TUBERCULOUS PERICARDITIS*

BY

R. McLAREN TODD

From the Department of Child Health, University of Liverpool

(RECEIVED FOR PUBLICATION FEBRUARY 2, 1950)

Tuberculous pericarditis was formerly thought to be an uncommon condition, but the more recent recognition of the tuberculous aetiology of many cases of chronic constrictive pericarditis (Sellors, 1946; Edwards, 1948; Parsons-Smith, 1948) should stimulate us to attempt the diagnosis of tuberculosis of the pericardium in its earliest stages.

The incidence of the disease is generally considered to be approximately 1% of all necropsies and 3% of patients dying from tuberculosis (Sweeney, 1940). A review of the literature of the past 50 years suggests that the frequency of tuberculous pericarditis has steadily increased. This trend is seen when the incidence is expressed as a percentage of all necropsies, as well as a percentage of tuberculous conditions (Table 1), but necropsy records in all probability do not reflect the true incidence because complete healing of the tuberculous pericardial lesion may occur. The clinical recognition of tuberculous pericarditis may also be difficult, and some of the cases of pericarditis diagnosed as rheumatic are probably tuberculous in nature. Furthermore, the symptoms of tuberculous pericarditis may be so mild that the patient does not seek medical advice, and even if suspected, the diagnosis may not be confirmed either by guinea-pig inoculation or by biopsy of the pericardium.

The case now to be described has many of the common features of the disease but is unusual because a caseous gland had ruptured into the pericardium.

Case Report

G.B., a boy aged 5 years, had measles six weeks before admission to hospital. He seemed to recover completely within two weeks, but one week later an irregular fever developed, associated with a slight cough and wheeze. Subsequently he had bouts of abdominal pain and vomiting. The bowels were regular and there was no disorder of micturition. He was an only child, his mother and father were well, and there was no family history of tuberculosis. Before the attack of measles he had enjoyed good health.

On admission he had a temperature of 100° F., pulse rate was 140 per minute and respiration rate 26 per minute. His tongue was moist, and the throat reddened, but no enlarged glands were felt in the neck. The left side of the chest showed diminished movement, and the percussion note was impaired over the left chest anteriorly, in the left axilla, and to the right of the sternum. The breath sounds were tubular in the left axilla. The heart was enlarged, the apex beat being in the left fifth intercostal space outside the mid-clavicular line, and the heart sounds were soft and distant. There were no bruits but a friction rub was heard down the left border of the sternum. No abnormality was detected in the abdomen, and the urine

TABLE 1
FREQUENCY OF TUBERCULOUS PERICARDITIS OVER 50 YEARS

Author	Year	Total Necropsies (All Ages)	Tuberculous Conditions	Cases of Tuberculous Pericarditis		
				Total	Percentage of All Necropsies	Percentage T.B. Necropsies
Osler ..	1893	1,000	275	7	0.7	2.5
Norris ..	1911	7,219	1,780	82	1.1	4.6
Blalock and Levy	1937	1,653	—	42	2.6	—
Suzman ..	1943	1,893	102	6	0.32	5.9

* Paper read before the Liverpool Paediatric Club on November 18, 1949.

FIG. 1.—Heart showing tuberculous pericarditis. The caseous gland is outlined and a glass rod (X) is in the position of the fistula between this gland and the pericardial sac.

was normal. A radiograph of the heart on the day of admission showed a pericardial effusion and consolidation at the left base. The electrocardiogram showed simple tachycardia only, with normal voltage. The Mantoux test in a 1 in 10,000 dilution was positive after 48 hours (1 cm. erythema with small central wheal). On the day after admission 20 ml. of greenish-yellow fluid were aspirated from the pericardial sac. This fluid contained a small number of lymphocytes. Tubercle bacilli were not seen or grown on culture. Penicillin (200,000 units) was injected into the pericardial sac at the initial and subsequent aspirations, from none of which were tubercle bacilli isolated. Pericardial fluid

accumulated rapidly in spite of repeated aspirations, the neck veins became congested, the liver became enlarged, and operative drainage of the pericardium was advised.

At operation the pericardium was immobile and on palpation the cardiac pulsation was minimal. When the pericardium was incised approximately 20 oz. of greenish-yellow fluid gushed out and the cardiac pulsations became visible.

A portion of the pericardium was removed and the histological report was as follows:

The granulation tissue arising from the pericardium was heavily infiltrated with chronic inflammatory cells, and the presence of numerous epithelioid follicles supported the diagnosis of tuberculous pericarditis. Tubercle bacilli were not found.

Subsequently the wound became secondarily infected and death occurred 36 days after operation.

NECROPSY. Necropsy was performed 18 hours after death. A cavity, 3 cm. in diameter, discharging offensive light green pus, was present over the lower end of the sternum slightly to the left of the midline.

