Postcarditic myocardiopathy

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THE two conditions, myocarditis and cardiomyopathy, under certain circumstances have an intimate relationship to one another, the cardiomyopathy being a direct consequence of a previous myocarditis. It goes without saying that the great majority of cardiomyopathies have no traceable relation to a previous infection nor is there any reason to believe any such association ever existed.

The present communication refers to a small number of instances of cardiomyopathy which appeared to arise from and be in direct continuity with a previous myocarditis, before which the heart was apparently normal.

The concept is not new, having been referred to by many authors who have written on this subject through the years. To find its origin, we have to go back to the thesis of Corvisart (1811) in which he speaks of certain people with a malady of the heart which sometimes was responsible for their death but otherwise allowed them to recover with some degree of disability. Translating this into contemporary terms, one can say that a person with acute myocarditis can take one of three courses, he may die, he may recover completely, or he may survive with

*The term 'myocardiopathy' is philologically preferable to 'cardiomyopathy'.

residual myocardial involvement. That is to say, he may have a cardiomyopathy*.

An infective origin of a cardiomyopathy may easily pass undetected because of its trivial or misleading feature.

A 15-year-old public schoolboy complained of nausea and vomiting immediately after a football match. He was admitted into the school infirmary where he remained slightly pyrexial and spoke of vague malaise until the fifth day when he was found dead in bed. Microscopy showed a profuse cellular invasion through the myocardium, typical of an acute myocarditis. If this patient had survived with permanent myocardial changes, the myocarditis presenting in such an atypical form might well have been overlooked.

In the following instances the infective origin could be more positively identified because the previous medical history and the acute illness were both well documented. The first patient was a 35-year-old Army officer who was admitted to the Middlesex Hospital complaining of pain in the chest, fever and diarrhoea of 7 days duration, beginning while on holiday in Spain. On admission, he had a raised venous pressure, enlarged heart, pericardial rub and a gallop rhythm. There were

FIG. 1. Male 35. (a) On admission, radiological signs of pneumonia in right lung. (b) Pulmonary oedema developed within 4 weeks.

radiological signs of pneumonia in the right lung (Fig. 1a). Within 4 weeks he developed pulmonary oedema (Fig. 1b) which responded to the customary antifailure measures. Apart from a high white cell count and raised sedimentation rate, all the blood studies were normal. The electrocardiogram (ECG) showed frequent ectopics and non-specific ST and T waves in the limb leads and V4 – 6 (Fig. 2). Although he improved symptomatically over the next 6 months, the venous pressure remained elevated and a gallop rhythm and cardiac enlargement persisted. The chest radiographs shown in Fig. 3 were taken 9 years before the illness when the heart was normal in all respects (Fig. 3a) and 8 months after the onset of the illness when the heart was enlarged and the diagnosis was cardiomyopathy (Fig. 3b). Viral studies at the acute phase and later, gave negative results. He died in congestive heart failure a year after the illness began. At necropsy, the heart was enlarged but otherwise normal. Histological studies showed areas of fibrosis, residual cellular infiltration here and there and cellular degeneration. The observation period spanned the entire natural history of this illness from its onset to death. The progression from myocarditis to cardiomyopathy justified the descriptive title of *post-myocarditic myocardiopathy*.

I will now summarize the histories of three patients who bridge the gap between the infection and the cardiomyopathy. The first was an Army officer aged 41 with an uneventful medical history and the subject of regular medical examinations

FIG. 2. Same patient as Fig. 1. ECG showing non-specific ST and T abnormalities in limb leads and V4–6, with frequent ventricular ectopics. Abnormalities still present 11 weeks later (lower tracing).

FIG. 3. Same patient as Fig. 1. (a) Heart size was normal 9 years before myocarditis. (b) 8 months after acute attack, cardiac enlargement persists.

FIG. 4. Male 41. ECGs retain abnormalities after myocarditis over a period of 3 years. Similar changes were present 4 years later.

FIG. 5. Male 26. Cardiac enlargement persisting after acute myocarditis had subsided (a). Before the attack, the heart was normal and similar to that of his identical twin (b).

including an ECG and a chest radiograph. They had always been normal. He now complained of fever, chest pain and breathlessness. A pericardial rub was heard and the venous pressure was raised, a gallop rhythm appeared, the heart was enlarged and the ECG was abnormal. This episode subsided, he lost all his symptoms, but the ECG and chest radiograph have remained abnormal over the subsequent 12 years (Fig. 4) We believe these changes represent myocardial and possibly pericardial residua of an initial myocarditis.

The next patient was a soldier aged 31. He was taking part in battle exercises in Germany when he developed a fever, breathlessness and palpitation. He was admitted to hospital where he spent 3 months, the diagnosis being heart failure of unknown aetiology. The fever quickly subsided, there were no blood changes and no evidence of any bacterial or virus infection. The ECG taken at the commencement of the illness showed non-specific ST and T abnormalities. An ECG taken 9 months later when he complained of breathlessness only on unusual effort, showed frequent ventricular ectopic beats superimposed on a non-specific abnormal pattern.

The third patient was a 26-year-old man, one of identical twins both of whom were star athletes of their infantry unit. One of them developed fever, fatigue and haemoptysis and within a few weeks a gallop rhythm and a loud systolic murmur appeared. The ECG showed flat or inverted T waves and the heart was enlarged (Fig. 5a and b). Similar changes were present 1 year later.

FIG. 6. Female 48. Gross cardiac enlargement discovered one year previously at routine examination when patient was full-time factory worker. Necropsy 1 year later, showed extreme dilatation of all chambers and slight rheumatic mitral and aortic valve disease without pericardial effusion.

All these patients described above were subject to a careful but negative search for a causative virus including blood and faecal studies.

The commonest sequel of acute rheumatic carditis is mitral valve disease with more or less myocardial involvement. Sometimes however, myocardial involvement dominates the clinical picture. The heart is greatly enlarged while signs of valve disease are trivial (Fig. 6). The reasons as to why the initial inflammatory process should affect the myocardium in such a way as to make it excessively compliant, are unknown. Retrospective review of the

clinical features of the acute rheumatic state throws no light on the problem. A cumbersome but more specific description is *rheumatic post-myocarditic myocardiopathy*.

Conclusions

Acute myocarditis may be fatal or resolve completely or incompletely. Incomplete resolution results in a wide spectrum of clinical conditions with the common designation of cardiomyopathy. Some of these may be trivial and recognizable only by minor electrocardiographic or radiological signs. Greater degrees of cardiac enlargement, raised venous pressure, gallop rhythm, cardiac arrhythmias and ST and T abnormalities are found in the more severe types. The myocarditis which is the precursor of these types of cardiomyopathy may present in a variety of clinical forms ranging from a florid infection to a trivial and evanescent illness easily overlooked. Virus studies in the acute stage or subsequently may be unrevealing. Necropsy usually shows non-specific changes, fibrotic replacement or, when survival has been relatively short, collections of cells. The case histories of four patients who developed a cardiomyopathy after an initial infection are presented. In two of them, a clinical diagnosis of myocarditis was possible. An aetiological agent, microbial or viral, was not discovered.

While contending that certain types of cardiomyopathy are the sequelae of myocarditis, it is not suggested that infection is a universal or even a frequent causative factor in the majority of cardiomyopathies.

Reference

CORVISART, J.N. (1811) *Essai sur les maladies et les lésions organiques du coeur et des gros vaisseaux*. Nicolle, Paris.