the CHON'S focus. The infection spreads from the parenchyma of the lungs, through the lymphatics to the lymphatic glands draining the parenchyma of the lung which is the seat of tuberculous broncho-pneumonia. The pulmonary lymphatic glands, the brencho-pulmonary, the trachea-bronchial glands, and the para-tracheal group of lymph glands show signs of tuberculous inflammation, similar to that occurring in the pulmonary parenchyma. These enlarged tuberculous mediastinal glands are called the "Glandular focus". The parenchymal focus together with the glandular focus is called the "PRIMARY COMPLEX". The parenchymal focus usually develops in the sub-pleural plane, and it is of small size, usually

that of a pea. In practically all the cases of primary tuberculosis, a few bacilli invade the blood stream, in other words there is a mild bacillaemia. This bacillaemia gives rise to post-primary foci in the bones, joints, kidneys, spleen, brain and the lungs and the lymphatic glands, etc. If a very large number of tubercle bacilli invade the blood stream and if there is inherited susceptibility, tuberelous meningitis and miliary tuberculosis develop. The post-primary tuberculosis faci, usually heal without giving rise to any sign and symptoms. Sometimes these post-primary foci, give rise to manifest disease of the affected organ, either immediately, or at a later period due to reactivation caused by lowered resistance of the body, primarily due to malnutrition, or due to debilitating diseases like measles and whooping cough.

Primary tuberculosis, in the vast majority of cases, is a benign process. The caseating parenchymal and the glandular foci heal spontaneously by calcification or by fibrosis. But the healing of the primary tuberculosis foci is a long drawn out process. It takes about two years for primary tuberculosis to heal completely. In some cases complications develop.

Signs, symptoms and diagnosis of primary tuberculosis.

Primary tuberculosis usually does not give rise to manifest illness in the majority of cases. There are no symptoms and no physical signs. In some cases about three to four weeks after the initial infection, there is a low-grade fever lasting about two weeks and in some continental countries like Scandenavia, Erythematous nodules develop just about the time the fever develops in cases of primary tuberculosis. These cutaneous nodules are called erythema nodosum. X-ray of the chest in cases of primary tuberculosis may reveal a soft ill-defined opacity with hazy margin of a small size in the right upper lobe or elsewhere, due to the (GHON'S) focus. The glands in the mediastinum might show enlargement which might be more conspicous than the parenchymal focus, in children. There may be a "DUMB BELL" appearance radiologically due to primary tuberculosis. Radiological abnormalities develop weeks after the initial infection, not immediately and that too, only in a small percentage of tuberculous infection of primary type. Enlarged mediastinal glands may occur in measles, whooping cough, Lymph-Adenoma and lympho-sarcoma, etc., which should be borne in mind in differential diagnosis. When the primary complex heals by calcification about a year or two after the initial infection the radiological abnormalities becomes circumscribed small and dense. Even the calcified foci might disappear.

In the diagnosis, a history of "CONTACT", that is living in close proximity to a tuberculous parent, or other relation, is of paramount importance.

Tuberculin test is *indispensible* for the diagnosis of primary tuberculosis. In practice, the intracutaneous test of *Mantoux* performed with 5 *tuberculin* units, i.e., 0.0001 mgm. of purified protein derivative (PPD) is the best. Old tuberculin 5 T.U., i.e., 0.1 c.c. of 1:2000 solution can also be used. The antigen is injected intradermally into the skin of the forearm. The test is read after 72 hours. The induration and erythema should be at least 8 m.m. and this is taken as a positive reaction.

In the final stages of meningeal and miliary advanced pulmonary tuberculosis and during the attack of measles or whooping cough the tuberculin test is negative, which should be borne in mind.

The gastric contents of a child might reveal tubercule bacilli either in direct smear, or by culture examination, in a small percentage of primary tuberculosis. Bronchoscopy is rarely necessary for the diagnosis of uncomplicated primary tuberculosis. It might reveal distortion and narrowing of the lumen of the bronchi due to pressure of enlarged mediastinal glands.

Primary tuberculosis can develop in the alimentary tract due to ingestion of bovine tubercle bacilli from cow's milk.

Complications of primary tuberculosis.—Complications are prone to develop when primary tuberculosis develops in infants and small children due to low resistance in this age group, and when the child is exposed to massive and repeated infections as from a tuberculosis parent, or a school teacher. Malnutrition, measles, whooping cough predispose to complications in primary tuberculous infectious.

The primary parenchymal focus instead of getting encapsulated, and healing by fibrosis or calcification, might spread by direct contiguity and give rise to spreading broncho-pneumonia and cavitation resembling the type of tuberculosis commonly found amongst the adults. This complication develops in older children. The signs, symptoms and treatment of this condition does not in any way differ from the type of tuberculosis commonly encountered with in the adults.

