Electrocardiographic Findings in Epidemic Hemorrhagic Fever

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Mounting experiences with the newly discovered disease, epidemic hemorrhagic fever indicate that the basic pathologic lesion affects the vascular system. An electrocardiographic survey in 53 cases has disclosed that during the acute phase of the illness more than 50 per cent of patients show alterations. These, however, are nonspecific and resemble changes recorded in other acute infections. There was no clinical or electrocardiographic evidence of residual cardiac injury in many cases re-evaluated up to six months after the acute illness.

During the late spring and fall seasons of 1952 the members of the medical service of the 11th Evacuation Hospital stationed in Korea cared for 53 cases of epidemic hemorrhagic fever. The patients were young men, and all but three were from the United States Armed Forces, one of the remainder was from the Turkish Brigade, and the other two from the Republic of Korea Army. The disease presented itself in all degrees of severity from a mild afebrile type, with minimal physical and laboratory findings, to an intensely severe form requiring prolonged hospitalization and convalescence. There were two deaths. All patients were seen in the acute stage of their illness and were followed throughout the disease. Many were evaluated at three- and six-month follow-up periods. The general nature of the disease has been presented in a recent symposium, and the present cases were similar in symptomatology and course to those described therein. Recently, somewhat similar electrocardiographic alterations have been briefly recorded in other series.

Early in the course of evaluation of these patients, it was noted that a significant number showed evidence of electrocardiographic alteration, and hence all subsequent cases were followed with one or more electrocardiographic tracings which included standard limb leads, unipolar limb leads and chest V leads. The vector method of cardiographic analysis as outlined by Grant and Estes and others was utilized when indicated. In all, 150 tracings were made on the 53 patients in the acute and convalescent phases, an average of nearly three per patient. Twenty-eight patients, or slightly more than one half, showed an abnormality in the electrocardiogram and 25 showed no abnormality. Most of the abnormalities were temporary and the tracings reverted to normal within several days.

There was no single diagnostic change observed, but in general the abnormalities were of four types: (1) T-wave alterations suggestive of a myocardial ischemia; (2) prolongation of the QRS complex indicative of a conduction defect; (3) T-wave peaking and Q-T prolongations suggesting electrolyte imbalances; and (4) bradycardia. No arrhythmias or A-V conduction defects were encountered. In some cases more than one of the above types of change were noted simultaneously or on consecutive tracings. S-T segment deviations were present in three cases. These suggested a focal anterior pericarditis.

The most common abnormality was that involving a shift in the direction of the T-wave vector. This change was seen in 14 patients. Invariably it was present early in the disease (third to fifth day) and was often correlated with a pronounced febrile state. Vascular hypotension and advanced uremia were not usually present at this time. The T waves were usually inverted in lead III, were inverted or diphasic in lead II, and, in two instances, were inverted in all three standard limb leads (fig. 1). The QRS-T spatial vector angle, normally 45 degrees or less, was widened in all 14 cases to between 85 and 180 degrees. These altera-
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T-wave alterations were characteristic of varying degrees of myocardial ischemia and strain. In all cases where serial tracings were available, the tracings reverted to normal in from 2 to 15 days. In conjunction with the above findings, or occurring separately, were low-voltage T waves in all leads on six occasions (fig. 2). These also reverted to normal within a few days. In one instance, in a patient with very severe necrotizing tracheobronchitis and pneumonitis, the pattern was typical of acute cor pulmonale. This patient ultimately recovered after coughing up large necrotic casts of his respiratory tree. The electrocardiogram also returned to normal.

Evidence of delayed intraventricular conduction time was present in five cases. The duration of the QRS ranged from 0.10 to 0.13 second, and the degree of block varied in several cases from time to time. Two were examples of right bundle-branch block (fig. 3), and the other three of an intermediate or indeterminate type. In four out of the five the block disappeared early, and in the fifth it was still present six months after the acute illness.

Tall peaked T waves were present six times. They were most prominent in the precordial leads and almost invariably coincided with the maximum period of anuria (fig. 4). They were characteristic of moderate degrees of hyperkalemia. The Q-T intervals were prolonged in three of the above cases, also at the height
of uremia, suggesting some concomitant hypocalcemia. These changes were transient and disappeared as soon as adequate diuresis was established. No example of hypokalemia during the diuretic phase was encountered.

Bradycardia (rates below 60 per minute) was seen in 11 patients. It occurred early in the convalescent phase of the illness, was not associated with fever and usually developed during the diuretic period. In none of these was there an accompanying bundle-branch block. Rates as low as 40 or 45 per minute were not uncommon.

**DISCUSSION**

The occurrence of a significant number of electrocardiographic alterations in epidemic hemorrhagic fever is not surprising when one considers the pathologic anatomy of this disease. The basic defect is a widespread capillary damage with its sequelae in multiple organs and systems. In this regard it resembles several of the rickettsial diseases. Specifically the heart has shown subepicardial and subendocardial hemorrhages, especially into the atria, and cellular infiltrates into areas in the myocardium, endocardium and epicardium.

The electrocardiographic changes, particularly the T wave alterations, are rather "non-specific" and are known to occur in some other infectious diseases. These include diphtheria, acute rheumatic fever, typhus and scrub typhus, infectious mononucleosis, poliomyelitis (early in the febrile stage), and others.

In the present cases the QRS and T-wave changes are most closely correlated with the maximum temperature and the crisis of the illness, and these alterations may represent...
the effects of fever and its increased metabolic demands on the myocardial musculature. It is likely, too, that other factors come into play at this time. These include: (1) acute circulatory insufficiency due to hemoconcentration, peripheral circulatory collapse, and others; (2) sympathetic and vagal nerve action; (3) relative coronary insufficiency; and (4) toxic effects of the infectious agent on the heart.

It is important to record that despite the relatively high percentage of alterations noted early in the disease, no evidence of permanent cardiac damage has been observed in this series, with the possible exception of a persistent bundle-branch block, of no obvious clinical significance, in one case. It is entirely possible that the latter defect may have been present prior to the onset of the hemorrhagic fever in this case.

**Summary**

In a total of 53 cases of acute epidemic hemorrhagic fever serial electrocardiograms revealed a variety of alterations in 28, or 53 per cent.

In general the changes were of four types: (1) T-wave variations both in direction and amplitude; (2) bundle-branch block; (3) evidence of electrolyte imbalances; and (4) bradycardia.

With one possible exception there were no evidences of residual cardiac damage on tracings taken in many cases three and six months after the acute illness. Hence latent heart disease should not be of concern to persons who have suffered with this disease.

**Sumario Español**

En un total de 53 casos de fiebre hemorrágica epidémica aguda electrocardiogramas en serie revelaron una variedad de alteraciones en 28, o sea 53 por ciento.

En general los cambios fueron de cuatro tipos: (1) variaciones en la onda T en dirección y amplitud; (2) bloqueo de rama; (3) evidencia de desbalance electrolítico; y (4) bradicardia.

Con una posible excepción no hubo evidencias de daño residual cardíaco en los trazados obtenidos en muchos casos tres y seis meses luego de la enfermedad aguda. Por lo tanto aquellas personas que han sufrido esta enfermedad no deben de preocuparse en cuanto se refiere a enfermedad cardíaca como resultado de la misma.

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