MYOCARDITIS IN ACUTE INFECTIVE DISEASES A REVIEW OF 200 CASES

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The acute infective diseases constitute the most important cause of myocarditis, the commonest heart disease in childhood. Increasing amount of evidence from electrocardiographic investigations of the heart in acute infective diseases shows that there can be a myocarditis when clinical signs and symptoms are slight, doubtful or completely absent. These investigations further revealed that many convalescent cases whose unsatisfactory condition was accounted for by post-infective or secondary anaemia were actually suffering from myocarditis. Therefore, since this involvement of the myocardium is so common an event and liable to be missed or misdiagnosed, it seems justifiable to give an account of 200 cases of myocarditis occuring in acute infectious diseases.

General signs. The children do not complain of any pains; sometimes there is abdominal discomfort. They do not resent examination and are not irritable.

Clinical signs. There is always some pallor and listlessness. Not infrequently vomiting occurs and may be the first sign. The urine often shows albuminuria, varying from the presence of a trace to a heavy deposit. The principal clinical signs are diminution in intensity of the first sound at the apex, indicating a weakness of the myocardium. The first sound becomes equal in intensity to the second apical sound, later on it becomes weaker and may even become entirely inaudible. Changes in the character of the cardiac rhythm take place, as shown by persistent tachycardia, less frequently bradycardia, embryocardia or gallop rhythm. Some degree of cardiac enlargement is common in severe cases of myocarditis. An important sign is a low blood pressure, the diastolic reading being often extremely low. The electrocardiogram is an important aid in the diagnosis of a myocarditis-The first sign is a flattened T wave; later on the T wave becomes isoelectric and eventually inverted. A second sign of myocarditis is an S-T segment below the isoelectric line in lead I and II or in both. It is conclusive evidence of a myocarditis when found together with a change in the T wave. A third sign of myocarditis is a diminution of voltage of the ventricular deflections to less than 1.5 mV

voltage in all three limb leads together. This sign occurs especially in cases of severe myocarditis.

Pathological anatomy. Whenever possible the heart of a fatal case was examined in the Pathological Department (Prof. B. Shaw) of King's College, Newcastle upon Tyne. Two illustrative cases are given :

1. Sheila F., ten years, died on the eleventh day of diphtheria. Immediately beneath the ventricular endocardium and also in the inner third of the wall are some scattered small foci of lymphoid and histiocytic cells. These foci sometimes occur in association with shrunken muscle fibres and what appear to be small delicate recent scars.

2. Iris N., eight years old, died on the fourth day of diphtheria. Beneath the endocardium of the left ventricle there are a few patches of interstitial myocarditis in which the muscle fibres are disappearing and there is accumulation of mobile histiocytes and lymphocytes.

In the present series of 200 cases there are :

- 122 cases of diphtheria;
- 10 cases of diphtheria in inoculated children :
- 40 cases of scarlet fever ;
- 24 cases of whooping cough ; and

4 cases of measles.

The diagnosis of myocarditis was made on clinical grounds in 55 per cent. of the present series, by means of an electrocardiogram with doubtful clinical signs in 20 per cent. and on electrocardiographic findings with no clinical signs present in 24 per cent. The duration of the myocarditis in the present series was two to three weeks in 17 per cent., five to nine weeks in 58 per cent., three to four months in 25 per cent. The termination of the myocarditis was ascertained clinically in 13 per cent., clinically and by means of an electrocardiogram in 12 per cent., and by means of a graphic record alone in 75 per cent., when the clinical signs had subsided or they had not been present at all.

Frequency of clinical signs (percentage)

| Vomiting | •• | •• | •• | | 23 |
|--------------|----------|-------|--------|----|----|
| Albuminuria | | •• | •• | | 38 |
| Enlargement | of cardi | ac du | llness | •• | 21 |
| Heart sounds | | | | | |
| 1=2nd apie | | | •• | •• | 15 |
| 2>1st apica | il sound | l | •• | | 29 |

Rhythm :

c m

| Persistent tachyca | ırdia | | | 36 |
|--------------------|---------|---------|----|----|
| Bradycardia | •• | •• | | 17 |
| Embryocardia (ti | c-tac r | hythm) | | 9 |
| Gallop rhythm | | • • • • | | 6 |
| Extrasytoles | | | •• | 16 |