Tuberculous broncho-pneumonia.—An acute form develops as complication in infants and children due to reasons enumerated above. The clinical picture is identical with broncho-pneumonia due to other causes, viz., rapid onset of high *continued fever*, cough, hurried respirations, etc. Failure to respond to penicillin, should make one think of the possibility of tuberculous

broncho-pneumonia confirmation of the diagnosis can be made by the history of '*contact*', tuberculin test which is positive and by demonstration of tubercle bacilli in the gastric contents. Radiologically there is infiltration of the lung fields, with soft opacities, which is not diagnostic of tuberculosis. Similar X-ray appearance can be found in pneumonocccal broncho-pneumonia; response to steptomycin and Isoniazid is diagnostic.

Sub-acute tuberculous broncho-pneumonia.—Usually there are no symptoms. Such children do not appear to be ill at all. But some children have malaise, failure to gain in weight or slight cough. There is no fever. But on clinical examination, there are pronounced physical signs like dullness and bronchial breathing raees, etc. Radiologically there are confluent opacities in one or both sides which may be lobar in character. Diagnosis of this condition by X-ray chest, tuberculin test, demonstration of tubercle bacilli in the gastric contents. Response to specific chemo-therapy with disappearance of radiological abnormalities, etc., is diagnostic.

Enlarged midiastinal glands.—In primary uncomplicated tuberculosis there are always enlarged lymph glands in the mediastinum draining the parenchymal focus, where there is extensive enlargement of lymph glands on both sides due to lymphatic spread—the condition is called tuberculous mediastinal adenites. There may be no symptoms whatsoever. Sometimes there may be failure to gain in weight, loss of appetite and the child is 'Off colour'. There may be cough resembling whooping cough and *wheezing* similar to Asthma. The syndrome is called "Wheezing and wasting syndrome". There may or may not be low-grade fever. Diagnosis is established by finding tubercule bacilli in the gastric contents. Radiologically there is widening of the mediastinal shadow, and a "*Chimney-stalk*" appearance, due to enlargement of the paratracheals glands. The enlarged lymph glands exert pressure on the trachea giving rise to stertorous breathing and dyspnoea. The bronchi may be pressed upon, giving rise to massive lobar, or segmental atelectasis, even the lobes or segments of the opposite side may be affected. The atelectasis is brought about by bronchial occlusion. The enlarged glands might rupture into the lumen of the bronchus after destroying the wall by erosion and give rise to aspiration broncho-pneumonia. It might be possible bronchoscopically to visualise the distorted and narrowed bronchi. In some cases there may be unilateral emphysema due to check-valve mechanism in the bronchi, allowing the entry of air, but preventing the exit, the lung getting distended and the child becoming severly dyspnoeic

calling for emergency thoracotomy with removal of enlarged glands.

After several months, or one or two years, the enlarged mediastinal glands may shrink and heal and the atelectatic lobe, or segment might re-expand, but in some cases the atelectasis is permanent, pre-disposing to bronchiectasis.

Massive caseating adenitis predisposes to widespread dissemination of the tubercle bacilli by the blood stream, giving rise to miliary and meningeal tuberculosis. Diagnosis of this condition is based on radiological appearances, bronchoscopy, a positive tuberculin test and demonstrating tubercle bacilli in the gastric contents and a history of 'Contact'.

Miliary tuberculosis.—There is always a mild bacillæmia in all cases of uncomplicated primary tuberculosis, giving rise to disseminated foci, which heal without giving rise to manifest disease. When there is massive invasion of the blood stream, due to reasons given above, miliary tuberculosis develops. The bacilli enter the blood stream, through the thoracic duct, etc. Tubercles develop in the sub-intimal layer of the blood vessels, which caseate and rupture directly into the blood stream, giving rise to widespread, and fulminant heamotogenous dissemination. Milder dissemination occur where the number of bacilli is smaller and where there is acquired resistances, giving rise to chronic miliary tuberculosis or isolated foci in a few organs.

Acute Miliary Tuberculosis is characterised by the formation of small foci of uniform size and density, which essentially show caseation in practically all the organs of the body, the lungs, spleen, brain, bones, joints kidneys, lymph glands, the serous membranes, etc.

Acute generalised miliary tuberculosis usually develops, as a complication of primary tuberculosis within six months of the initial infection. It can also occur at a later age due to surgical operations on tuberculous joints, following measles, or whooping cough. Curretting of a tuberculous endometrium, etc., clinically there is a continued fever similar to enteric fever for which it is easily mistaken. There may or may not be cough. In the final stages, there is cyanosis, dyspnœa, etc. The spleen may be palpable. Fundus examination with the ophthalmoscope, may reveal tubercles in the choroid. Sternal marrow examination by the microscope is helpful in some cases.

Examination of the chest may reveal no abnormality. X-ray chest shows characteristic bilateral and symmetrical tiny opacities involving more particularly the upper zones.

X-ray chest may show the "*snow-storm*" appearance or "*pin-point*" stippling. Rarely the X-ray chest reveals no abnormality. In about 30 per cent of cases of miliary tuberculosis there is tuberculous meningitis also. Acute miliary tuberculosis is fatal unless adequately treated, with specific drugs.

Chronic Miliary Tuberculosis.—This occurs i n older age-groups, usually adults. It is asymptomatic and without physical signs, in most cases. It is discovered accidentally by X-ray, which shows coarser, more discrete, less numerous bilateral nodular opacities, which have a tendency to heal by calcification. This condition may regress spontaneously without treatment.

