NON-TUBERCULOUS PLASTIC PLEURISY IN CHILDREN.

BY

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Spontaneous pleurisy occurring with or without an accompanying effusion in adults or in children, has always been considered to be more or less pathognomonic of active tuberculous disease. This communication presents two cases of proved non-tuberculous dry pleurisy in children.

Case 1. F. N., aged 6 years, was admitted to the East London Hospital for Children on February 5th, 1929, and was discharged on February 28th, 1929. She woke on the morning of the day of admission, crying out with pain in her left chest which was worse on deep inspiration. She vomited once. There was no cough. The bowels were open normally. The appetite had been good and there was no lassitude. There were no rigors and no delirium.

Past history. A full term, normal child; breast fed. She had had cervical abscesses at twelve months, diphtheria at thirteen months, pertussis and measles at three years, and bronchopneumonia on three different occasions.

Family history. Mother and father alive and well. Six other children alive and well. One, by the first wife, died of pulmonary tuberculosis.

On admission. Weight, 43½ lb. Temperature 101°, pulse 120, and respirations 30. The child was well nourished and of a good colour. No herpes and no respiratory difficulty. Two healed scars were visible in the neck, where submaxillary abscesses had been incised. The tongue was furred. The tonsils were normal. Some lymphatic glands in the neck were palpable.

The chest examination on inspection and palpation was negative. The percussion note was normal over both lungs. On auscultation, a definite friction rub was heard over the left anterior chest and in the left axilla. A few fine râles were audible. There was no tubular breathing. The right chest, heart and abdomen were normal.

Course. Temperature, pulse and respiration fell to normal within two days. The rub disappeared in one week. She gained weight to 44½ lb. on discharge. Throat and nose swabs were negative for diphtheria. Two test with old tuberculin (0.1 mgrm.) intradermally were definitely negative. X-ray of the chest was negative, showing no increased hilum shadows, calcified glands or other evidence of tuberculous infection. Blood examination showed leucocytes 9,800 (polymorphonuclears, 65 per cent., leucocytes, 35 per cent.). The urine was negative. Seen in the out-patient department on April 15th, 1929, her weight was 49 lb. There was no further pain or cough. The appetite was good and the child was vigorous and healthy. She was again seen on June 21st, 1929. There were no symptoms. The weight was constant. Chest examination was negative. 1 mgrm. intradermally of old tuberculin gave a negative reaction.

Case 2. J. D., aged 7 years, entered the Hospital on March 14th, 1929, and was discharged on April 9th, 1929. He had had a slight cough for two weeks with the sudden onset of pain in the right lower chest two days before admission. This was worse on coughing and on deep inspiration. There was no vomiting. The bowels were open normally. Fever was present one day before admission. There had been no rigors and no delirium.

Past history. A full term, normal child; breast fed. Weight at birth was 7 lb. He had had pertussis at one month and measles at three years. There had been no lassitude or chronic cough. He played normally and with plenty of vigour.

Family history. The mother and father and five other children were alive and well. There was no history of tuberculosis in the family.

On admission. Weight was 48½ lb. Temperature 101.5°, pulse 130, and respirations 40. The child was well nourished and had a good colour. There was no herpes and no respiratory

difficulty. The tongue was clean. The tonsils were somewhat reddened and slight hypertrophic. The lymphatic glands in the neck were palpable. Inspection and palpation of the chest showed nothing abnormal. The percussion note was resonant throughout. On auscultation, there was an inconstant friction rub heard over the right lower anterior chest. The breath sounds were slightly diminished over this area. There were no râles or tubular breathing. The heart and abdomen were normal.

Course. The friction rub became very definite over the right lower chest, with the appearance of a few râles and rhonchi, on the fourth or fifth day following admission. The fever of 101° for one day had dropped to 99.5° on the third day and was normal thereafter. The pulse and respirations also reached normal in three days. The rub had disappeared ten days after admission. The throat and nose were negative for diphtheria. 0.1 mgrm. of old tuberculin intradermally was negative, as was also 1 mgrm. X-ray of the chest was negative. Blood examination showed 10,000 leucocytes (59 per cent. polymorphonuclears; 33 per cent. lymphocytes; 2 per cent. transitionals; 2 per cent. eosinophiles; and 4 per cent. mononuclears). The urine was negative. The child gained 1 lb. in weight while in the hospital and made an uneventful recovery after having been treated only symptomatically, as was also done in the first case.

