Electrocardiographic Pattern of Massive Myocardial Infarction without Pathologic Confirmation

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WITH rare exceptions the appearance in the electrocardiogram of Q waves in association with elevated ST segments represents the most reliable criteria of myocardial infarction. It is the purpose of this paper to present a case in which there was dissociation between electrical and anatomic findings. In this case the electrocardiographic changes were "diagnostic" of myocardial infarction, yet at autopsy the heart showed no evidence of infarction.

Case Report

The patient, a 67-year-old woman, was admitted to the local mental hospital on December 10, 1963, with a diagnosis of "chronic brain syndrome." The medical history included onset of diabetes mellitus at the age of 60 adequately controlled with 1 Gm. of tolbutamide per day. No history of hypertension, angina pectoris, or other cardiovascular disease was elicited.

On February 19, 1964, the patient was transferred to the infirmary because of a temperature of 103 F. and diabetes mellitus out of control. Physical examination at the time of admission revealed a thin, elderly, hirsute woman. Blood pressure was 144/64 mm. Hg and the pulse was 76 and regular. The lungs were clear. The heart was normal in size and the only abnormality was a grade-II/IV systolic ejection murmur at the base of the heart. A large abscess was noted on the left elbow. Hemoglobin was 16.6 Gm. with a hematocrit value of 49 per cent and a white blood count of 15,000. An electrocardiogram on February 20, 1964 (fig. 1), revealed a prolonged QT interval, prominent U waves, and clockwise rotation. The patient was placed on chloramphenicol 500 mg. four times daily, and the abscess was incised and drained. The temperature promptly returned to normal, and on February 27, 1964, the antibiotics were discontinued.

Two days later, however, the temperature again began to rise and on the morning of March 1, 1964, the patient suddenly became cyanotic, the peripheral pulses were not palpable, and the blood pressure was unobtainable. Before any therapy was begun, however, the blood pressure returned to 80/60 mm. Hg, at which time the temperature was 103.6 F. For the first time rales were noted over the base of the left lung posteriorly. Because of nuchal rigidity a lumbar puncture was performed but the examination of spinal fluid showed no abnormalities. The electrocardiogram at that time revealed elevation of the ST segment in leads II, III, and aVF as well as I, aVL, and V$_5$-V$_6$ with loss of R waves in V$_2$-V$_6$. There was shift of axis to the left when compared with the control electrocardiogram (fig. 1). These changes were interpreted as being compatible with an acute myocardial infarction. The patient was treated with nasal oxygen, vasopressors, and antibiotics. She remained comatose with blood pressure in the range of 100 mm. Hg systolic by palpation. Laboratory studies on March 2, 1964, revealed a hemoglobin of 18 Gm., a hematocrit value of 55 per cent, and a white blood-cell count of 10,500. The blood sugar was 418 mg. per cent, blood urea nitrogen 74 mg. per cent, sodium 145 mEq./L., and potassium 6.2 mEq./L. Enzyme studies were not available at this institution. Repeat electrocardiograms revealed lowering of ST segments consistent with an evolving myocardial infarction (fig. 1). On March 3, 1964, a pleural friction rub was heard over the left base posteriorly and chest x-ray revealed definite infiltrate in this area. A third electrocardiogram obtained on March 4, 1964, revealed a pattern consistent with progressive evolution of a massive myocardial infarction including pathologic Q waves in II, III, and aVF as well as loss of R wave in the precordial leads. A nonparoxysmal nodal tachycardia was noted at that time (fig. 1). The patient’s condition remained essentially unchanged and she died on March 4, 1964.
Compared to control electrocardiogram (2-20-64), the limb leads (upper) reveal development of QS pattern with elevated ST segment in II, III, and aVR. Small Q waves with elevated ST segment are also noted in I, aVL, and aVR. An abnormal R wave is present in aVR. The precordial leads (lower) demonstrate QS pattern with elevated ST segment in V4-V5 and qR with elevation of ST segment in V6.

An autopsy was performed on March 5, 1964. The heart weighed 340 Gm. The pericardial sac contained a small amount of fluid. The surface of the heart was not remarkable. The heart was firm and uniform in consistency throughout and the cut surfaces showed no evidence of fibrosis.
or infarction. Examination of the coronary arteries revealed scattered calcified plaques in the anterior descending and circumflex branches of the left coronary artery. All major branches were dissected out and there was no occlusion or significant reduction of the lumen. Microscopic examination of numerous sections taken from various parts of the heart showed no evidence of recent or old infarction. Examination of the lungs revealed a left-lower lobe pneumonia, purulent in nature. The remainder of the autopsy was non-informing.

Discussion

The electrocardiographic patterns noted in our case have so consistently been associated with myocardial infarction, that the remotest possibility of an error in proper identification of the electrocardiogram and pathologic specimens would have made the publication of this case prohibitive. However, the fact that one of the authors (E.L.W.) cared for this patient, took the electrocardiogram dated March 1, 1964, and attended the autopsy, and that no other deaths occurred nor were other autopsies performed during a two-day interval before and after the demise of our patient, makes this possibility unlikely.

The heart was examined independently by two pathologists; neither one found any evidence of infarction. Should such have taken place, the 4-day interval between onset of illness and death provided optimal time for both gross and microscopic recognition of myocardial infarction. The cause of death in our patient was purulent pneumonia complicated by peripheral vascular collapse.

In 1938 Dittler and McGavack described a case in which the electrocardiogram indicated myocardial infarction but the autopsy showed acute pancreatic necrosis, the coronary arteries were normal, and there was no evidence of myocardial infarction. In 1954, Bauerlein and Stobbe also reported an autopsied case of acute pancreatitis in which electrocardiographic changes were similar to those noted in our case. However their patient died 30 hours after onset of symptoms and serial electrocardiograms were not recorded. A similar instance of acute pancreatitis was recently described by Fulton and Marriott with serial electrocardiographic tracings showing the development of QR pattern which reverted to an RS pattern the day prior to the patient’s death.

Summary

A case of a 67-year-old patient with an electrocardiographic pattern of a massive myocardial infarction but with a normal heart at autopsy is described. The cause of death was purulent pneumonia complicated by peripheral vascular collapse.

The findings in our case simply reconfirm the well-established principle that the surface electrocardiogram mirrors the electrical phenomena of the heart, which may or may not parallel histologic changes.

References

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