Tuberculous Meningitis.—A detailed description is beyond the scope of this article. The classical signs may be lacking in infants and children. Usually of insidious onset, with alteration in personality of the child, irritability, low continued fever, convulsions, headache, vomiting, stiffness of the neck, strabismus, irregular pupils. Hydro-ceaphalic cry, and a bulging anterior fontanelle in infants below eighteen months. C S.F. pressure is increased, clear, the sugar and chlorides are diminished the protein and cells are increased. Tubercle bacilli may be found in the cob-web coagulum found on standing the C.S.F. in a test tube overnight by direct smear or culture. Rarely the C.S.F. shows no abnormality. Tuberculous meningitis occurs at all ages, but more common in children below three years. It is a fatal conditions, unless given adequate and prolonged specific chemo-therapy. The mortality rate depends at the stage at which treatment is commenced. If treatment is commenced at the commencement of the disease, the recovery rate is high. If treatment is commenced late, in the course of the disease, the mortality rate is high.

Tuberculous pleurisy.—There is involvement of the pleura overlying the parenchmal focus in all cases of uncomplicated primary tuberculosis, which heals giving rise to a localised pleural adhesion, or thickening. Sero-fibrinous pleurisy develops as a complication of primary tuberculosis, either immediately or after some months. Practically all cases of idiopathic pleurisy with effusion are tuberculous. Effusions developing insidiously may not give rise to symptoms, but in effusions developing rapidly there may be dyspnœa, continued fever, cough, etc. Physical examination may reveal the classical signs, viz., the mediastinal shift to the opposite side limited movement of one hemi-thorax, absent, or markedly deminished vocal fermitus. "Wooden" dullness on percussion, absent, weak, or distant Bronchial breathing. Adventitious sounds, with the exception

of a pleural friction rub are conspicuous by their absence. Excepting the "Wooden" dullness, most of the other physical signs may be absent.

X-ray chest in free effusion reveals, a homogenous opacity in the lower part of chest or the entire lung field, obliterating the costo-phrenic angle. The upper border of the opacity is curved, with a concavity upwards in small free effusions. The trachea and mediastinum may or may not be shifted to the opposite side.

The pleural fluid is clear with a high specific gravity and protein content, with a tendency to coagulate spontaneously. The cells are mostly lymphocytes. The pleural fluid on culture and guinea pig inoculation may reveal tubercle bacilli. Rarely bacilli can be seen in the direct smear.

Tuberculous pleurisy with effusion responds well to treatment with S.M., isonlozid and corticosteroids. Whenever there is dyspnœa aspiration is indicated.

Epituberculosis.—This term is condemned by many *phthisiologists.* It is a resolving non-caseating tuberculous pneumonia, giving rise to signs of consolidation in the upper part of the chest like dullness, and bronchial breathing. Radiologically it gives to a fan-shaped opacity spreading from the hilum to the periphery of the lung fields. The condition clears up completely radiologically after some time. It is due to atelectasis of a lobe caused by the pressure of an enlarged tuberculous gland in the bronchus, or a perifocal pneumonia.

Prognosis.—Tuberculous infections occurring in infants below one year have a grave prognosis, with a mortality of about 60 per cent miliary and meningeal tuberculosis occur frequently in children who develop primary tuberculosis below the age of three years—children above the age of four years contracting tuberculosis have a mortality of about 20 per cent. Malnutrition, overcrowding, repeated exposures due to "Contact" with a tuberculous parent very adversely affect the prognosis. Early institution of adequate and prolonged chemotherapy favourably influences the prognosis.

A CASE OF LEPROSY WITH VISCERAL INVOLVEMENT.

A 44-year old policeman in Madras City was admitted in Cardew Ward, Stanley Hospital, on the 16th of October 1959, complaining of marked loss of appetite since two months. A month prior to admission he had irregular fever for ten days and during the pyrexial period developed swellings of the epitrochlear, inguinal, cervical, and axillary group of lymph glands in that order. The size of glands gradually increased but the enlargement was painless. In addition, at times patient noticed oozing of blood from the eyebrows on scratching. The oozing persisted for a few minutes and arrested without any treatment. There was no history of epistaxis, or jaundice nor did he have any subjective sensory disturbances. Bowels were regular.

He was a fairly well-nourished, afebile, ambulant adult slightly anaemic but not jaundiced. Palms and soles showed generalised dark pigmentation. Shiny patches with indefinite margins and corrugated skin could be seen over the front of scalp, anterior and lateral aspects of both legs and forearms. The patches were macular and erythematous at places with indefinite margins. The ulnar and lateral popliteal nerves were moderately thickened. Tactile sensation was slightly diminished and sensation of pain and temperature appreciably impaired over the areas described. There was no obvious wasting of muscles.

The cervical, axillary, epitrochlear and inguinal lymph glands were markedly enlarged on both sides. The glands were firm in consistency, discrete, non-tender and were freely movable. In the abdomen there was some amount of free fluid; liver was palpable up to two inches below costal margin, tender with an even surface and a smooth edge. Spleen was also tender, palpable even near the umbilicus. Scrotal skin was irregularly thickened and hairless, adherent to testes on the right side. Epididymis was slightly thickened o n both sides B.P. 92|62.