DISCUSSION.

The pleura may become infected via the blood, lymph, or by direct extension. The ordinary dry, or plastic form of pleurisy may arise from various causes and be a part in the course of several diseases. The most typical form occurs in lobar pneumonia and Holt and Howland¹ in observing 398 hospital cases of pneumonia, noted 27 or 6.8 per cent. which could be classed as pleuropneumonia, the diagnosis being confirmed at autopsy or operation. Acute rheumatism in childhood may be complicated with pleurisy in 4 per cent. of the cases. Various forms of trauma to the chest wall resulting in fractured ribs may produce a local pleurisy, but many accidents to the chest wall can occur without any pleurisy resulting. Dry pleurisy in children is sometimes manifested in the specific fevers, especially typhoid, measles, and scarlet fever; occasionally it complicates a nephritis or a pyogenic infection; finally it may result as the extension of an infectious process involving the chest wall, pericardium, lung, spine, or diaphragm. Extension from the latter two points is usually considered to be tuberculous in origin.

Tuberculosis of the pleura is generally considered as not primary, but secondary to tuberculosis in the lungs or hilum lymph nodes (Miller²). The role of a tuberculous process in the causation of plastic pleurisy is summed up by Carmichael³ when he states that it is practically agreed that a dry pleurisy, in the absence of disease in adjacent tissue, or specific fevers, is due to a tuberculous infection to the same extent that a pleurisy with effusion is considered tuberculous. Robert C. Patterson⁴ thought tuberculosis to be so frequently the ætiological factor concerned, that it was safe to consider every pleurisy as tuberculous unless there was some other definite cause. Roughly, pleurisies with effusion are considered as tuberculous in 50 to 75 per cent. of cases. Primary pleural effusions due to the pneumococcus are said to occur in children without the co-incidence of the usual accompanying pneumonia⁵. Nobel⁶ in his observations at the University Children's Hospital in Vienna on 78 cases of pleuritis in children, concluded that almost all of the cases, with the exception of a few in which a definitely different ætiology could be demonstrated, were of a tuberculous origin. However, he regarded the prognosis as very favourable,

According to Acuna, Casaubon, and Macera⁶ dry pleurisy occurring in smaller children and infants is rarely of a tuberculous nature but is usually pneumococcal as would be expected from the high incidence of pneumonia in infancy. They also considered influenza as a common cause in infants. But Levin⁷ diagnosed pleurisy with a rub in only one case out of 293 cases of influenzal pneumonia observed during the 1918-19 wave of influenza in the United States. He noted fifteen cases of pleurisy in influenza uncomplicated by a pneumonia, or a total of 3.7 per cent. of the non-pneumonic cases. In the 1920 epidemic, he observed no cases of pleuritis except in those cases in which an influenzal pneumonia existed, a total of seven out of sixty-one cases. The higher incidence of evident pleurisy in the 1920 epidemic was in his opinion due to the fact that there was more fibrin in the pleuræ in the 1920 cases than in the 1918 epidemic. Talbot⁸ reported 31 cases of influenza in children in 1918 with no mention of a pleuritis. Howard⁹ also reported 70 cases of influenza in private practice in 1918 with no evidence of a pleurisy in any of them.

Signs and Symptoms. Only very brief mention will be made of the signs and symptoms of plastic pleurisy since they are generally familiar. Usually the onset is an abrupt one with pain in some part of the chest. It is most commonly noticed about the costal margin if the pleurisy is pneumococcal, a result of the more frequent basal position of pneumonia. Tuberculous cases on the other hand, tend to be more commonly apical in type. The pain is definitely made worse by coughing or deep respiratory excursions. Dry cough is present at the onset and usually a fever of 101° to 102° with some prostration. The rub is probably more often absent than present, and is certainly often very transient in character. Respiratory excursions are apt to be short and shallow, *i.e.*, guarded in character. The duration of the fever, the rub and other symptoms depends upon the underlying ætiological factor. In pneumococcal cases with a coincident pneumonia, symptoms may exist from two days to two months, the shorter period being more common.