The dura was tense, and the brain (320 g.) was congested.

On elevation of the sternum it was apparent that the cavity led directly into the pericardial sac which contained about 10 ml. of purulent fluid. The visceral and parietal layers of the pericardium

were covered with thick purulent exudate of a green colour, and there were light adhesions between the heart and diaphragm. There was no pleural fistula. The under surface of the sternum, just cephalad to the drainage wound showed scanty tubercles. At the extreme left upper zone, lateral to the pulmonary artery and in the position of the ductus arteriosus, there was a fistula 2 mm. in diameter leading into a caseous lymph node which was adherent to the parietal pericardium (Fig. 1).

The pleural cavity of the right lung contained a few millilitres of clear yellow fluid. There were recent adhesions between the anterior border of that lung and the pericardium. Tubercles were visible when the surfaces were separated.

The anterior portion of the upper lobe of the left lung was firmly adherent to the pericardium, and in the portion overlying the heart there was a caseous focus

FIG. 2.—Tomogram (A.P. view) to compare the diameter of the normal right main bronchus and the narrowed left main bronchus.

showed the features of tuberculous bronchopneumonia.

The brain showed no evidence of tuberculous meningitis.

Discussion

Some writers consider that tuberculous pericarditis may be either primary or secondary to tuberculosis elsewhere. Blatt and Greengard (1928) state that primary tuberculous pericarditis, in the strict sense of the term, is extremely rare, and Ellman (1945) considers that it does not exist for all practical purposes. Furthermore, it is unreasonable to assume that inhaled or ingested tubercle bacilli will cause disease of the pericardium only and not of adjacent organs. The probable explanation is that the primary focus was so small and had healed so completely that it has become unrecognizable macroscopically. Thompson (1933), however, collected 21 cases from the literature and added seven of his own in which the pericardial lesion was the only manifestation of tuberculosis. These cases

approximately 3 by 2 cm. There was caseous bronchopneumonia of the left lower lobe.

There were 50 ml. of clear yellow fluid, with small fibrin clot, in the peritoneal cavity. Tubercles were observed on the under surface of the diaphragm above the spleen. The gastro-intestinal tract was healthy. The spleen (75 g.) was firm and congested. The liver (752 g.) was firm, and showed marked nutmeg mottling. The kidneys, adrenals, and mesenteric glands were normal.

MICROSCOPY. The liver showed marked fatty infiltration, largely peripheral in distribution with light portal infiltration of lymphocytes and plasma cells.

The spleen showed scanty areas of caseation with early giant cell formation, blending, without a marginal lymphocytic zone, into the surrounding pulp.

The pericardium consisted of granulation tissue showing varying degrees of organization and dotted with soft tubercles showing central caseation, grading into the surrounding tissue without a defined lymphocyte border. There were also deposits of fibrin with intense polymorph infiltration indicating an added pyogenic inflammatory reaction.

Section of the caseous area of the left upper lobe overlying the heart showed a cavity lying centrally in a zone of caseating lung tissue, and surrounded by caseating tuberculous bronchopneumonia. The portion of the left lower lobe sectioned

FIG. 3.—Tomogram showing the outline of the two main bronchi and glandular pressure on the left main bronchus.

were in adults between the ages of 49 and 84 years. In Harvey and Whitehill's (1937) 34 cases, there was only one case of primary tuberculous pericarditis in which the mediastinal glands were not caseous, the lungs were normal, and no tuberculous focus was found elsewhere.

Secondary tuberculous pericarditis may occur in one of the three following ways. First it may occur as part of a miliary spread and usually presents no clinical evidence during life. Such a condition was observed in a girl of 12 years with tuberculous meningitis, in whom the visceral pericardium was studded with miliary tubercles as part of the generalized miliary dissemination.

Secondly it may be caused by lymphatic spread from a caseous lymphatic gland, the route which is considered by most observers to be the commonest by which the pericardium is involved.

Osler (1893) held that tuberculous pericarditis was due in the majority of instances to infection of the pericardium through lymphatic channels by retrograde spread from caseous mediastinal glands. One of his cases was a boy aged 5 years, another was a boy aged 16 years, and the oldest was a man of 72 years. Kinney and Douglass (1937) also emphasized the importance of mediastinal gland tuberculosis in the aetiology of tuberculous pericarditis. Blalock and Levy's 42 cases (1937) all had tuberculosis of mediastinal nodes, and Heimann and Binder (1940) found tuberculous root glands in all their 31 cases. Barrett and Cole (1944) described a male patient aged 22 years who was found at necropsy to have, in addition to tuberculous pericarditis, a caseous tuberculous gland near the left bronchus between the main left pulmonary artery and the arch of the aorta. The gland was situated at 1.5 cm. from the pericardium at its nearest point, and no definite track between gland and pericardium was apparent.