The following investigations were done:—

Blood—

Total leucocyte count was 10,000|c.m.

Differential count was normal.

R.B.C. count was 3.8 ml|c.m. and HB 9 gms.

Blood smear: No immature cell seen.

Prothrombin time was 30 sec.

Bleeding time 1 min. 30 sec.

Coagulation time 8 min.

VDRL—Negative.

Gel and chopra tests—Negative.

Paul Bunnel test—Negative.

In the sternal marrow smear there was slight preponderance of leucopoietic tissue, with normoblastic reaction. There were no L.D. bodies. A supratrochlear gland was excised and sent for biopsy. It showed collections of fat-ladden RE cells with lepra bacilli. A liver biopsy was done and the report read as follows: " A few parenchymal cells show fatty change. The kupffer cells are prominent. Special staining methods showed considerable number of lepra bacilli in the liver and lymph node. Patient was given D.D.S. This precipitated an acute reaction in the patient with fever, toxemia and pains all over the body. He developed painful, subcutaneous nodular swellings all over the body especially over the legs and thighs. The lymph glands, liver and spleen became accutely tender. The reaction was thought to be one of the episods of Erythema Nodosum leprosum, and corticosteroids with antibiotics were administered. The fever subsided, nodules disappeared and the patient felt relieved. At the time of discharge on 9th December 1959 the lymph glands were not palpable, but the liver and spleen were still enlarged.

The patient had been attending the Leprosy OP for 3 months and was readmitted with fever for one week, enlarged tender inguinal glands and a few firm, discrete, slightly tender subcutaneous nodules over the right forearm and right shoulder. Liver and spleen were still palpable. The reaction was treated with cortisone, calcium and achromycein when the general condition improved he was discharged and advised to continue treatment for leprosy.

Discussion.—This case of Lepromatous Leprosy with obvious visceral involvement for the reason that clinical involvement with enlargement of the lymph glands and internal organs like liver and spleen is very unusual, though in a case of lepromatous leprosy liver biopsy may often show leprabacilli.

There is no definite statistics available as to the relative involvement of various viscera in lepromatous leprosy. Pittsbury mentions that lymph nodes are affected in varying degree. Lungs are rarely affected. Nodes of hilum of liver are at times enlarged. Bacilli can often be found in the kupffer cells and

interlobular connective tissue with occasional small lepromatous foci in the portal spaces. The entire spleen may team with leprabacilli in some cases. Kidney may be involved in the form of amyloidosis or glomerulonephrites. The tuberculoid type of leprosy does not involve the viscera.

Manson Bhar believes that leprous affection of lymphatic glands is often histologically obvious. The inguinal glands are it appears, is in reality a disease of skin, certain mucous especially affected. Lepromatous leprosy, a systemic discase membranes peripheral nerves and testes. Internal organs are affected only incidentally and only so long as these principal centres harbour the disease. The liver, spleen, lymph glands and possibly the bone marrow act as filters and destroyer of infection fork, while the liver may harbour millions of organism, not even a few proliferate to form the miliary lepromata. Apart from septic complications, lymph glands are not enlarged to any significant extent as in T.B. Caseation and liquefaction do not occur in leprosy.

Sharma and Srivastava, reviewing 155 patients, found that in 95 of them enlargement of lymph nodes were present. The epitrochlear group was involved in 62 per cent, inguinal 45 per cent, cervical 33 per cent, and axillary 20 per cent, in 40 of these cases biopsy was done and 24 were found to be lepromatous. They concluded that,

Visceral involvement is considered to be of bad prognostic value. Where the patient cannot tolerate adequate treatment with sulphones and the sedimentation rate is above 10 prognosis is considered generally poor (Hamelton Fairley) for ESR is a valuable index of patient's resistance. Nodular leprosy is particularly prone to complication like pulmonary tuberculosis, renal disease and secondary amyloidosis. The lepra reactions or the occurrence of liver and erythema nodosum are said to be more common among Indians and were once considered to be due to sudden destruction of lepra bacilli and release of toxins similar to herxheimer reaction or due to sudden bacillemia itself but now it is believed to be a form of allergic reaction. In lepra reaction the disease gets exacerbated with fever accompanied by a rapid proliferations of organisms. In erthyema nodosum leprosum with fever and erythematous nodules the number of bacilli are not increased. Indeed it may be diminished. It is considered that though it is distressing, is not of bad prognostic significance. On the contrary it may be the reverse. Nevertheless contradictory opinions prevail as to the significance of such reactions. Pittsbury considers this as a

favourable sign in respect of probable improvement of lepramatous infiltration and feels that this reaction represents an attempt to destroy the causative bacilli.

