

PRIMARY TUBERCULOSIS OF THE LUNG AND SOME OF ITS CONSEQUENCES

By A. MARGARET C. MACPHERSON, M.D., M.R.C.P.

(*Chief Assistant, Children's Contact Investigation Department, Brompton Hospital.*
Physician, Elizabeth Garrett Anderson Hospital.)

Tuberculin tests show that by the time childhood is ended, over 80 per cent. of London children have received their first infection by the tubercle bacillus (Lloyd and Dow) (1). On the other hand, the number of notified cases of tuberculosis in childhood in urban districts is at a very much lower level. It follows therefore that for the majority of children the entrance of tubercle bacilli into the body is unaccompanied by evidence of disease. Moreover, in a series of children studied in the Children's Contact Department at the Brompton Hospital it was possible to detect symptoms or clinical signs in only a few of the children who received their first tuberculous infection while under observation (2). A primary tuberculous infection, therefore, may be considered to be a more or less normal happening at the present time in the lives of most children in urban districts and the mere presence of a positive tuberculin test need not necessarily give rise to undue apprehension if it is unaccompanied by abnormal clinical findings or gross radiological changes.

Primary Complex.

The primary complex resulting from this original infection by tubercle bacilli consists of a lesion in the lung parenchyma accompanied by changes in the corresponding lymph nodes. The lesion in the parenchyma is usually small and often cannot be detected in the X-ray film and not always at autopsy even after careful search has been made. During healing of the lesion by fibrosis, and specially if and when calcium is deposited, then what previously passed unnoticed may become a distinct, but still small, opacity in the X-ray film. In some cases the pleura covering the primary lesion is involved and becomes thickened or a small interlobar effusion appears and these will indicate the position of the lesion in the lung. It is surprising that pleural involvement is not of more frequent occurrence since the primary lesion is often found in the peripheral part of the lung immediately beneath the pleura.

Glandular lesions associated with primary infection are usually more evident than the pulmonary lesion and this is specially so in younger children. The corresponding glands at the hilum or perihilar regions become enlarged and are usually visible in the X-ray film as an enlarged hilar or paratracheal shadow; in some cases individual glands can be distinguished but as a rule this is not possible, especially during the early stages. The opacity is formed not only by the caseous glands but by periglandular reaction and, where the glands are much enlarged, by collapse of lung tissue in the immediate neighbourhood due to pressure.

The enlarged glands diminish in size slowly, taking several months or even years to return to normal size and, during the healing process, calcium may be deposited in them giving rise together with the calcified lesion in the parenchyma to the nodes and foci described by Parrot and Ghon.

Although most children receive a primary tuberculous infection without it showing any clinical evidence and the primary lesions pass through the stages just described without any but radiological manifestations, there are some children who are less fortunate and may develop definite signs and symptoms. These are caused either by a spreading of the pulmonary lesion or by secondary effects of glandular infection. It is not possible to predict how any given child will react to primary infection but there is no doubt that certain conditions will predispose to the development of these complications. It stands to reason that the degree of infection to which a child is exposed must be of significance; a large number of bacilli inhaled at one time may overwhelm the natural resistance of the lung and lymphatic tissues. Natural resistance itself is not a constant condition. It varies in different races, in different localities, in different families and probably in the same person at different times, as is shown by the effect of conditions such as measles and whooping cough on the incidence of tuberculosis in children.

Spreading primary lesion.

Instead of the usual small, localised and encapsuled lesion, the parenchymatous lesion may develop into a large area of tuberculous broncho-pneumonia involving a considerable part of one

Postgrad Med J: first published as 10.1136/pgmj.18.201.139 on 15 May 1962. Downloaded from http://www.bmj.com/ on May 7, 2022 by guest. Protected

lobe of the lung. There may be steady progression of this process ending fatally with a terminal miliary spread. In some cases, the condition is subacute and healing may take place with little residual scarring.

Again, the primary lesion may spread, but in a less acute manner, by formation of "daughter" tubercles outside the capsule, and so give rise to slowly spreading fibro-caseous disease. In my experience this is not a common occurrence, but it has been described by H. C. Sweaney (3) as a possible mode of development of the adult type of disease.

Others have reported non-specific pneumonic consolidation surrounding small primary tuberculous lesions of the lung. This was the explanation given by Eliasberg and Neuland (4) of the clinical condition which they described and named epituberculosis. Pathological confirmation of this condition is scanty but H. S. Reichle (5) has described such a reaction found at autopsy.

Among the children I have examined these are not the usual lesions; much more frequent are the abnormal conditions consequent on reactions in the lymph glands.

Secondary effects of glandular lesions.

Lesions developing as a result of enlarged caseating glands are to a great extent due to the anatomical relationship of the hilar and paratracheal glands to (1) the bronchi, (2) the pulmonary veins and arteries and (3) the thoracic duct.

Effect on the Bronchi.

Simple pressure of the enlarged gland on a neighbouring bronchus may cause narrowing or complete occlusion of the bronchial lumen and in young children the relatively soft and yielding bronchial wall is specially liable to be affected in this way. Clinical evidence of pressure on a bronchus depends on the size of the bronchus involved and on the degree of narrowing of the lumen. Small areas of collapse, only to be detected by radiography, will result from occlusion of smaller bronchi. Blocking of a main bronchus will give rise to the characteristic signs produced by collapse of a pulmonary lobe. It should be noted, however, that with collapse due to hilar or paratracheal adenitis the mediastinum may remain fixed and not show the usual movement towards the side of the collapse. In this case compensatory emphysema of the remaining lung tissue on that side will be found and a rise of the diaphragm. Partial obstruction of a bronchus is often associated with spasmodic cough resembling whooping cough and with "wheezing" or "whistling" respiration.

