Probable Myocardial Infarction in a 14-Year-Old Boy

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After vigorous physical activity a 14-year-old boy from a family with a strong history of cardiac disease developed symptoms consistent with a myocardial infarction, a relatively rare event in this age group. Isoenzyme patterns of creatine kinase and lactate dehydrogenase were consistent with the occurrence of an infarction.

Additional Keyphrases: isoenzymes • risk factors for cardiac disease • dysrhythmia

Myocardial infarction in adolescents is so rare that the World Health Organization Acute Myocardial Infarct Community Register Study of 1971, which recorded 10,121 infarcts in a European population of about three million, listed 20 years as the earliest age of a patient with an infarction (1). Here I report the case of a 14-year-old boy who had symptomatic and enzymatic evidence of an acute myocardial infarct.

Case Report

This patient was admitted to his local hospital after complaining of chest pain, nausea, and tachycardia. He had been working all day, engaged in vigorous manual labor on the family farm on a relatively warm day. His past medical history was significant for only a broken leg and appendicitis. He denied any previous chest pains, palpitations, shortness of breath, or dizziness. On admission, 4 h after the onset of chest pain, his electrocardiogram indicated supraventricular tachycardia. Values for blood gases, electrolytes, glucose, and urea nitrogen were all within normal limits. Specimens for assay of creatine kinase (EC 2.7.3.2) and lactate dehydrogenase (EC 1.1.1.27) isoenzymes were drawn at the local hospital 2.5 and 10 h after the onset of chest pain. They were sent to a local reference laboratory, where these isoenzymes were evaluated electrographically. The first specimen was negative for CK-MB or increased LD1. The second specimen, however, was positive for CK-MB (>10% of the total) and also positive LD1 > LD2.

The patient was treated with lidocaine intravenously and with digoxin and nitroglycerin. When the tachycardia failed to resolve, he was given verapamil, after which he converted to sinus rhythm. He also received propranolol and propranolol.

During the night and next day he experienced several more episodes of tachycardia with heart rates of 180 to 200, and therefore was transferred to our institution 18 h after the onset of chest pain. On admission his temperature, heart rate, respiration, and blood pressure were normal, and he was experiencing no chest pain. Results of his physical examination and electrocardiography were normal. A chest roentgenogram and an echocardiogram showed no evidence of cardiomyopathy or abnormal cardiac anatomy.

Specimens were obtained for isoenzyme assay 32 and 37 h after the onset of chest pain. The results for total CK were, respectively, 269 and 583 U/L (normal 35–200 U/L) and for total LD 428 and 403 U/L (normal 100–225 U/L). These specimens showed 10% and 13% CK-MB, respectively, and LD1 43% LD2 34% at 32 h, and LD1 40% LD2 36% at 37 h. Total CK was determined with reagents from Boehringer Mannheim Diagnostics, Houston, TX (CK-NAC activated) in an Abbott ABA-100 discrete analyzer (Abbott Diagnostics, Irving, TX). Total LD was determined in a continuous-flow analyzer (SMA 12/60; Technicon Instruments Corp., Tarrytown, NY). LD and CK isoenzymes were electrophoresed on cellulose acetate membranes supplied by Helena Laboratories, Beaumont, TX.

Unfortunately, additional determinations of cardiac-derived enzymes and isoenzymes were not ordered later in his hospitalization. Two days after the onset of chest pain all cardiac medications were discontinued, and he was examined with a 24-h Holter monitor. The results of this test were normal, with no dysrhythmias. He continued to do well and on the fifth and last hospital day was given a graded-exercise test; the results were normal with no chest pain or dysrhythmias observed. The patient's daily dose of digoxin after leaving the hospital was 125 μg per day.

The patient returned to this clinic seven weeks later. At that time he reported that one episode of supraventricular tachycardia lasting 2–3 min and associated with chest pain and headache had occurred five or six days after his discharge. Along with four of his siblings, he underwent lipid screening at the Lipid Research Clinic Laboratories at the University of Iowa. All values for total cholesterol, triglyceride, and high-density lipoprotein cholesterol were within normal limits for all five individuals. The patient's digoxin dose was increased to 250 μg per day.

Discussion

Myocardial infarction in children and adolescents is relatively rare. With respect to the risk factors associated with myocardial infarction (2), this patient's serum showed normal concentrations of lipid and glucose, and he was of normal weight and his blood pressure was normal; serum uric acid was not measured. No smoking history was obtained from him, but it is unlikely he could have accumulated a significant number of "pack-years" at age 14. He did, however, have a very strong family history for cardiac disease. His father, who had been obese and hypertensive, died at age 34 of an infarction. A paternal uncle died at 42, also of an infarction, and a maternal aunt suffered from tachycardia.

Many infarctions in younger individuals are associated with vigorous physical exercise (3). This patient had been working on his family farm all day, with only a break for the evening meal, on a relatively warm day. The symptoms first appeared at around 20:00 hours, while he was still engaged in physical labor: baling hay.

The symptoms and isoenzyme patterns were consistent with an acute myocardial infarction. It is unlikely that the dysrhythmia alone could cause the isoenzyme changes. Strauss and Roberts (4) described 38 patients with dysrhythmias, 22 of whom also had chest pain, who did not have infarctions; in none was CK-MB detected. They concluded that cardiac necrosis is needed for release of CK-MB, not
just transient ischemia. Machlinski et al. (6), describing 24 patients with electrophysiologically induced ventricular tachycardia or fibrillation, reported that 10 of these patients showed increases in total CK, but none showed an increased CK-MB in their serum.

References

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p 1814: In this paper the figures were illustrated in the incorrect order. The correct figure order is shown here.

Fig. 1. Protein electrophoreograms obtained by “high-resolution” agarose gel zone electrophoresis
S, serum; C, cerebrospinal fluid (CSF). Healthy subject. In Figs. 1–5, the values of the albumin quotient and IgG index of the same samples are included, for comparison with the electrophoretic patterns

Table 1

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<th>Albumin Quotient</th>
<th>IgG Index</th>
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<tr>
<td>5.1</td>
<td>0.45</td>
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Fig. 2. Protein electrophoreograms obtained by high-resolution agarose gel zone electrophoresis
S, serum; C, CSF. From a case with increased blood–brain barrier permeability and diffuse increase of γ-globulin in the CSF.

Table 2

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<th>Albumin Quotient</th>
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<td>12.0</td>
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Fig. 3. Same as Fig. 2, with a mini-monoclonal band in the γ-globulin zone of the serum and CSF.

Table 3

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<td>14.0</td>
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Fig. 4. Protein electrophoretic patterns obtained by high-resolution agarose gel zone electrophoresis
S, serum; C, CSF. Samples from a patient with multiple sclerosis, demonstrating a normal serum pattern and oligoclonal banding in the CSF.

Table 4

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<th>Albumin Quotient</th>
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Fig. 5. Protein electrophoretic patterns obtained by high-resolution agarose gel zone electrophoresis
S, serum; C, CSF. Samples from a case with acute infection in the central nervous system, indicating increased synthesis and permeability of γ-globulin in the CSF.

Table 5

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Correction