Treatment of this case.—Sulphones are found to be of definite benefit in severe lepromatous lesions which are least responsive to treatment with chaulmoogra preparations. One of the most serious side effects is anaemia in addition to the lepra reaction already described. The severe anaemia in this case may partly be due to the disease itself and partly due to the sulphone treatment he had during the past three months. The anaemia may be treated with iron therapy orally or parentarally. In severe cases transfusion can be given. Sulphones are reintroduced slowly and carefully. Tendency to chronic and subacute allergic reactions progressively lessens. Mild intolerance to sulphones is treated with prolonged administration of corticosteroids in small dosage. While in severe cases of complete intolerance to D.D.S., Cochrane advises treatment with streptomycein, I.N-H, and sulphetrone.

Treatment is to be continued till there is complete clinical improvement with bacilli are not demonstrable definitely in the lesions. Leprosy being notoriously a disease of remissions, it may be difficult to judge the results from the clinical findings or even bacteriological results.

Summary.—Involvement of viscera is an uncommon clinical finding in leprosy. Viscera are involved in lepromatous leprosy. A case with hepatosplenomegaly and lymph glandular enlargement in lepromatous leprosy is reviewed. Liver biopsy showed leprabacilli. The patient suffered an attack of erythema nodosum and was successfully treated with sterioids, liver and lymph gland enlargement in leprosy are discussed.

A CASE OF STAPHYLOCOCCAL SEPTICEMIA.

Arogyaswami, aged 16 years, was admitted on 20th September 1960, with a history of intermittent fever and epigastric pain of twelve days.

Examination.—Patient was toxic. Temp.: 102 F. Pulse: 126 per minute. Tongue coated and dry. B.P. 100|70. Resp.: 38 p.m. A warm tender flutuating swelling 4 inches in diameter was seen over the upper lateral aspect of left thigh.

Abdomen.—Liver enlarged 3 inches below the costal margin in the right mid-clavicular line. The left lobe was also enlarged and showed fluctuating areas. Spleen enlarged 1 inch below the costal margin. Other systems normal.

Investigations.—(i) WBC—Total count 17,000; D.C. P.M. 80 per cent, E: 16 per cent, M: 2 per cent, E: 2 per cent; (ii) X-Ray chest: Right dome of diaphragm raised.

Treatment and progress.—On 21st September 1960 the abscess in the left thigh was incised and pus was sent for culture. Patient was given penicillin 10 lakhs, 6th hourly and sulphadiazine 1 G. thrice daily and I.V. Peristonium. Aspiration of liver abscess was performed on 22nd September 1960 and yellowish white pus was sent for culture. Penicillin 50,000 units was instilled in the abscess cavity. Staphylococcus aureus (coagulase positive) was grown in culture. Blood culture also showed staph. aureus. Patient was given achromycin 250 mg., 6th hourly. Temperature continued to be intermittent. On 28th September 1960 while on antibiotics, he developed signs of consolidation in the right lower lobe of the lung. Next day he expectorated frank pus, from which staphylococcus aureus was isolated. Patient was started on ilotysin 200 mg., 6th hourly. On 30th September, extensive pleural rub was heard on the right side of chest and signs of effusion were present two days later. Straw coloured fluid was aspirated on 2nd November 1960 and the culture was sterile. Temperature was still intermittent and a combination of achromycin 250 mg., 6th hourly and penicillin 100 lakhs 6th hourly was started. On 13th October 1960 patient complained of pain in the chest and pericardial rub was heard. The extensive pericardial rub persisted for a week. Paracentesis pericardii did not reveal fluid. E.C.G. did not show features of pericarditis.

Liver was just palpable and signs of loculated pleural effusion were present posteriorly on the right side. Aspiration in

his space resulted in withdrawal of pus, which on culture showed staphy aureus. Temperature was normal subsequently. Achromycin 250 mg. t.d.s. was continued. Patient complained of intestinal colicky pain on 14th November 1960. The temperature was 102 degrees F. and he passed melaena. Coagulants, stimulants, fluids, blood were given and patient had normal motion on 18th November 1960. He was on Achromycin and penicillin till 24th November 1960. Patient continues to maintain normal health.

SUMMARY.

A case of stapylococcal pyaemia, involving various sites as kin, liver, lung, pericardium, intestine has been presented. The advent of antibiotics, especially a combination has cured the case, which is in the pre-antibiotic era was almost fatal.

A CASE OF ADDISON'S DISEASE FROM DR. M. VISWANATHAN'S UNIT.

Kathavarayan, aged 35 years, was admitted on 9th October 1960 for acute gastroenteritis with peripheral circulatory failure evidenced by a rapid thready pulse, cold and calmy extremities and low blood pressure. The severe astheia and marked hyperpigmentation of skin both in the exposed as well as the unexposed parts and the mucosa of the oropharynx helped us to focus on the supra renals for the cause of gastroenteritis and peripheral failure and accordingly biochemical investigations were carried out.

Routine.—B.P. 100|70.

Urine: Alb.—No. T.C. 8600 D.C. P64 L30 E6. Sugar—No.

E.S.R. 1 hr. 23 min.

R.B.C. 3.5 million.

Hb. 70 per cent.

Special—

(1) Robinson Kepler's Test:

The first part is based upon the principle of decreased capacity to excrete water by the kidneys in Addison's Disease.

