Acute Myocardial Infarction Revealed in an Isolated Premature Ventricular Beat

By Kermit H. Katz, M.D., Morton S. Berk, M.D., and Chaim I. Mayman, M.D.

Electrocardiograms of 2 patients, suspected instances of acute myocardial infarction, offered an early diagnostic clue in that there were isolated premature ventricular beats manifesting the characteristic changes produced by infarction of the myocardium. In each instance these features were present before the remainder of the electrocardiographic record was conclusive.

The electrocardiogram is the most consistently useful diagnostic tool in the identification of myocardial infarction. Over 90 per cent of instances of myocardial infarction permit diagnosis by conventional electrocardiographic technics at some time during the clinical course. There remains, however, a small group of patients in which electrocardiographic changes are not sufficiently marked, or do not occur promptly enough to be helpful in making an early diagnosis. Any diagnostic clue afforded by the electrocardiogram that would diminish the size of this group of patients could be very useful.

Recently we encountered such a diagnostic precursor in 2 patients, simultaneously hospitalized, who were suspected of having acute myocardial infarction, but whose electrocardiograms on admission were not conclusive. In each instance, typical changes of early myocardial infarction (prominent Q wave, elevated S-T segment, inverted T wave) were not present in the dominant rhythm, but were apparent in a premature beat. As the evolution of serial electrocardiographic tracings proved, the configuration of these premature beats was adequate to permit a diagnosis of myocardial infarction.

Case Reports

Case 1. While lying in bed in the hospital on January 1, 1958, 1 month after fracturing her hip and undergoing insertion of a Smith-Petersen nail, an 81-year-old woman experienced precordial aching of increasing severity with radiation to the back and down both arms to the fingertips. Apart from slight nausea, she disclaimed any other symptoms. Digitalis was being administered because of mild congestive heart failure.

On examination, frequent premature beats were noted. The heart sounds were normal; there were no murmurs or a friction rub. There were no signs of congestive failure. An electrocardiogram (fig. 1) on January 1, 1958 revealed sinus arrhythmia, atrial and occasional ventricular premature beats. The S-T segments were depressed in leads II, III, and aVp, and elevated in leads aV1 and V2. In lead V2 a conspicuous premature ventricular beat manifested a deep Q wave, marked elevation of the S-T segment, and late inversion of the T wave.

The next day the temperature rose to 100 F. The heart sounds became distant and there were no premature beats. On January 3, 1958 a cardiac friction rub was audible in the fourth intercostal space near the left sternal border. An electrocardiogram (fig. 2) taken at this time revealed the T waves to be isoelectric in lead I and inverted in the left precordial leads. In lead V3 there was a small broad Q wave with marked elevation of the S-T segments and terminal inversion of the T waves.

On January 14, 1958 an electrocardiogram (fig. 3) showed advanced changes characteristic of the evolution of the pattern of acute myocardial infarction.

The patient required several doses of opiates during the first 72 hours after the onset of chest pain, but she recovered slowly thereafter.

Case 2. A 63-year-old white widow entered the Boston City Hospital on January 3, 1958 because of severe squeezing, retrosternal pain, radiating down the arms, of several hours' duration. Two years previously the patient had experienced protracted chest pain, and was kept in bed for 2 weeks. Subsequently, she had become progressively limited in her physical activities by dyspnea and by recurrent, oppressive chest pains.

Examination showed rales at the right lung base
and occasional premature beats. There were no thrills or rubs. The sedimentation rate was 44 mm. per hour (Wintrobe). The white blood cell count was 12,200, with 84 per cent neutrophils and 16 per cent lymphocytes. The serum glutamic oxalacetic transaminase level was 82 units. A roentgenogram of the chest showed bilateral increase in the bronchovascular markings. The heart size was at the upper limit of normal. The aorta was elongated. An electrocardiogram (fig. 4) taken on entry showed a moderate number of premature beats, sometimes resulting in bigeminy. Deep Q waves were present in leads III and aVF. S-T segments were depressed in leads I, II, aVL, and the left precordial leads. In leads aVF and V1 there were ventricular premature beats with prominent Q waves and very high take-off of the S-T segments. In an electrocardiogram (fig. 5) 3 days later the T waves in leads III and aVF were inverted.

