PULMONARY LESIONS IN RHEUMATISM

BY

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Many writers include among the reactions to the rheumatic infection inflammation of the lung and pleura. In most reputable text-books of medicine it is stated that the rheumatic infection plays a part in the ætiology of idiopathic pleurisy. Paul¹ in a recent review of the question of the pulmonary lesions in rheumatism states that pleurisy is present in 10 to 15 per cent. of all examples of rheumatism and that it may be either primary or secondary. He quotes Rolly as saying that it is chiefly associated with pericarditis and usually situated on the left side. This association with pericarditis and localization on the left side are mentioned by most writers and are quite in accordance with my own experience. Paul states that he found it present 4 times in 30 post-mortem examinations on cases without pericarditis, but I suppose, though he does not say so, there was almost certainly some form of carditis as rheumatic patients do not usually die from any other manifestation of the disease.

Pleurisy as a primary manifestation of rheumatism I personally have never encountered, or at any rate recognized as such, but in association with carditis, and especially pericarditis, pleurisy, or at least a pleural effusion, is not infrequent. When we consider the close proximity of the serous layers of the pleura and pericardium, it is indeed remarkable that an extension from the pericardium to the pleura does not occur frequently. This immunity of the pleura under these conditions is inexplicable and can only be quoted as an example of the well known selective action of infective agents.

In my experience a true inflammation of the pleura even as a secondary lesion is of the rarest occurrence. It sounds almost a platitude to say that the presence of fluid in the pleural sac is not evidence of pleurisy, and yet this would seem to be at times forgotten. In a failing heart from whatever cause, and rheumatic carditis is one of the most frequent, pleural effusion from a passive exudate is not unexpected, and in the rheumatic pleural effusions the French writers (Revaut², Bezancon and Weil³) all lay great stress on the presence of desquamated endothelial cells, either singly or in plaques, which is the characteristic cellular feature of a transudate.

Within recent years it has been suggested, but in our opinion without sufficient justification, that a special type of pneumonia is met with in rheumatism. Both Naish⁴ and Fraser⁵ have recorded the post-mortem appearances and the histological features of a pulmonary lesion which they

claim to be truly rheumatic in nature. In all the patients there was severe cardiac disease. The mischief was usually very widespread, in fact it involved almost the whole of both lungs, and yet Naish suggests that it may explain the limited area of dullness at the left base so frequently encountered in pericarditis. Histologically they describe hyperplasia of the alveolar walls to which McNeil has recently drawn attention as being characteristic of broncho-pneumonia, and in places cellular conformations simulating Aschoff's bodies.

In all I have records of 25 cases of the rheumatic infection in which there was a pulmonary complication. In this review I omit, of course, consideration of cases in which there was obtained a history of pleurisy or pneumonia some considerable time before the onset of chorea or arthritis, since it does not seem reasonable to connect in any way these infections with one another. I am quite alive to the fact that there is a general belief that idiopathic pleurisy may be rheumatic in nature, but the extreme rarity with which definite rheumatic manifestations develop after the appearance of a pleurisy to my mind makes such a contention untenable. In only one or two instances out of several hundred cases of carditis were the cardiac symptoms stated to date from an attack of pleurisy, and as the history was somewhat indefinite I always entertained a doubt whether the original illness—a so-called pleurisy—was not in reality pericarditis. Pleurisy is so frequently diagnosed on the ground of pain in the side, which we know is not infrequent in pericarditis as well as in endocarditis, that in the absence of definite facts relating to the illness, there is always room for doubt. I may be permitted to mention here that while other manifestations of rheumatism are most frequent during childhood and adolescence, pleurisy with effusion occurs more commonly after 20 years of age than before it.

In all the cases reviewed in this communication there was at the time of the pulmonary lesion definite evidence of a rheumatic carditis. Without carditis I have not seen a pulmonary complication unless as above mentioned. I am not unmindful of the fact that subcutaneous nodules usually, if not invariably, go with carditis.

Pulmonary lesions complicating pericarditis.

In 16 of the cases the cardiac lesion was pericarditis with effusion, and as I have records of a total of 52 examples of pericarditis, pulmonary complications were present in practically 30 per cent. In 9 of these 16 cases the condition was one of effusion into the pleural sac as verified by exploratory puncture. In 3 other children I am inclined to believe that the condition was also one of pleural effusion, but at the time these cases came under observation I was not sufficiently alive to the possibility of fluid and interpreted the physical signs as evidence of consolidation or collapse of the lung. In 9 cases the effusion was at the left base and in 3 at the right base. In one case the history, physical signs and X-ray examination pointed to an intercurrent lobar pneumonia: this child was admitted with pericarditis which by one week had subsided, when fever returned with dullness to percussion and friction at the right base

and later tubular breathing. In 2 cases there was impairment of the percussion note at both bases with some moist râles which I ascribed to passive congestion: in one of these the diagnosis was verified at the post-mortem examination. In another case of pericarditis a terminal broncho-pneumonia of the left lower lobe and of the left apex was also verified at the autopsy.

