

## COEXISTENCE OF LYMPHADENOMA AND TUBERCULOSIS.

BY

T. KRISHNA MENON, M.B., C.M.,  
HONORARY PHYSICIAN, GENERAL HOSPITAL, MADRAS;

AND

T. BHASKAR MENON, M.B., M.R.C.P.  
(Department of Pathology, Medical College, Madras.)

(With Special Plate.)

THE possibility of lymphadenoma and tuberculosis coexisting is well recognized. Ewing has pointed out that tuberculosis follows in the wake of lymphadenoma; and Ziegler estimated that 20 per cent. of these patients died of miliary tuberculosis. It has, indeed, been held that the two diseases are identical. Sternberg first put forward the view that Hodgkin's disease was a form of tuberculosis, due, probably, to infection with an attenuated form of the tubercle bacillus. Clinically, cases occur in which the two diseases are almost indistinguishable. In the morbid histology of tuberculous adenitis a type is recognized without definite giant cell systems and in which endothelial hyperplasia is the characteristic feature. It is well known that an endothelial hyperplasia may be the sole feature of early lymphadenoma, a point which is emphasized by Christian and Warthin. Even Andrewes, though he denies the identity of the two diseases, considers it is difficult to distinguish atypical lymphadenoma from a typical tuberculosis.

We do not intend to lay any great stress on this view, but the occurrence of tuberculosis in cases of Hodgkin's disease is of great interest, since both may present lesions which are difficult to allocate. This is particularly so since there occur in lymphadenomatous glands in the late stages certain yellowish-white areas which are referred to by Ewing as "necrotic foci" and by Longcope as "areas of necrosis." The difficulty arises in distinguishing them from areas of caseation, and the question may be asked

whether these necrotic foci are really due to a mixed infection with the tubercle bacillus, or whether there is in lymphadenoma a type of "caseation" similar in certain aspects but differing in others. This latter view is of importance since, if this is so, the analogy with other infective granulomata like syphilis, tubercle, and actinomycosis becomes more striking, for in all these conditions we have alteration of the normal structures by endothelial and lymphoid tissue with the presence of giant cells, followed later on by "caseation" and by sclerosis.

The following case of coexistent Hodgkin's disease and tuberculosis is of interest since these necrotic foci were present in the glands.

A man, aged 30, was admitted under the care of one of us in the General Hospital, Madras, on August 28th, 1927, with a history of enlargement of the glands in the neck, pectoral region, and axilla of five months' duration. He was sent to the surgical ward for excision of a gland for pathological report. The report was "lymphadenoma," and the patient was subsequently re-admitted to the medical wards on September 11th. He was then complaining of difficulty in swallowing, possibly from pressure of the glands in the neck. The glands in the parotid region were also enlarged. There was, however, no dyspnoea, loss of voice, or enlargement of the superficial veins, or any feature suggestive of a mediastinal growth. The chest was, however, asymmetrical and there was slight fullness in the second and third interspaces on the left side. From the first a dullness on percussion of both sides of the chest was noticed and on auscultation a few rales could be heard. The heart showed a slight softening of the first sound in the aortic and pulmonary areas. Throughout his stay in hospital there was a remittent pyrexia, with, however, no periods of apyrexia. Puncture fluid from one of the glands showed marked eosinophilia. The Wassermann reaction was negative.

The blood on admission showed 1,720,000 red blood cells per c.m.m., 58,000 white corpuscles, haemoglobin 56 per cent. The differential count revealed polymorphonuclears 88 per cent.; lymphocytes 8 per cent., myelocytes 3 per cent., eosinophils 1 per cent. The second examination of the blood showed 3,748,000 red blood cells, 56,250 white corpuscles; with polymorphonuclears forming 86 per cent., myelocytes 3 per cent., lymphocytes 9 per cent., and eosinophils 1 per cent. Poikilocytosis was a well-marked feature. Biochemical investigations indicated a blood calcium figure of 8.6 mg. in 100 c.c.m., and blood fragility was complete in 0.4 per cent. of saline; van den Bergh's test gave a direct negative and an indirect weak positive reaction. The blood pressure was 110 mm. systolic and 70 mm. diastolic. The patient was submitted to a course of deep x-ray therapy, under which the leucocyte count came down to 7,800, and the haemoglobin to 42 per cent. The patient died after a fit on September 13th.