Because of gradually increasing pulmonary edema, the patient was digitalized about 12 hours after admission. One week after entry, the patient had a recurrence of severe anterior chest pain requiring treatment with morphine. Electrocardiograms showed transient left bundle-branch block. At this time treatment with heparin was begun. The patient's subsequent clinical course was relatively uneventful until February 3, 1958, 1 month after admission, when she was observed to become suddenly pale and weak, with no perceptible pulse or blood pressure. In a matter of minutes the patient had died.

At postmortem examination there was extensive healing of a relatively recent myocardial infarction, with more recent extension, involving the greater portion of the interventricular septum and posterior left ventricular wall—from base to apex. Over the posterior aspect of the left ventricle was a fibrinous pericarditis. There was focal fibrosis of the left ventricular myocardium. The coronary arteries showed marked atherosclerosis, with segmental old occlusions of the right coronary artery and the left circumflex branch of the left coronary artery.

**DISCUSSION**

Dressler and Simonson et al. each reported an instance of characteristic changes in ventricular premature beats pointing to the diagnosis of myocardial infarction that was not otherwise detectable by electrocardiogram because of the pattern of bundle-branch block. Scherf and Schott discussed this phenomenon and added an instance in which the diagnosis was dependent upon the characteristic appearance of an atrial premature beat.

The finding of the electrocardiographic pattern of myocardial infarction in a premature ventricular beat in one of our cases led to the detection of the same phenomenon in the other. Inasmuch as premature ventricular beats are expected to be bizarre in appearance, no detailed attention is consistently
FIG. 2 Top. Changes from the previous electrocardiogram include inversion of the T waves in leads V1-4. In lead V5 there is a small, broad Q wave and marked elevation of the S-T segments.

FIG. 3 Bottom. Two weeks after the onset of symptoms the electrocardiogram now shows many characteristics of acute myocardial infarction of the anterior wall of the left ventricle. T waves are inverted in the 3 bipolar limb leads and in leads V2-6. R waves are rudimentary in the right precordial leads.

given to their configuration. The possibility suggests itself that this phenomenon might be more frequently encountered if clinicians were alert to it.

Premature beats, as commonly encountered, show the configuration of a bundle-branch block ventricular complex. The T wave is usually directed opposite to the main deflection of the QRS complex. If an S-T segment is present, as is usually the case, it is dis-
placed in the direction of the T wave. In the cases here reported Q waves were prominent, and although the T waves were directed opposite to the main deflection of the QRS complex, the S-T segments were elevated and displaced opposite to the direction of the T waves.

In neither of the 2 cases here presented was there any difficulty in judging, on several bases, that the patients suffered from serious
coronary artery disease. In case 1, however, because of the patient's prolonged period of inactivity and her preceding operation, the possibility of the sudden onset of chest pain being a manifestation of pulmonary embolism was initially considered. The finding of a pattern of myocardial infarction in the premature ventricular beat, before the remainder of the electrocardiogram would permit this diagnosis, suggests its value in more obscure clinical problems.

In case 2, although there was no question as to the presence of coronary artery disease, it was possible to contend—early in her hospitalization—that the findings in the initial electrocardiogram were those of acute coronary insufficiency, superimposed upon the signs of old myocardial infarction, rather than of acute myocardial infarction. Here the significance of the single premature ventricular beats, showing the configuration encountered in acute myocardial infarction, was decisive.

**Summary**

Two cases are reported in which the correct diagnosis of acute myocardial infarction was made possible by characteristic electrocardiographic features of infarction in isolated premature ventricular beats. These findings were present before the dominant electrocardiographic pattern became completely characteristic of the disease. It is suggested that the phenomenon here reported might be encountered more frequently as an early electrocardiographic evidence of acute myocardial infarction if clinicians were alert to its significance.

**Summario in Interlingua**

Es reportate duo casos in quæ le correcte diagnoze de acute infarcimento myocardial eseva possibile gratias al characteristic aspecto electrocardiographic de isolate pulsos ventricular prematur. Iste aspecto eseva presente ante que le dominante configuration electrocardiographic deveniva completamente characteristic del morbo. Es opinate que le phenomeno hic reportate va possibilmente incontrar se in plus frequente occasiones si le clinicos es rendite conscie de su signification.

**REFERENCES**


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**The Coronary Arteries.** A few essential points in the anatomy and physiology of the heart may here engage our attention for a few minutes. The coronary arteries are the Abana and Pharpar of the vascular rivers, "lucid streams," which water the very citadel of life.—**William Osler, M.D. Lectures on Angina Pectoris and Allied States, 1897.**
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