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TUBERCULOUS PERICARDITIS AS A SOLE TUBERCULOUS LESION IN AN OLD MAN.*

By J. M. ALSTON, M.B., M.R.C.P.E.

THIS case presents the unusual features of a very advanced tuberculous infection of the pericardium in an old man of very fine physique, in whose body no other naked-eye tuberculous lesion could be discovered; and it raises interesting questions in regard to the occurrence of active tuberculous lesions in old people, as well as to the route of infection when the pericardium is the only tissue affected.

CASE.—George B., aged 70. The patient was an old soldier who gave a history of unusually good health and who stated that he had had no illnesses previous to an attack of influenza about two months before his admission to hospital. Since this illness he had suffered from breathlessness and a slight precordial pain which was increased on coughing.

When the patient was examined on admission to hospital, great dilatation of the heart was found; the heart sounds were feebly audible and auricular fibrillation developed later.

The patient died on 3rd July 1926, eighteen days after admission.

(For permission to make use of these clinical notes I am indebted to the kindness of Professor G. Lovell Gulland, to whose ward the patient was admitted.)

Post-mortem Notes.—Post-mortem examination was performed on the day of death. The body showed very fine development and was well nourished. There was a little œdema of the feet and legs.

When the thorax was opened the pericardial sac was found to be entirely obliterated. The visceral and parietal layers of the pericardium were thickened and were covered by a hæmorrhagic, fibrinous exudate which bound them firmly together. After these two layers had been

* Read 28th January 1927.

separated, tubercles were found beneath the fibrinous exudate on the right and left auricles posteriorly. The exudate on each layer was roughly in the form of a meshwork, and this appearance became more marked after the specimen had been hardened.

The maximum thickness of the fibrinous exudate separating the layers of the pericardium was $\frac{1}{4}$ to $\frac{1}{3}$ inch. At some parts, particularly at the base of the heart, there was a layer of healthy sub-epicardial fat

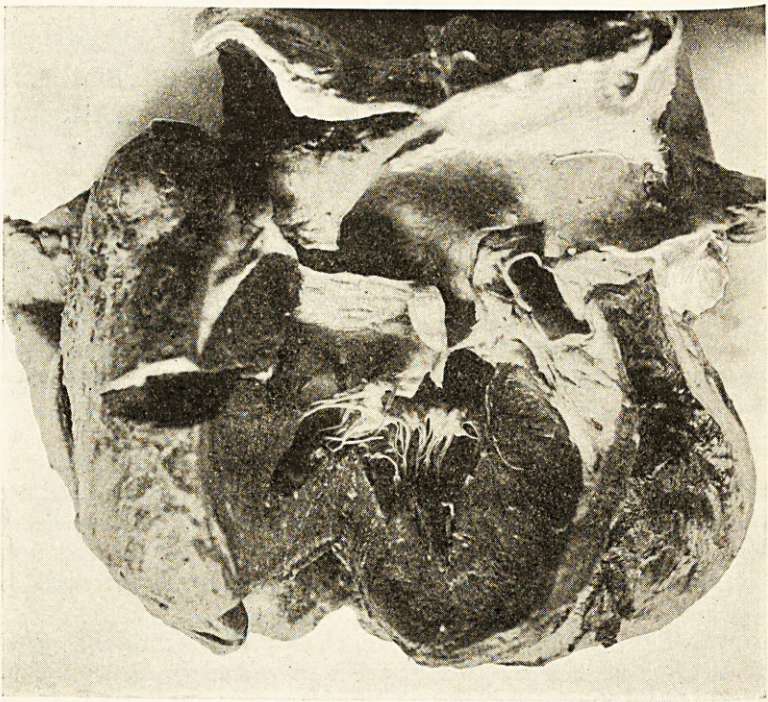

FIG. 1.

between the pericardial exudate and the myocardium, and this layer of fatty tissue decreased as the apex was approached until the tuberculous exudate lay directly on the muscle, although there was no sign that the muscle was invaded.