The necropsy showed marked enlargement of the anterior and posterior chains of the glands in the neck. There was a swelling in the middle line of the neck, which on incision was found to be an abscess of the size of an orange, with a well-defined wall; it contained yellow pus. There were a number of smaller glands in the axilla, which were discrete and slightly movable; a group of glands could be felt also in the groin. Obliterating pleural adhesions were present on the right side, while in the left pleural cavity there was an empyema pushing down the diaphragm and causing a pressure collapse of the base of the left lung, which was adherent above, completely shutting off the empyema. There was some oedema of the tissues of the superior mediastinum. A lump of matted glands formed a large swelling surrounding the bronchi and the aortic arch, filling up the shoulder girdle, and extending behind the heart. There was no infiltration of the pericardium. The left lung was oedematous at the apex, the base was collapsed; the pleura was thickened and adherent, shutting off the empyema. The tracheo-bronchial glands were enlarged and pigmented; they showed foci of caseation. The right lung showed pleural tubercles near the hilum suggestive of lymphatic spread. Microscopically, thickening of the pleura was found, and miliary tubercles were seen. The heart showed brown atrophy. In the liver there were a few miliary tubercles, underneath the capsule as well as inside the organ; there was also acute congestion. The spleen was not enlarged, but was smaller than normal; there were white opaque areas of variable size which, on microscopical examination, proved to be caseating and conglomerate tubercles, with well-developed giant cell systems and commencing caseation. There was marked fibrosis. Both kidneys contained pyaemic abscesses and a few retention cysts. The other organs were healthy, except that the peri-pancreatic glands were enlarged. The periosteum of the right clavicle was infiltrated, and the marrow of the bone itself was involved.

The glands on section presented those curious yellowish-white opaque flecks which resembled tuberculous caseous material, but were rather more firm, more opaque, and discrete; they stood out prominently from the surrounding tissue. Some glands showed a number of these tiny opaque flecks which were not fused together like tuberculous caseous material. Other glands showed marked enlargement, and on section no necrotic areas were found, but there were typical gelatinous areas with strands of fibrous tissue running into the gland from the capsule. There was some peradenitis in the intrathoracic group, but very little in the smaller glands.

Microscopically the glands that were uniformly enlarged showed well-marked fibrous transformation; here and there a few groups of lymphoid cells were seen, but the differentiation of the gland into lymph sinuses and follicles was entirely lost. There was, however, no diffuse endotheliosis, but well-marked fibrosis of the

sclerosing type, with fully formed fibrous tissue. The lymphoid accumulations between these strands were found to be composed of a few endothelial cells, with rather pale large nuclei arranged in a cluster in the centre, so that the protoplasm formed only a scanty rim round them—a picture which Andrews has defined as quite distinct from that of tuberculous lymphadenitis. The nuclei were mostly oval, with a ring of distinct nuclear membrane, and one or two dark chromatin nodes. Eosinophils were very scanty in the sclerosed glands, but numerous in the soft ones. The lymphoid cells were of the large and small lymphocytic type.

The presence of myelocytes in the blood is interesting and unusual. It will be remembered that the earlier authors have described cases of lymphadenoma with a myelocytic blood picture, and Cohnheim first applied to it the term "pseudo-leukaemia," assuming that it was a form in which the leukaemic blood picture was absent. Rolleston holds that the presence of leukaemic blood changes would now, with our modern accuracy of haematological methods, rule out a diagnosis of Hodgkin's disease. But in this undoubted case of Hodgkin's disease the presence of a few myelocytes indicates response of the bone marrow, showing that the process is not very far removed from a real leukaemic hyperplasia. It may be added that Fabian has recorded a few myelocytes in cases of Hodgkin's disease, but they were too few to estimate. The presence of myelocytes may indicate involvement of the bone marrow, since undoubted involvement of the clavicle was found in the case mentioned above.

The hyperleucocytosis of 56 per cent. is evidently a reaction to sepsis from the empyema and abscess in the gland. The polynuclear leucocytosis of Hodgkin's disease is both absolute and relative, but the counts seldom exceed 20,000.