The patient's night volume urine was collected and measured (30 oz.) in the morning, he was hydrated with 20 cc of water|kilogm body weight. The hourly specimens of urine passed were collected and measured. In no case the hourly specimen exceeded the night volume. Actual figures being 4, 3½, 4 and 3, respectively. This decreased capacity is due to increased activity of the antidiuretic hormone of pituitary, due to supra renal failure.

The second part of the test is based upon this decreased capacity to excrete water along with the increased chloride output in urine with urea retention which is calculated by the formula.

$$\frac{\text{Urine urea}}{\text{Blood urea}} \times \frac{\text{Plasma chloride}}{\text{Urine chloride}} \times \frac{\text{Day volume urine.}}{\text{Night volume urine.}}$$

This figure if it is below 25 is taken as positive for Addison's Disease. In this case it was worked out to be 5.

(2) The 17 ketosteroid excretion for 24 hours was 4 mg. as against a normal of 25 mg. for male.

(3) *Thorn's Test.*—It is based upon the fact that with the administration of ACTH, the supra renal cortical tissue which gets stimulated brings about a reduction by more than 50 per cent in the circulatory eosinophils. This was positive in this case due to the fact that there was no significant fall of circulatory eosinophils thereby confirming the fact that there was no supra renal tissue to be stimulated.

The ketosteroid estimation was also done after administering ACTH 20 units 6th hourly and it was that ketosteroid elimination by the kidney was very low, 5 mg.|24 hours, thus confirming the absence of adrenal cortical tissue.

(4) Plain X-Ray abdomen—No supra renal calcification.

(5) X-Ray chest P.A. View—Calcified spots in both right and left upper lobe of lung fields.

With these bio-chemical findings diagnosis of supra renal cortical failure was made. Etiology is presumed as tuberculosis as there is no evidence for other causes as malignancy, atrophy, degeneration.

Management.—During the stage of crisis, the water loss, chloride loss were made good and the patient was treated with parenteral hydrocortisone.

At present he is on substitution therapy with cortisone acetate 25 mg. desoxycorticosterone acetate and a high salt diet.

Streptomycin 1 gm. daily and INH 300 mg. daily is being given an antituberculous treatment.

Summary.—A case of Addison's Disease with the classical features of weakness, wasting, anorexia, marked pigmentation and hypotension, admitted and treated for adrenal crisis has been described. The bio-chemical investigations described in detail confirmed the diagnosis.

ANTHRAX.

Name: D. Dakshinamoorthy, Age: 60 years.

c|o Shanmugam, Hindu, male.

37, Perianna Mudali street, G.T., Madras-1. Date of admission.—14th October 1959.

Diagnosis.—Anthrax.

NOTES.

Complaint and duration.—Swelling on the forearm (right) 4 days duration.

History.—After 3 days of fever, patient found a small nodular papule on the anterior aspect of the right forearm on 11th October 1959 which was rapidly increasing in size. He came with this swelling to Madras and a doctor gave him 5 L. penicillin. The lesion was increasing in size, oozing out sero-sanginous fluid. He has been further given some more penicillin. Finding no improvement he was referred to Stanley Hospital.

On enquiry, the patient gave a history of unusual mortality of cattle and sheep in the recent months in the village (Peddapattu), Chengam taluk, North Arcot district. The patient who is an agriculturist has his own sheep farm. He himself lost a few sheep in the recent disaster and found one of his sheep sick, slaughtered and ate it. At the time of slaughter, he had an open ulcer in the right forearm in the healing stage. Few days later, the patient developed a small pustule which later grew into a small abscess with all signs of inflammation.

History of gastro-enteritis or pulmonary involvement.

14th October 1959:

Terramycin 100 mg. 1 M. thrice a day.

15th October 1959, 10 a.m.:

Terramycin 100 m. 1 M. thrice a day.

2 p.m.:

Penicillin (PAM) 6 L.; crystolline penicillin 5 L.

8 p.m.:

Terramycin 100 mg. 1 M.

16th October 1959: Terramycin 100 mg. IM.

17th October 1959: Terramycin 100 mg.

18th October 1959: Terramycin 100 mg.

19th October 1959: Terramycin 100 mg.

20th October 1959: Terramycin 100 mg.

In addition, we had two more cases of anthrax, diagnosed bacteriologically proved, and treated as above. Both improved and discharged.

PROCEEDINGS OF THE CLINICAL SOCIETY, GOVERNMENT TUBERCULOSIS SANATORIUM, TAMBARAM, FOR 1960.

Case I.

A case of cavity in the apico-posterior segment of the left upper lobe treated by resection.

Miss C., aged 43 years, school teacher, was admitted on 5th August 1958.

Complaint.—Cough with expectoration and fever for 6 months prior to admission. The present illness started with symptoms of diarrhoea and cough. Investigations were done at Stanley Hospital, and a diagnosis of pulmonary tuberculosis, with tubercular enteritis was made. Then the patient was admitted in the Sanatorium.

Investigations—

Sputum:

Smear—Negative for A.F.B.

Culture—Negative for A.F.B.