Collapse of lung may remain for a few days only or for months and occlusions of long standing may produce permanent changes with bronchial dilatation distal to the block. It is not uncommon to find transient collapse of one lobe followed by collapse of another lobe on the same or opposite side as the different groups of lymph glands at the lung roots become involved.

Caseating glands may ulcerate through the wall of a bronchus and thus produce a block of the lumen. Bronchoscopy will show the mass in the bronchus and its nature can be proved by biopsy. The presence of tuberculous material in the bronchial lumen is more likely to produce serious consequences such as inhalation of tuberculous material into the lung distal to the lesion with resulting tuberculous broncho-pneumonia or dissemination throughout both lungs. As the result of ulceration of the wall, bronchial stenosis may become permanent and established bronchiectasis is more likely to follow this type of lesion than occlusion due to pressure alone.

Effect on pulmonary vessels.

Active tuberculous lesions in the lymph glands at the hilum may spread not only to the bronchial walls but to neighbouring blood vessels. Post-mortem findings in children who have died of miliary tuberculosis not infrequently show involvement of the walls of pulmonary veins or arteries by ulceration spreading directly from a caseating tuberculous gland with a consequent acute and fatal dissemination of the disease. A clear picture of this catastrophe is given by H. C. Cameron and S. de Navasquez (6) in their description of the autopsy of such a case. Although this is the cause of miliary dissemination in many cases, it is not the only, or possibly the most frequent cause of a haematogenous spread. This may also occur when tubercle bacilli pass from the hilum glands to the thoracic duct and so are discharged into the blood stream.

Spread by the Thoracic Duct.

When the bacilli taking part in the primary invasion are numerous, it is probable that the lung parenchyma and the lymph glands draining the point of entry are unable to deal with all

the bacilli and a certain number pass along the lymphatic channels and reach the thoracic duct and so pass directly into the circulation. These will enter the pulmonary circulation and return to the lungs in the capillaries where presumably some organisms will remain. Others will be returned to the heart and enter the systemic circulation and be detained in the kidneys, bones, joints, etc. forming possible nuclei for tuberculous lesions at a later date.

Careful study of good X-ray films taken during the reaction to the primary infection when there is enlargement of pulmonary glands will sometimes show diffuse mottling throughout the lungs, suggesting miliary lesions and further pictures taken a year or more later may confirm the presence of miliary lesions by showing scattered small calcified foci in the lungs or in the spleen and liver. As E. A. M. Hall (7) points out the bacilli reach their destination in the lungs during the primary phase of infection before allergy is established and so produce no serious disturbance. The possible fate of these lesions and of quiescent lesions in the lymphatic system has been discussed recently by R. C. Wingfield (8). He stresses the probability that these dormant lesions are stimulated to reactivity by break-down products of tubercle bacilli inhaled in later life, when the individual has become tuberculin sensitive, and that from these reactivated lesions develop the typical pulmonary lesions of adults.

The fact that dormant lesions in lymph glands or in the lungs may play such an important part in adolescent and adult life emphasises the necessity for providing with adequate treatment children who have been subjected to an initial infection of such a degree that it is unlikely to heal completely.

Discussion.

From this description of primary tuberculous infection it will be seen that, apart from the possibility of an immediate fatal result, more remote consequences may arise. Most important among these are (1) the possibility of direct spread from the primary lesion giving rise to pulmonary tuberculosis of the adult type, (2) bronchial obstruction which gives rise to atelectasis and bronchiectasis, (3) the sowing of lung and lymphatic tissue with scattered lesions which become quiescent but do not heal, and which remain as potential sources of trouble when the child reaches adult life and is subjected to further infection or to conditions which lower usual resistance to endogenous spread of infection.

These are all serious conditions and justify a claim for proper treatment of the early acute phase. There is a tendency at present to do no more than keep under observation even those children who have gross glandular lesions on the assumption that they will heal themselves. So they do in many cases, but is that healing as rapid or as complete as it could be? The rate of healing of the primary complex is, of course, partly dependent on the amount of the original infection but is also influenced by the environmental conditions and general health of the child. Among children attending the Childrens' Contact Department at the Brompton Hospital, it is observed that those in poor circumstances may take over a year before calcium is deposited in the infected glands whereas those who have had proper care develop calcified lesions earlier and the maximum deposition of calcium is reached more quickly.

The importance of environment has been recognised for many years in cases of tuberculosis of abdominal and cervical glands and it does not seem unreasonable to demand similar care and treatment for tuberculosis of the glands of the hilum. When infection has been severe and where the necessary conditions are not obtainable in the child's home then, as with tuberculosis of abdominal glands, the child should be treated in a suitable institution where every facility is available for improving the general condition of the child and where there is no danger of continued infection from cases of open tuberculosis.

REFERENCES

- (1) LLOYD, W. E., and DOW, D. J. (1932), *Brompton Hosp. Rep.*, **1**, 51.
- (2) MACPHERSON, A. M. C. (1939), *Brompton Hosp. Rep.*, **8**, 30.
- (3) SWEANY, H. C. (1939), *Acta Tuberc. Scandinavia*, **13**, 67.
- (4) ELIASBERG, H., and NEULAND, W. (1920), *Jahrbuch für Kinderheilk.*, **93**, 88.
- (5) REICHLE, H. S. (1933), *Amer. J. Dis. Child.*, **45**, 307.
- (6) CAMERON, H. C., and DE NAVASQUEZ, S. (1936), *Guy's Hosp. Rep.*, **86**, 366.
- (7) HALL, G. A. M. (1941), *Brit. J. Tuberc.*, **35**, 133.
- (8) WINGFIELD, (R. C.) (1942), *Brit. Med. J.*, **1**, 637.