CARDIAC INVOLVEMENT IN SPIROCHÆTAL JAUNDICE

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There are relatively few published articles on the cardiac complications of spirochætal jaundice. Our purpose is to review briefly the available papers dealing with spirochætal jaundice complicated by cardiac disturbances, and to report a case.

REPORTED CASES

Clinical material. Garnier and Reilly (1916) described a case in which an aortic diastolic murmur and a collapsing pulse appeared in the convalescent stage, and assumed that there was a direct involvement of the aorta by the spirochætal infection, in analogy to syphilitic aortitis: this interpretation, however, is hardly acceptable and it is very unlikely that there was any correlation between the aortic regurgitation and the spirochætal infection. Dawson and Hume (1916-17) were the first to report an instance of a cardiac disorder that arose in the course of spirochætal jaundice and appeared to bear a definite relation to it. This occurred in the form of paroxysmal auricular fibrillation which persisted for five days; it was confirmed by polygraphic tracings. Costa and Troisier (1917) observed four cases in which transient dilatation of the heart appeared without any other cardiac manifestations. Marchal, Soulié, and Roy (1935) reported one with transient cardiographic changes consisting of a slight prolongation of the P-R interval and abnormal T waves, and also with a presystolic gallop rhythm and a low blood pressure: no electrocardiograms were reproduced in the original paper and the patient was having digitalis, which makes the interpretation of these findings rather difficult. Clapper and Myers (1943) published two cases with cardiac complications: in one, there was a delayed auriculo-ventricular conduction which lasted seven days; in the other a pericardial friction rub and auricular fibrillation appeared during the height of the illness, persisting respectively for one and two days.

Pathological material in fatal cases in man and in experimental spirochætosis. Mollaret and Ferroir (1935) reported a case in which the autopsy revealed parenchymatous and interstitial changes in the heart, the nuclei showing swelling and chromatolysis and the interstitial tissue being infiltrated by lymphocytes and polymorphonuclear leucocytes. Beitzke (1916) observed perivascular cellular infiltration in the myocardium. Ashe, Pratt-Thomas, and Kumpe (1941) described a case in which, in the authors' own words, "the myocardium certainly showed severe toxic damage, and although this is common in severe toxæmia, the vacuolization and loss of striation and hyalinization of portions of muscle bundles are suggestive of injury of a type that is rather characteristic of the muscular damage that occurs in Weil's disease." Raun-Byberg (1941) published a case of spirochætosis complicated by fatal myocarditis, but the original paper was not available to us and we are unable to give any details on the nature of the myocardial involvement in this case. Dräger (1934) reported two cases of acute leptospiral vegetative endocarditis in which he was able to demonstrate spirochætes in the vegetations.

Hæmorrhages in the endocardium, myocardium, and pericardium in fatal cases in man and in experimental spirochætosis in guinea-pigs have been observed by several authors. Dawson, Hume, and Bedson (1917) found in inoculated guinea-pigs widespread hæmorrhages in several organs, including the heart, and they were able to demonstrate spirochætcs in the heart muscle. Martin and Pettit (1919) observed minute myocardial hæmorrhages in inoculated guinea-pigs and also mentioned the presence of spirochætcs in the heart muscle. The presence of spirochætcs in the heart muscle in man was stressed by Kaneko (1917). Stokes and Ryle (1916) mention in their paper a fatal case, in the care of Capt. Floor, R.A.M.C., in which the post-mortem examination revealed multiple hæmorrhages in the pericardium. Stokes, Ryle, and Tytler (1917) found hæmorrhages in the heart in two out of four fatal cases; and in inoculated guinea-pigs they found spirochætcs in the heart muscle. Watson, McLeod, and Stewart (1935) observed punctiform hæmorrhages in the pericardium in a fatal case; the heart muscle was soft and flabby. Swan and McKeon (1935) found sub-endocardial and subpericardial hæmorrhages in three out of four fatal cases. Jeghers, Houghton, Rae, and Foley (1935) described hæmorrhages in the pericardium and endocardium in one case. Davidson and Smith (1936) found hæmorrhages in the pericardium in one case and in the endocardium in another; in one of these the myocardium showed fine fatty stippling of the axial portions of the fibres. In a fatal case of Clapper and Myers (1943) the epicardial surface and the endocardial surface over the chordæ tendinæ showed minute hæmorrhages.

CASE REPORT

A seaman, aged 52 years, was admitted to the Newcastle General Hospital on June 24, 1943. His illness started ten days before admission with violent sickness and pain in the legs and the back. Very soon after he became jaundiced. He had fallen into the Thames ten days prior to the onset of symptoms. Except for a syphilitic primary sore in 1918 and malaria in 1926, there was nothing of significance in his previous history.