In the typical case of dry pleurisy where the pleura is alone affected, one naturally expects to find little or no evidence clinically except fever, the presence of pain in the chest which is aggravated by deep breathing, and perhaps a friction rub. However, in so-called pleuro-pneumonia in which more of the parenchyma of the lung is affected, the physical signs of a pneumonia will be in evidence, *i.e.*, dullness, tubular breathing, diminished air entry; in addition a leucocytosis is practically invariable to an extent of more than 15,000 in primary croupous or lobar pneumonia, in which pleurisy as a complication is much more likely to be found.

The signs and symptoms to be expected in a case of tuberculous origin are similar to the above, but there is usually a history of lassitude, anorexia, and variation in or diminution of weight in the child. A family history of tuberculosis may be obtained. Fishberg³ has stressed the diagnostic value of an enlarged gland at the inner end of the clavicle, under the sternomastoid, in cases of tuberculous pleurisy.

Differential Diagnosis. It is necessary to bear in mind several possibilities when confronted with a child presenting the signs and symptoms of an acute

dry pleurisy. Pneumonia and tuberculosis certainly stand in the foreground. Since it is reckoned that a tuberculous pleurisy is usually secondary to pulmonary and secondary hilum glandular involvement, an X-ray examination of the chest for hilum adenopathy, calcified foci, or parenchymal changes, is likely to reveal a lesion. A negative tuberculin test consisting of the intradermal injection of 0.1 c.cm. followed by 1.0 c.cm. of one in one thousand old tuberculin, can be taken as proof of the non-tuberculous nature of the pleurisy. A strongly positive reaction can be taken as fairly conclusive proof of its tuberculous nature in the absence of any other definite cause such as pneumonia. A negative family history and a negative history in the child as regards lassitude and inconstant weight, all weigh heavily against tuberculosis as the cause.

In the pneumococcal cases, there are usually enough physical signs in the chest to warrant the diagnosis of pneumonia, but it is of course possible that the pneumococcus may be the real cause even in cases which show no clinical signs of pneumonia, since it is known that the pneumococcus may cause a pleurisy with effusion in children without any demonstrable lung signs. In the first class of case the pleurisy is secondary to the underlying lung lesion, in the second the condition can be regarded as a primary pneumococcal pleurisy.

The location of the pleurisy may give some aid, those of a tuberculous nature usually occurring in the apices or interlobar spaces while the pneumococcal forms are usually basal. The presence of tuberculous bone or glandular disease would be suggestive. With no evidence of tuberculosis or pneumonia, it must be borne in mind that other conditions may cause pain in a similar region of the body, such as herpes zoster, pleurodynia, intercostal neuralgia, rheumatism, and diseases of the chest or spine. A careful history and physical examination will serve to distinguish these conditions from a dry pleurisy.

In the cases reported above the repeated negative tuberculin tests, the negative X-ray examination of the chest, the negative past history in the child with the negative family history, the absence of lassitude or loss of weight ; but rather a rapid gain in weight associated with freedom from any symptoms whatever, all serve to rule out tuberculosis. Again, the absence of physical signs in the lungs (tubular breathing, dullness, crepitant râles, and flattened percussion note), the absence of leucocytosis, the negative X-ray evidence of consolidation, absence of herpes and of the typical onset of a pneumonia, the length of time during which the friction rub persisted after the fever had gone, and the very rapid return of the temperature pulse and respiration to normal, combine to exclude pneumonia as a cause. There was no evidence of rheumatism, disease of the spine, or of any of the specific fevers. If judgment were based upon the commonly accepted dictum that any pleurisy occurring in the absence of disease of adjacent tissues or in the absence of specific fevers, is a tuberculous one, then cases here presented would certainly be considered as tuberculous ; but the clinical observations and the X-ray and tuberculin tests do not allow of such a diagnosis ; and it is therefore definitely suggested that they are non-tuberculous.

Since the influenza epidemic was occurring at the time these cases were observed, it is possible that this may have been the ætiological factor concerned,

CONCLUSIONS.

1. Two cases of acute dry pleurisy in children are reported.
2. Evidence is presented against their being of a tuberculous origin.
3. In the absence of any accompanying pneumonia, influenza is suggested as a possible ætiological factor.

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