Skiagram of chest showed a spherical cavity of about 3 c.m. in diameter, with a thin wall, occupying the left apex with dense adhesions from the cavity wall; a few nodules in the upper lobe; and a shadow by the side of the aortic area, due to mass of glands by the side of the left upper lobe bronchus. Tomogram showed a thin walled cavity at 6 c.m. level. The upper lobe bronchus was clearly seen and the apicoposterior segmental bronchus could be traced to the cavity. Bronchoscopy revealed a normal tracheobronchial tree. Treatment given:

Streptomycin 30 grams.

P.A.S. 4710 grams.

I.N.H. 965 grams.

Skiagram of the chest after chemotherapy showed clearing up of the inflammation all round the cavity, but the cavity remained unaltered.

After adequate preparation, with morphia and atropine as premedication, under endotracheal gas and oxygen, anaesthesia, resection on the apicoposterior segment of the left upper lobe was done on 26th April 1960.

The thin-walled cavity occupied the left apex and was densly adherent to the apex. It was mobilised by sharp dissection. There were few glands near the origin of the left lobe bronchus. The cavity balooned every time the lung was inflated. The cavity, with few nodules around, was confined to the apicoposterior segment. The rest of the lung was free of palpable disease.

Post-operative period was uneventful. X-ray taken during the post-operative period (after two months) showed complete expansion of the left lung and the right lung did not show any change.

DISCUSSION.

Q.—Whether the shadow by the side of the aortic area seen in the skiagram were glands?

A.—Yes. They were glands, three in number, pressing on the left upper lobe bronchus near the taking off, of the apicoposterior bronchus, as confirmed on the operation table.

Q. 2.—What did the gland show on section?

A.—It did not show any caseous mass but the cut surface was uniformly dark—anthrocotic pigment.

Q. 3.—What did the resected specimen show?

A.—The specimen showed a thin-walled cavity, 3 c.m. across with a communicating bronchus.

Q. 4.—Whether one can be sure of the diagnosis?

A.—The onset of the disease with fever, cough and enteritis were suggestive. A solitary report on the sputum by direct smear was positive. The location of the cavity with nodules around points to a tuberculous origin of the cavity. There were also three caseous nodules in the posterior segment. The smear from the cavity wall and from the nodules were negative for A.F.B.

Q. 5.—Why was A.P. not tried in this case?

A.—A.P. is contraindicated in a case of this type with a tension cavity.

Q. 6.—Whether resection was justified in this case?

A.—This patient could have been managed with chemotherapy alone, and resection performed when the patient had

complications like secondary haemoptysis, etc. The patient might have to be under constant medical supervision.

In view of the size of the cavity, the possible dangers from reactivation, haemoptysis, or secondary infection we decided to operate on this patient.

A CASE OF RENAL TUBERCULOSIS.

Miss M., aged 24 years, was admitted in the Sanatorium on 15th April 1959.

Complaint.—Pain in the left loin since two years. Frequent painful micturition since six months. Heamaturia frequent at irregular intervals.

Past history.—Cough with expectoration and irregular fever. The present complaint started about two years ago with fever and pain in the left lumbar region, left hypochondrium, and left loin. She said she was unable to lie on the left side due to pain. An year and a half later she developed frequency of micturition with pain in the suprapubic region during micturition. Heamaturia developed later and occurred at frequent intervals.

Examination of the patient.—Emaciation, anaemia, palpable inguinal glands, and clubbing of fingers were present. Movement of the anterior abdominal wall during respiration, size and shape of the abdomen revealed no abnormality. Tenderness was present on the left lumbar region on deep, palpation but there was no palpable mass.

Respiratory system.—Diminished air entry over both the apices was present.

Investigations—

Sputum.—Smear examination negative for A.F.B.
E.S.R. 115 mm. per hour.

Blood.—Hb. R.B.C.
D.C. P E L M.

Urine.—Reaction albumin.
Microscopic examination: Pus cells seen.
Culture: Diphtheroids grown.

Skiagram chest.—Right upper zone emphysematous.

I.V. Pyelography.—No excretion of the dye was seen on the left side. Compensatory hydronephrosis on the right side with thimble bladder were seen.

Treatment.—Patient received streptomycin 120 grams, I.N.H. 2640 Milligrams.

Patient showed signs of improvement in general condition, absence of fever, etc.

As the I.V.P. showed no excretion of the dye on the left side nephrectomy was advised and was operated.

Post-operative period was uneventful and the patient recovered completely.

A CASE OF BRONCHIECTASIS OF TUBERCULAR AETIOLOGY.

Patient Mustafa, aged 17 years, was admitted in the Sanatorium on 8th June 1959.

Complaint.—Cough with expectoration, fever, loss of weight, and increasing general weakness, since two years. Also had exertional dyspnœa.

Gives history of having had treatment at Coimbatore with 30 grams of streptomycin and I.N.H. 180 tablets.

On examination.—Clubbing of fingers, wasting of muscles of the anterior chest wall on the right side, general emaciation, diminished respiratory excursion, deviation of the trachea to the right, were present.

Percussion revealed impaired resonance over the right lower lobe. Auscultation showed bronchovesicular breath sounds over the right infraspinous, subscapular regions. Sibilant rales were heard over both sides of chest, more over the right lower lobe. Cardiovascular and central nervous systems revealed no abnormality.