The heart itself was enlarged. The cavity of the right ventricle and the orifice of the tricuspid valve were distended, and the myocardium of this chamber showed fatty infiltration. The wall of the left ventricle was greatly hypertrophied and the cavity was very much diminished. The arch of the aorta showed general dilatation. There were a few patches of atheroma, especially in the descending aorta.

Microscopically, on the surface of the visceral pericardium there was found a thick layer of tuberculous tissue, of subacute type, consisting of large numbers of small round cells and endothelial cells, areas of caseous material with numerous giant cells, and a small amount of fibrous tissue. This tuberculous tissue did not at any part of the section invade the myocardium, although it was in direct contact and continuation with it at some parts, and was separated by some healthy sub-epicardial connective tissue at others. On its pericardial surface, the tuberculous tissue described was covered by a thick layer of fibrin,

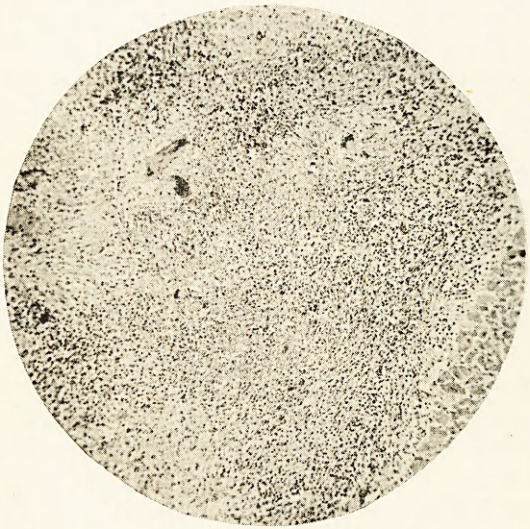

FIG. 2.—Note the healthy myocardium on the right at the bottom, and the cellular tuberculous tissue, with several giant cells, in the rest of the field.

among which there were hæmorrhages. A section of the parietal pericardium showed a similar condition of tuberculous tissue covered by fibrin. The myocardium seen in the section showed a slight degree of atrophy of the muscle fibres. By Ziehl-Neelsen's stain only two or three acid-fast bacilli could be seen in any of the sections prepared from the visceral pericardium.

No other naked-eye lesion of tuberculosis was present in any organ, including the lungs, tonsils, cervical, bronchial and abdominal lymph glands, gastro-intestinal tract, liver, spleen, kidneys, bladder and prostate.

Microscopically, a mediastinal lymph-gland of normal size was found to show a uniform proliferation of cells of the reticulo-endothelial system and in different sections various very small tuberculous lesions,

some showing commencing caseation and giant-cell formation, were seen. It was noticeable that many of the endothelial cells in this gland contained carbon pigment particles. No acid-fast bacilli were seen in sections stained by Ziehl-Neelsen's method.

The lungs showed only a slight degree of venous congestion and were otherwise healthy. In particular, there was no puckering at the apices, no thickening of the pleuræ at any point, and though the lungs were thoroughly investigated there was found no naked-eye evidence of tuberculous infection. There was an excess of clear fluid in the pleural sacs. The liver and spleen showed by naked-eye and microscopical examination, a moderate amount of venous congestion but no evidence of tubercle. The kidneys showed some interstitial fibrosis of irregular distribution and small in amount, and most probably due to the age of the patient. The gastro-intestinal tract showed no pathological change. There were no tuberculous glands in the mesentery.

No lesions of the bones were discovered.

Remarks.—The case is interesting from both the clinical and the pathological points of view. The presence of a grave tuberculous pericarditis without any clinical evidence more definite than breathlessness, slight precordial pain and slight œdema is remarkable.

Pathologically, the case raises the question of senile tuberculosis and particularly of the occurrence of active tuberculous lesions in old people without any obvious old-standing infection. In this case, the lesion is single except for the very slight and very recent lesion in a mediastinal gland, and the route of infection is very difficult to detect on that account. It is usually considered that a new tuberculous lesion in adult life is brought about by the activity of tubercle bacilli which have remained latent in the body for a longer or a shorter time; the primary infection may or may not have been associated with demonstrable signs or symptoms. The second outbreak of the disease may be at the site of the first, or it may occur in some other organ or tissue which the bacilli may have reached by any of the modes in which bacteria spread within the body. The alternative to the theory of a long-lasting infection is that the lesions which develop in adult life are the expression of a recent first infection.