The striking feature, then, about these cases of pericarditis is that the most frequent pulmonary complication was effusion into the pleural sac, and be it noted the left pleural sac. Previously I had accepted the view that dullness at the left base with deficient and perhaps tubular breathing in pericardial effusion was, as Ewart⁶ had suggested, due to collapse of the lung from compression, but at present I am of opinion that a pleural effusion is the more frequent cause of this physical sign. Experience of the physical examination of the chest in the child has taught me that with moderate amounts of fluid in the pleural sac the signs may simulate very closely those met with in consolidation of the lung. The dullness to percussion is not extreme, there is no evidence of displacement of organs, e.g., by a positive Grocco's sign, the respiratory murmur may be exquisitely tubular in character and at the same time accompanied by râles, due probably to hypostatic congestion of the subjacent lung. In fact, the only way in which to decide definitely, even in the presence of an X-ray picture, is, as I was taught by my friend and teacher—the late Dr. Geo. S. Middleton—exploratory puncture. The not infrequent experience of finding an empyema in the post-mortem room, though it had not been suspected during life, is in support of the wisdom of the above dictum.

At first it seemed possible, since the effusion was usually situated on the left side, that the involvement of the pleura was consequent on an extension of the pericardial inflammation just as it is in left-sided pneumonia that pericarditis is more likely to develop. This seems to be the assumption of many writers on the subject. But that this is not so is definitely shown by the fact that the effusion is of the nature of a transudate and not of the nature of an exudate. Invariably the cellular elements which dominated the picture were endothelial in nature, arranged either singly or in plaques, which we know to be characteristic of passive pleural exudates as met in conditions of failing heart. This feature of pleural effusion in pericarditis has already been remarked upon by the French writers (Revaut, and Bezançon and Weil).

It might be suggested that just as the rheumatic virus exerts a selective action on certain tissues so also might it present a peculiar type of reaction. But when the pericardium is involved the cellular elements in the exudate are almost entirely polymorphonuclear in type, as is also the case in affected joints. I was fortunate in obtaining from the same case both pericardial and pleural exudates, and a more perfect contrast or more typical pictures of what usually obtains could not be found. The three accompanying micro-photographs show: (Fig. 1) the cellular reaction in rheumatic pericarditis; (Fig. 2) the cellular deposit from pleural effusion complicating pericarditis (1 and 2 are from the same case and were removed at the same time); and (Fig. 3) the cellular reaction in rheumatic arthritis.

FIG. 1. Film from rheumatic pericardial exudate showing chiefly polymorphonuclear cells: from same case as Fig. 2.

FIG. 2. Film from pleural exudate in case of pericarditis showing plaques of desquamated endothelium; from same case as Fig. 1.

FIG. 3. Film from rheumatic arthritic effusion showing chiefly polymorphonuclear cells.

Pulmonary lesions complicating endocarditis.

In eight of the cases there was endocarditis. This proportion of pulmonary complications in endocarditis is much less than would be met with in material compiled from an adult population in which a failing heart with passive pulmonary congestion and a passive pleural exudate are not uncommon. When one considers the frequency with which endocarditis, and severe endocarditis of rheumatic origin, is met during childhood it is remarkable how rarely the condition of failing heart is encountered. To my mind this speaks against the usual teaching that in all examples of active rheumatism the myocardium participates. To anyone who has had much experience of the after-care of rheumatic children, the good cardiac efficiency that prevails over long periods of years in the presence of hypertrophy of the heart and a severe valvular flaw must have seemed remarkable. There is little evidence too of such implication from electro-cardiographic records taken during the acute phase of the disease. One must not, of course, forget that electro-cardiographic tracings reveal rather flaws in the conducting system than in the contracting system.

These facts would seem to suggest that during childhood at least the acute phase of rheumatism does not as a rule tell severely on the myocardium. It is not suggested that the myocardium does not suffer, and at times suffer severely, during the acute phase of the disease, but when this does happen a speedy fatal issue is not infrequent. Perhaps the most striking feature of rheumatism during childhood is the large proportion of the deaths which occur within a year of the onset of the mischief. ..

In three of the cases of rheumatic endocarditis there was, just as in the examples of pericarditis, a left-sided pleural effusion; in two this was shown clearly by X-ray examination, though the dullness and tubular breathing might have suggested consolidation; in the other there was general anasarca, and exploration revealed a thin layer of blood-stained serous fluid showing a centrifugalized sediment composed of lymphocytes and desquamated endothelial cells. Three children, the subjects of rheumatic endocarditis, were admitted with intercurrent pneumonia, croupous in two instances and catarrhal in one. I have already remarked on the comparative rarity of this occurrence for which it is difficult to offer any explanation unless it be that the concomitant passive congestion which is inimical to tuberculous infection is also antagonistic to the development of a lobar pneumonia. The remaining two patients were examples of mitral stenosis and regurgitation who died from a terminal broncho-pneumonia, in one of which there was in addition empyema.

The only other feature in the above series of cases which calls for special comment is the fact that the pleural effusion has in the majority of cases been situated on the left side; in only 3 of the 15 cases was it present on the right side. Most authors state that the right is the side on which a passive exudate in a failing heart is found, though Price⁷ says that in his experience both pleural sacs are usually involved.

Conclusions.

1. The rheumatic poison shows no predilection for the pleura or the lung
2. The most frequent pulmonary complication in rheumatism in childhood is pleural effusion.
3. This occurs most frequently with pericarditis.
4. This pleural effusion is of the nature of a transudate.
5. The pleural effusion occurs most frequently at the left base and should always be suspected when the percussion note is impaired at the left base, even if the respiratory murmur is intensely tubular in character and râles are present.
6. Pulmonary lesions in rheumatism are of the nature of
 - (a) an intercurrent disease (lobar pneumonia), or
 - (b) a terminal hypostatic or broncho-pneumonia complicating cardiac disease.

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