Investigations—

Sputum.—Smear for A.F.B. negative. Positive on 24th August 1959.

Culture: Strepto and staphylo cocci were grown.

Quantity: Six ounces for 24 hours.

Blood.—Hb. 75 per cent R.B.C. 3 millions.

W.B.C. 7600.

D.C. P60, L32, E7, M1.

Weight of patient.—95 lb.

Skiagram of chest taken on 15th June 1959, showed circular areas of increased density near the right hilar region and base.

On the left side streaks and striations of opacity on the hilar region down to the base were seen.

Bronchogram.—Done on 24th July 1959 showed bronchectatic changes of right middle and lower lobe bronchi.

Treatment.—Since the sputum was negative for A.F.B. and culture showed growth of strepto and staphylo cocci he was put on penicillin in 5 lakhs b.d., prednisolone 5 mg. t.i.d. for a few days, followed by ambramycin capsules (250 mg. each) and elkosin tablets (0.5 gm. each) for a sufficient period. Since there was no appreciable improvement in the condition of the patient, and taking into consideration history and results of clinical examination, patient was put on streptomycin, I.N.H. and vitamins. Condition of the patient improved. The search for A.F.B. was continued and on 24th August 1959 examination of the sputum was positive for A.F.B.

Patient's weight increased to 101 lb.

Conclusion.—Since the acquired bronchiectasis revealed by the bronchogram is not a disease *per se*, the primary condition in this patient must be pulmonary tuberculosis.

A CASE OF RECURRENT SPONTANEOUS PNEUMOTHORAX.

Patient Subbammal, aged 2½ years, was admitted in the Sanatorium on 27th February 1960.

Complaint.—Cough with expectoration and irregular fever since six months.

History of having been treated in Military Hospitals with streptomycin. The child is said to have suffered from whooping cough.

Examination of the patient.—Looked anaemic, breathless, emaciated and abdomen prominent. Free fluid in the abdomen present. Spleen and liver were not palpable.

Cardiovascular system revealed no abnormality.

Respiratory system.—Hurried respiratory movements, with impaired resonance all over on either side, with diminished air entry over the left base. Rales were heard all over.

Investigations.—Sputum smear negative for A.F.B.

E.S.R. 22 mm. per hour.

Blood.—Hb. 50 per cent. R.B.C. 2.4 millions. W.B.C. 7600 (D.C.P. 57, E6, L36).

P.P.P. done on 7th March 1960. Negative.

P.P.D. done on 28th May 1960. Positive.

Weight of patient 22 lb.

Skiagram chest suggested bilaterial tuberculosis lesion.

Treatment.—Anti-anæmia treatment, vitamins, streptomycin, PAS and INH were started.

Patient showed improvement. Weight increased to 30 lb.

Temperature became normal.

On 13th June 1960 the child became suddenly dyspneic. A bed X-ray was taken and the wet film showed spontaneous pneumothorax on the left side.

Deflation was done and 1500 cc of air was removed from the left side. Oxygen inhalation was given, with intermittent aspirations. Child recovered.

Again on 19th September 1960, the child developed again spontaneous pneumothorax, and 600 cc of air was removed. The child recovered.

On 29th September 1960 the child had developed spontaneous pneumothorax for the third time. Intercostal drainage under a water seal was put. After 48 hours the left lung was found to be fully expanded and the drainage was removed.

A skiagram taken showed multiple cysts in both lungs.

Discussion.

Q. 1.—Is this a case of pulmonary tuberculosis giving rise to recurrent spontaneous pneumothorax? If so, why was it that tuberculous emphyema did not develop?

A.—Clinical signs and symptoms, and radiology suggested a diagnosis of pulmonary tuberculosis. Emphyema was probably prevented by the anti-tuberculous drugs cover.

Q. 2.—Could it be a case of cystic disease of the lungs or bronchiolectasis?

A.—Radiology does not suggest bronchiolectasis but the picture taken later showed multiple cystieg spaees in the lung.

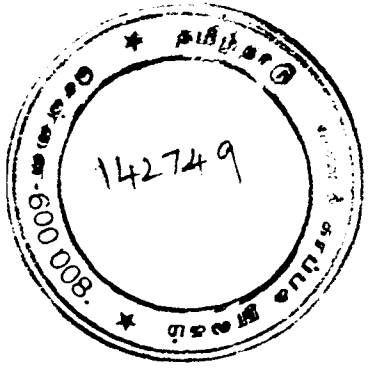
Q. 3.—Is surgery indicated in this case?

A.—No. The cysts are multiple and bilateral. Recurrent occurrence of pneumothorax could be prevented by encouraging adhesion of visceral and pariatal pleura by means of sclerosing agents.

Q. 4.—What is the prognosis in this case?

A.—It is very grave as the patient has developed recurrent spontaneous pneumothorax and the general condition is very poor.

Acquired cysts in the lung are generally post-infectius, the primary infection is usually a staphylococcal pneumonia. Interest has recently centred in the 'bullous' cavity or cystic cavity or end stage cavity followed the use of isoniazid in pulmonary tuberculosis. This is seen in about 10 to 20 per cent of cavities treated effectively.



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