Suzman's (1943) case of a man of 23 years showed caseous glands at both hila, and he concludes that 'in this case the primary focus was a tuberculous gland in the mediastinum secondarily affecting the pericardium; and later as the disease progressed, further dissemination occurred, causing generalized miliary tuberculosis.'

The third route is direct extension from an inflamed mass of hilar glands or pleura. This may also give rise to tuberculous pericarditis but is usually considered to be a most unusual mode of origin.

One of Osler's cases (1893), a man of 39, had a mass of caseous glands adherent to the pericardium. In Keefer's (1937) 20 cases, 11 had enlarged tuberculous mediastinal glands and 'in several the pericardium had become involved as a result of

rupture of a caseous lymph node directly into the pericardial sac.' In others there was evidence that the process had extended to the pericardium from the pleura, the lung, or even the peritoneum. Among these cases there was only one detailed case report which described the rupture of a gland directly into the pericardial sac.

Seligman and Lederer (1940) reported a case of acute suppurative pericarditis in a woman of 54 years which had resulted from the perforation of a pyogenic abscess in a tracheobronchial lymph node and in which the tubercle bacilli were found in the wall of the gland draining a fibrotic pulmonary Ghon focus.

Smellie (1925) reported a similar happening in a girl of 8 years. She had caseous glands in the superior mediastinum, at the tracheal bifurcation, and in both lung roots. Almost all the caseous material had been extruded from the bifurcation gland, leaving a cavity about $1\frac{1}{2}$ in. $\times$ $\frac{7}{8}$ in. A secondary infection with *Bact. coli* had taken place. There was a pin point perforation into the pericardium with localized suppurative pericarditis.

Among Harvey and Whitehill's (1937) 95 cases is a report of a man of 64 with tuberculous pericarditis which arose from the rupture of a caseous gland into the pericardium.

Churchill (1937) found an extrapericardial tuberculous abscess containing 1 oz. of pus at operation for constrictive pericarditis on a man who had suffered from tuberculous pericarditis in early life. This abscess was situated external to the pericardium but perforating into it, suggesting that a suppurating tuberculous mediastinal lymph node had at one time set up a transient tuberculous effusion which had healed, or possibly, that the lymph node abscess had by contiguity caused a pericardial reaction resulting in scar formation.

These examples of tuberculous pericarditis caused by rupture of a caseous lymph node are the only ones I have been able to trace in an extensive search of the literature, and they support the view of Hannesson (1941) that 'pericarditis as a result of direct extension of infection from a neighbouring focus is most uncommon.'

In the patient I have described, the gland which ruptured into the pericardial sac was the gland (Engel's gland) of the ductus arteriosus (Engel, 1947). In a further case of tuberculous pericarditis treated at St. Thomas's Hospital by Dr. Goadby this same gland was enlarged.

Case Report

The patient was a boy of 14 years admitted to hospital with a severe, non-productive cough of one month's duration. He had a temperature of 102° F., there were

signs of pericardial effusion, and this diagnosis was confirmed radiologically. Over the course of three weeks the patient's condition gradually improved, although there was intermittent fever and his pulse averaged 120 per minute. Seven weeks after admission the patient was allowed up for the first time, having had a normal temperature during the preceding month and an average pulse rate of 88. His condition, however, deteriorated and he developed a tachycardia of 120 and an irritative reflex cough. Two weeks later he was allowed up once more but the cough and increasing congestion of the neck veins and liver required further bed rest. At this time x-ray screening was performed, and it was noticed that the left side of the chest was more expanded than the right, its movement was greatly decreased, and the whole lung field was noticeably more translucent than the right. A glandular shadow was seen opposite the arch of the aorta and tension emphysema due to partial obstruction of the left main bronchus was diagnosed. The tomograms confirmed this diagnosis (Figs. 2 and 3).

Thoracic surgeons (Edwards, 1949) are aware of the thinness of the pericardium in the region of the ductus arteriosus when they operate for patency of this structure, but I am doubtful if it can be argued from this that the pericardium of patients with normal obliteration of the ductus might also be thin at this point. In fact it could be reasonably held that the normal fibrosis of the ductus would result in an increased thickness of the pericardium in this area.

At Alder Hey Hospital the gland of the ductus arteriosus was present in eight out of 10 consecutive necropsies. Its size was variable and up to 1 cm. in length. The gland was situated anterior to the ligamentum arteriosum and overlapped the pericardium to a variable degree (Fig. 4). The node was separated from the pericardium by very loose areolar tissue. A similar arrangement was found posteriorly where the inter-bronchial node overlapped the pericardium, though not so frequently. The fold of pericardium in the vicinity of the ligamentum arteriosum was found to be slightly thinner and of looser texture than that over the phrenic nerve area of the left ventricle, though the difference was not very great.