Blood pressure (mm. Hg)

| Age | Normal | | | Lowest readings in present series | | |
|----------|----------|-----------|----------|--------------------------------------|--|--|
| in years | systolic | diastolic | systolic | diastolic | | |
| 3 | 90-100 | 70 | 48 | 30 | | |
| 4 | 100-115 | 70 | 40 | 30 | | |
| 56 | 110-120 | 75-80 | 40 | 20 | | |
| 7–10 | 115-120 | 8090 | 62 | 24 | | |
| 12–17 | 120-130 | 90 | 70 | 38 | | |

Frequency of electrocardiographic signs. (Percentage) LIMB-LEADS

LIMB-LEADS

| Changes of I wave : | | | | | |
|--------------------------------------|------|----------|-------|------|----|
| T of less than 0.1 mV voltage, round | | | | 47) | |
| T isoelectric | •• | •• | | 18 > | 54 |
| T inverted | | | | 6) | |
| S-T segment below the base line | | | 27.5 | | |
| QRS of less than 1.5 | mV v | oltage i | n all | | |
| three leads | | •• | | 38 | |
| Extrasystoles | | | | 12 | |
| Sinus tachycardia | | | | 32 | |
| | | | | | |

CHEST-LEADS

| QRS inverted | 64 | |
|---------------------------------|----------|----|
| Voltage of QRS less than 0.8 mV | 80 | |
| S-T segment depressed | 17 | |
| S-T segment below the base line | 6 | |
| Changes of the T wave: | | |
| T of less than 0.1 mV voltage | ן 17 | |
| T inverted | 18 (| 86 |
| T isoelectric | 36 (| 00 |
| T diphasic | 5) | |

The changes of the T wave in the chest-leads enumerated above cannot be interpreted as a myocarditis if not combined with other alterations because they often occur in healthy infants and young children.

Once a myocarditis has been present there is the danger of its recurring in the course of another infective disease. Each of these subsequent attacks may cause further injury to the heart and may prove fatal. Actually it was found that in the majority of the 200 cases the patient had one, two or more acute infective diseases within the last year and in some instances up to five acute diseases within the last three years.

Incidence of preceding infective diseases in 200 cases of acute myocarditis (percentage).

| Measles | | 25 |
|----------------------------|----|----|
| Measles and whooping cough | | 22 |
| Measles and chickenpox | | 9 |
| Whooping cough | | 7 |
| Scarlet fever | •• | 6 |
| Lobar and bronchopneumonia | •• | 15 |

Death occurred in 4 per cent. of the present series. It is noteworthy that the fatal event occurred in two cases of diphtheria, where the history reported four infective diseases within three years (whooping cough, chickenpox, measles, scarlet fever) in one case and three diseases in another case.

Diphtheria

It would be superfluous to discuss in detail here the myocarditis in diphtheria. Vomiting, changes in the quality of the first apical sound, changes in the heart rhythm, and low blood pressure are clinically the most suggestive evidence of a myocarditis. Severe albuminuria early in the disease has always been regarded as a signum mali ominis. In severe cases not only the myocardium but also the conducting tissue is involved resulting in partial or complete heart block or intraventricular block. In the present series, however, only cases of pure myocarditis are represented. In 7 per cent. of the cases there was a slightly prolonged P-R interval. It is important to underline the occurrence of myocarditis in diphtheria in inoculated children. Severe complications are rare in diphtheria in the inoculated, but myocarditis does occur. General signs and symptoms, and clinical findings are the same as in the non-inoculated but occur in milder degrees and the electrocardiogram gives a good clue to the diagnosis; involvement of the conducting tissue is rare in these cases.

Scarlet fever

Heart complications in scarlet fever occur towards the end of the second week, more frequently during the third week of disease and even later. The first symptom is pallor, the temperature is slightly raised for a few days or is normal. The pallor becomes more intense after a week or two and persists into The patient often loses late convalescence. weight. The cardiac dullness is enlarged, but only in severe cases is the dilatation extensive. The intensity of the first apical sound becomes less than that of the second and accompanying or following this change, a murmur develops. The development of murmurs is common in scarlet fever and it is important to distinguish functional murmurs from those caused by myocarditis or endocarditis or by both. Generally speaking myocarditis is more common than endocarditis in scarlet fever. Hence murmurs which occur in the course of scarlet fever have to be regarded as due to a diseased myocardium in the first place and to an endocarditis in the second place. There are three criteria of a carditis in scarlet fever : (1) The blood sedimentation rate is markedly increased as in rheumatic fever; (2) The electrocardiogram shows definite signs of a myocarditis and signs of a valvular disease, e.g. a mitral stenosis in some instances, later on; (3) the x-ray examination reveals the enlargement of the heart. However, since the clinical signs are varied and poor at the onset, diagnosis might be difficult. The clinical and electrocardiographic signs are similar to those seen in rheumatic fever and last for several weeks or even months. When the myocarditis subsides the murmur disappears and the patient makes so complete a recovery that all evidence of the disease vanishes. Murmurs caused by valvular disease persist in the majority of cases. Permanent cardiac damage was observed in some of the cases recorded here.