On admission, the patient looked ill and was deeply jaundiced. The temperature was normal. The lungs were normal, the liver and spleen were not enlarged. The central nervous system appeared to be normal. On physical examination, the heart was found to be perfectly normal, the pulse of good volume and regular, 90 a minute. The urine contained bile and albumin, and the urinary deposit showed some red cells, epithelial cells, and numerous hyaline and occasional granular casts.

FIG. 1.—(A) Auricular fibrillation, 26/6/43. (B) Sinus rhythm with T wave changes, 30/6/43. (C) Normal tracing, 10/7/43.

The blood urea was 175 mg. per 100 c.c. The Wassermann reaction in the blood was negative. The number of white cells was 7400 per c.mm. The diagnosis of spirochætal jaundice was made and this was confirmed by a positive agglutination titer against *Leptospira icterohæmorrhagiae* (1 : 1000, 1 : 10,000, and 1 : 30,000 on three different occasions). On June 26, the thirteenth day of the illness, the heart rhythm was found to be irregular and a cardiogram taken the same day showed auricular fibrillation (see Fig. 1A). The blood-pressure was 110/75 and the heart did not seem to be enlarged. The auricular fibrillation persisted for four days and on June 30 there was sinus rhythm with T wave changes and a P-R interval at the upper limit of normal (Fig. 1B). The blood pressure was 105/70 and there was marked asthenia. The heart sounds were distant. The size of the heart appeared to be within normal limits. A third cardiogram, taken on July 10, the twenty-seventh day of the illness, was practically normal (Fig. 1C). At that time the jaundice and the albuminuria were subsiding, but the blood pressure remained low and the temperature was now elevated. The lowest blood pressure reading, 90/70, was recorded on July 14, at a time when the renal signs cleared up completely and the blood urea became normal. Blood pressure, pulse rate, and temperature are charted in Fig. 2. After this the patient's general condition gradually improved, the temperature

FIG. 2.—S—Systolic blood pressure; D—Diastolic blood pressure; P—Heart rate; T—Temperature. The figures represent the average reading for each week.

became normal, and the blood pressure started to rise, reaching 125/80 before his discharge in a satisfactory condition on August 25. The jaundice completely disappeared by the end of July. A screen examination on August 13 showed the heart to be normal in size and shape.

DISCUSSION

The case reported here showed evidence of a transient myocardial involvement. In the first instance it was the arrhythmia that drew our attention to the heart and prompted us to do electrocardiographic investigations. It is generally agreed that in the absence of disturbances of the rhythm the diagnosis of acute myocarditis is extremely difficult on purely clinical grounds, and as an example Saphir in his remarkable paper on myocarditis quotes Scherf and Boyd stating that "the frequency of myocarditis and the difficulty of its diagnosis are generally appreciated only after electrocardiographic studies of clinical material have been conducted regularly." If more extensive electrocardiographic studies were made in cases of spirochætal jaundice, especially in cases with very low blood pressure and changes in the character of the heart sounds, more instances of myocardial involvement might perhaps be encountered.

In the light of the anatomical findings in fatal cases in man and in experimental spirochætosis in guinea-pigs, the electrocardiographic changes can be accounted for by direct

involvement of the heart, either in the form of multiple hæmorrhages or of direct toxic damage to the heart muscle, or both. Although it is more likely that the electrocardiographic changes in our case and in the cases quoted were due to direct toxic damage to the heart muscle, hæmorrhages in the heart may have been a causative factor. We believe that widespread minute hæmorrhages in the heart, whatever the primary disease is, can produce electrocardiographic changes, especially displacement of the S-T segment and T wave changes. In support of this we mention an unreported case of traumatic asphyxia in a child, aged 5, with bluish discoloration of the chest and neck, widespread petechiæ over the upper part of the chest extending up to the neck, and subconjunctival hæmorrhages, in which we were able to demonstrate marked and transient depression of the S-T segment. We think that in this case the cardiographic changes were caused by subendocardial hæmorrhages. The pericarditis in one of the cases of Clapper and Myers (1943) appeared at a time when the blood urea was 420 mg. per 100 c.c., and it was very probably uræmic in origin. On the other hand, it is unlikely that the high blood urea, so common in spirochætal jaundice, could have been responsible for the electrocardiographic changes observed in the cases reviewed in this paper.

SUMMARY

The papers dealing with the cardiac complications of spirochætal jaundice are briefly reviewed.

One case with cardiographic evidence of transient myocardial involvement is added to those already reported.

The probable causes for the electrocardiographic changes are discussed.

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