FIG. 4.—Post-mortem specimen demonstrating the gland of the ductus arteriosus (Engel's gland) which has been reflected to the side, the original position being marked with interrupted lines. The opened aorta is on the left, and a marker (X) is placed behind the ligamentum arteriosum. The other marker is in the fold of pericardium on which the gland is situated.

The gland of the ductus arteriosus drains a relatively small area of lung, namely the apex of the left upper lobe, but it frequently contains caseous material even when no tuberculous focus occurs in its drainage area. The following two post-mortem reports illustrate this point.

At necropsy on a boy of 3 years with tuberculous meningitis a caseous focus was shown a third of the way along the fissure separating the left upper lobe from the lower lobe. There was a caseous gland at the hilum in relation to the upper lobe bronchus and the gland of the ductus arteriosus overlying the reflection of the pericardium was also involved: it was firm and white with scanty caseous areas.

At necropsy in a child of 2 years with tuberculous meningitis adhesions showed between the medial aspect of the upper lobe of the left lung and the mediastinum over an enlarged lymph node $1\frac{1}{2}$ cm. $\times$ 1 cm. lying against the pericardium at its point of reflection from the pulmonary artery. The lymph node was caseous and showed areas of calcification. At the inferior border of the upper lobe approximately half way along there was a small caseous focus. Similar lesions were present in the left lower lobe. The most probable explanation is that the gland of the ductus arteriosus was

secondarily involved by spread from neighbouring lymphatic glands.

Summary

Tuberculous pericarditis is still considered to be a rare disease in childhood but the recognition at operation of the tuberculous nature of a high proportion of cases of constrictive pericarditis suggests that the disease may occur more frequently than it is diagnosed.

Even when suspected the diagnosis is difficult to confirm either by guinea-pig inoculation or by biopsy of the pericardium.

The primary and secondary types of tuberculous pericarditis are discussed, and the clinical details of a case of tuberculous pericarditis resulting from the rupture of a caseous gland adherent to the pericardium are described together with other cases in which the gland of the ductus arteriosus (Engel's gland) was responsible for the spread of tuberculosis to the pericardium. The involvement of this gland may be merely fortuitous but the anatomical situation of the gland, contiguous with the pericardium at its reflection, would readily facilitate pericardial involvement.

I wish to express my thanks to Dr. E. G. Hall, pathologist at Alder Hey Hospital, for the detailed pathological investigations; to Dr. H. K. Goadby for

permission to refer to one of his cases; and to Professor Norman B. Capon for his advice and criticism.

REFERENCES

- Barrett, A. M., and Cole, L. (1944). *Brit. Heart J.*, **6**, 185.
 Blalock, A., and Levy, S. E. (1937). *J. thorac. Surg.*, **7**, 132.
 Blatt, M. L., and Greengard, J. (1928). *Amer. J. Dis. Child.*, **35**, 631.
 Churchill, E. D. (1937). *J. thorac. Surg.*, **7**, 150.
 Edwards, F. R. (1948). *Brit. med. J.*, **1**, 513.
 — (1949). Personal communication.
 Ellman, P. (1945). *Brit. Heart J.*, **7**, 147.
 Engel, S. (1947). 'The Child's Lung.' London.
 Hannesson, H. (1941). *Tubercle, Lond.*, **22**, 79.
 Harvey, A. M., and Whitehill, M. R. (1937). *Medicine, Baltimore*, **16**, 45.
 Heimann, H. L., and Binder, S. (1940). *Brit. Heart J.*, **2**, 165.
 Keefer, C. S. (1937). *Ann. intern. Med.*, **10**, 1085.
 Kinney, W. M., and Douglass, J. E. (1937). *Trans. nat. Tuberc. Ass. N.Y.*, **32**, 154.
 Norris, G. W. (1911). 'Studies in Cardiac Pathology.' Philadelphia.
 Osler, W. (1893). *Amer. J. med. Sci.*, **105**, 20.
 Parsons-Smith, B. T. (1948). *Proc. R. Soc. Med.*, **41**, 431.
 Seligman, B., and Lederer, M. (1940). *N.Y. St. J. Med.*, **40**, 130.
 Sellors, T. H. (1946). *Brit. J. Surg.*, **33**, 215.
 Smellie, J. M. (1925). *Brit. J. Child. Dis.*, **22**, 110.
 Suzman, S. (1943). *Brit. Heart J.*, **5**, 19.
 Sweeney, J. A. (1940). *Amer. Heart J.*, **20**, 345.
 Thompson, W. P. (1933). *J. Amer. med. Ass.*, **100**, 642.