The occurrence of an abscess in one of the glands in the neck is unusual, and raises the question whether these necrotic foci are the starting point of the abscesses. This, again, may suggest the view that lymphadenoma is a secondary infection—a view which is emphasized by Horder—an infection supervening on some previous chronic lymphadenitis.

With regard to the eosinophilia, Longcope holds that it is most common where there are necroses in the glands. Bunting, on the other hand, contends that eosinophils are low in the blood in the active stages of the disease, seeing that they all emigrate into the lymphatic glands, the destruction of the lymphocytes there apparently serving as a stimulus. In this case it is noticeable that necrosis in the glands was well marked, but the eosinophilia was slight.

## LITERATURE.

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T. K. MENON AND T. B. MENON: COEXISTENCE OF LYMPHADENOMA AND TUBERCULOSIS.



FIG. 1.—Photograph of the intra-thoracic glands with the foci of necrosis. (By the courtesy of Captain Barnard, Radiologist, General Hospital, Madras.)

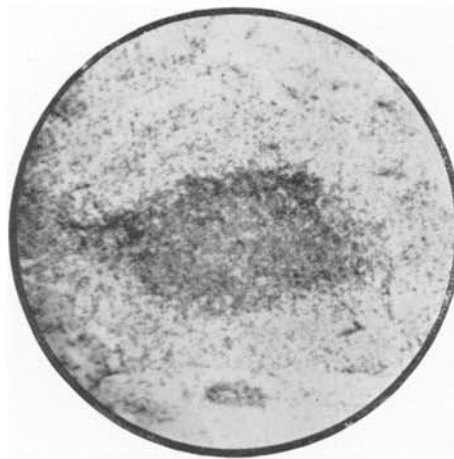


FIG. 2.—Showing the lymphoid areas surrounded by dense fibrous tissue. ( $\times 82$ .)

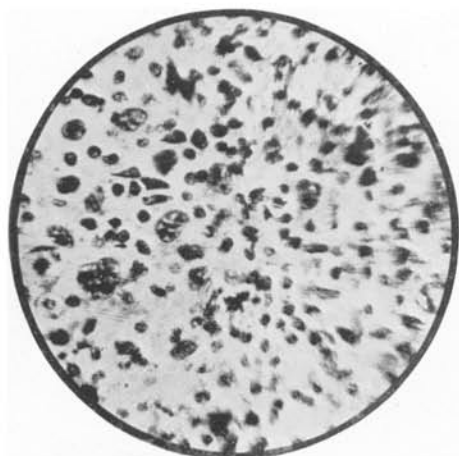


FIG. 3.—Showing the endothelial giant cells and multinucleate endothelial cells. ( $\times 720$ .)

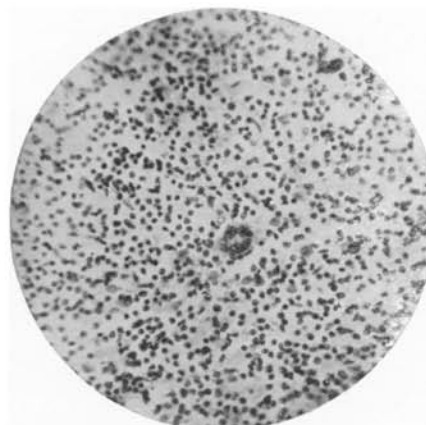


FIG. 4.—Showing an endothelial giant cell with large nuclei arranged round the periphery. ( $\times 450$ .)

R. MORTON: RAPID HEALING OF A GASTRIC ULCER.

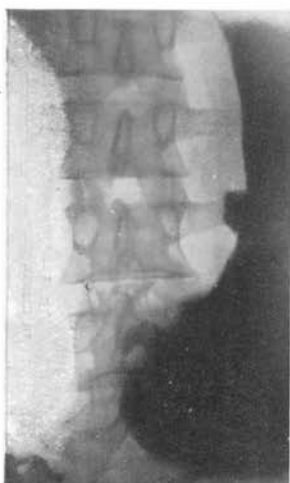


FIG. 1.



FIG. 2.



FIG. 3.