Whooping cough

Myocarditis is caused by two factors in whooping cough. One is merely mechanical : it is the strain put upon the heart by the severe paroxysms of cough during the third and fourth week of the disease. The second cause is the toxic effect of the infection on the heart-muscle. It is obvious that myocarditis is more often seen in cases of whooping cough complicated by bronchopneumonia. The pallor, lassitude, and tiredness of these patients is remarkable. The cardiac dullness is enlarged, especially to the right. Tachycardia is persistent and often of extreme degree. Blood pressure is lowered. The electrocardiogram shows right ventricular preponderance besides the typical signs of mvocarditis and sinus-tachycardia. In many instances clinical and electrocardiographic signs might be found eight to twelve weeks after the whooping cough subsided. In cases of severe whooping cough, when there is anaemia or general weakness, listlessness and palpitation on exertion in late convalescence, which cannot be explained otherwise, the possibility of a myocarditis should always be considered.

Measles

Heart disease in measles is rare. Indeed, when a myocarditis is encountered in an uncomplicated case of measles it is suspected to be a flare up of a myocarditis which originated in a preceding infectious disease. However, myocarditis is not uncommon in measles complicated with bronchopneumonia. The cases reported here are such cases. The general pattern of the myocarditis in measles is similar to that in all acute infectious diseases. It makes itself manifest during the febrile stage and subsides after the acute period.

The convalescent child

A pale child with shadowed eyes, cold hands and feet, poor appetite and easily tired on exertion is often presented as the convalescent from an acute infective disease. Examination frequently yields few clinical findings or none at all, and since the blood picture is often one of an hypochromic anaemia, this anaemia is considered to explain the child's condition. It should be remembered, however, that anaemia is more often a sign of a disease than a disease in itself and that the partial examination of such a child—that is one which does include an electrocardiogram—is more not dangerous than no examination at all, because of the grave risk of the child being declared healthy when a myocarditis is actually present. This point cannot be stressed too often, an example of its practical importance is illustrated in a case such as the following :

A child, ten years old, inoculated against diphtheria, contracted 'tonsillitis.' As the child had suffered from tonsillar enlargement on a previous occasion, tonsillectomy was considered advisable. But since it made a slow recovery from its illness, being pale and easily tired, iron and other tonics were given to combat anaemia and strengthen the child. After five weeks of this treatment, the tonsillectomy was undertaken. About 60 minims of chloroform were given, followed by open ether : the pulse failed and the usual measures failed to bring the child round. Post-mortem examination revealed a myocarditis.

The protracted convalescence which may follow whooping cough has been mentioned before. It is interesting to see how an older generation of clinicians attacked this problem : they were fully aware that a child who exhibited the signs described above could not be regarded as healthy and recommended convalescence at the seaside or in the mountains, for several weeks. In this way, the patients did indeed procure the rest so essential as part of their treatment. If complete rest and good nursing be applied at once, the heart may be saved from irreparable damage. Some patients, it is true, do recover undiagnosed and untreated. Nevertheless, it is important that the correct diagnosis be made, because one key to the problem of lowering the incidence of heart disease lies in the early and correct diagnosis and adequate treatment of heartcomplications of acute infective diseases in childhood.

Observations carried out over several years suggest that heart disease in adults might be due to repeated infections during childhood and adolescence, and that the lesions so produced often remain latent, so that clinically manifest heart disease does not appear until months and years afterwards.

Thanks are due to Dr. J. A. Charles, former medical officer of health, Newcastle upon Tyne, and to Dr. E. F. Dawson-Walker, medical superintendent of the Hospital for Infectious Diseases, Walkergate, for permission to carry out the investigation.

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