In this case positive evidence of the time and route of infection was conspicuously absent, but the view that the pericarditis was due to a secondary blood-spread infection from a small lesion, not discovered at autopsy, is considered the most

likely explanation on the following grounds. First, the tissues adjacent to the pericardium and those in lymphatic connection with it—namely, the myocardium, the mediastinal tissues and the lungs—showed no evidence of a tuberculous infection except for a small lesion, more recent than the pericarditis, in a mediastinal gland, so that it would seem most probable that the bacilli were brought to the pericardium by the blood; and secondly, a blood-spread infection of tubercle bacilli is nearly always from a previously developed lesion and not by direct passage from the surface of the body to the blood stream. If the primary infection had been recent the lesion produced by it might be expected to be evident, but no such lesion was found, so that the suggestion is tentatively made that the primary lesion is an old-standing one.

No evidence was to be found pathologically of a cause predisposing the pericardium, in particular, to infection by tubercle bacilli. In view of the connection (especially in former days) between the patient's occupation (that of a soldier) and localised thickenings of the epicardium it would be interesting to know if tuberculous pericarditis is found to be of commoner occurrence in soldiers than in people of other occupations.

Chronic adherent pericarditis is often associated with hypertrophy of the myocardium, especially if there is chronic mediastinitis and adhesions between the parietal pericardium and the tissues surrounding it. In the present case there was no mediastinitis, and there was hypertrophy, seen most, and in high degree, in the left ventricle.

The following considerations can be made as to the age of the pericarditis. The lesion shows a uniform appearance, as noted above, of a thick layer of tuberculous tissue, with very scanty tubercle bacilli, covered by a layer of fibrin which shows hæmorrhage. About two months before death an acute illness occurred, and the onset of pericardial symptoms followed it. It is suggested that the tuberculous pericarditis had commenced before this acute illness and that it became more active after it. Judging by the microscopical appearance of the lesion, six months to a year is suggested as the probable age of the infection of the pericardium.

During the time that the patient was in hospital auricular fibrillation was present and presumably it accounted for the slight œdema of the lower limbs, the pleural effusion and the

venous congestion of the abdominal organs; and presumably the cardiac irregularity, as well as the embarrassment caused by the pericarditis, was the cause of death.

This case illustrates among other things hypertrophy of the left ventricle in association with tuberculous pericarditis, and Professor J. Lorrain Smith has drawn my attention to a case, of which an account[1] was published by him and H. L. McKisack, which contrasts markedly with the present one. The contrasting case was of a boy who died at the age of twelve years with massive tuberculous inflammation of the pericardium and smaller tuberculous lesions in the right lung and right knee. There was no hypertrophy of the myocardium and the chambers of the heart were smaller than normal, but the total amount of blood and of hæmoglobin and the number of red corpuscles per c.mm. were all enormously and progressively increased while the patient was in hospital and up to the time of his death. There seemed to be an attempt to compensate for the lack of hypertrophy of the myocardium by an increase of the oxygen-carrying function of the blood. While he was under observation the hæmoglobin content was found by several estimations to be rising steadily, and by assuming that the rate of increase had been uniform throughout the process, the commencement of the increase was dated back, about a year, to the time when the earliest clinical evidence of tuberculous infection, in the form of synovitis of the right knee, made its appearance.

I have much pleasure in expressing my thanks to Professor Lorrain Smith for permission to publish this case, and for the help which he has given me in preparing it for publication.

REFERENCE.—[1] Lorrain Smith, J., and McKisack, H. L., "A Case in which Cyanosis and Plethora occurred in association with Adherent Pericardium" (*Trans. of the Path. Soc. of London*, Vol. LIII., Pt. 1, 1902).