Severe Coronary Artery Disease in a Marathon Runner*

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A 55-year-old male runner was found to have severe triple-vessel coronary disease on angiography soon after completing a 25-mile marathon with no symptoms. This patient raises important questions concerning our understanding of ischemic heart disease.

The relationship between marathon running and coronary artery disease has been controversial. Initially it was felt that marathon running was protective against coronary artery disease after the report of an autopsy in a 70-year-old marathon runner who had large coronary arteries and no coronary disease. Since then, however, there has been ample documentation that marathon runners are at risk for coronary events including sudden death, angina while running, unstable angina, and myocardial infarction.

We report the first case of a runner who completed a marathon with no symptoms who was found to have severe triple-vessel coronary artery disease on coronary arteriography five weeks later.

Case Report

A 55-year-old Hispanic man had a history of hypertension for 23 years for which he was treated with propranolol 80 mg bid and captopril 50 mg bid. The patient had participated actively in athletics since his youth and three years prior to the present admission, he began long-distance running. He ran approximately 30 miles/week and completed the New York marathon in 1984. Prior to running, he consistently took his propranolol and captopril. Occasionally, shortly after beginning to run, he would experience precordial burning discomfort. Usually, this symptom was mild and would disappear as his "body heated up." Because the patient was planning to re-run the marathon in 1985, he was advised to undergo exercise electrocardiography prior to doing so. Due to a misunderstanding, the stress test was scheduled for three days after the marathon. On October 27, 1985, the patient completed the 26-mile run in 4 hours 44 minutes and was asymptomatic throughout. On October 30, 1985, an exercise treadmill test was performed using the Bruce protocol after discontinuation of antihypertension medications. Resting initial blood pressure was 170/110 mm Hg and pulse rate of 60 rose to 220/120 mm Hg and 150 (89 percent predicted maximum) respectively, at peak exercise. The patient experienced no symptoms; however, ST segment depression appeared in leads V$_5$-V$_6$ and increased to a maximum of 4 mm sagging depression at 94 s min (stage 4), at which time the test was terminated by the physician. The marked ECG abnormalities persisted for 5 minutes into the post exercise recovery phase. Complete return to the pre-exercise ECG pattern occurred only after 12 minutes. Exercise was repeated with the addition of thallium one week later. No perfusion defect was demonstrated; however, the marked ECG abnormalities recurred during exercise. The patient was referred for coronary arteriography. Review of systems and social history were non-contributory. The patient's sister had angina pectoris at 45 years of age. Physical examination revealed a healthy appearing, well conditioned man. Blood pressure was 140/104 mm Hg, and pulse rate was 60 and regular. Heart examination revealed an S$_2$. Physical examination was otherwise normal. ECG showed normal sinus rhythm and a pattern of left ventricular hypertrophy. Serum electrolytes, CBC, blood urea nitrogen and serum glucose were normal. Serum cholesterol was 224. Coronary arteriography performed on December 3, 1985 revealed severe coronary artery disease (Fig 1-3). The left ventriculogram showed moderate inferoapical hypokinesis and minimal anterior hypokinesis. Global ejection fraction and end-diastolic pressure were normal.

Discussion

In recent years the cardiovascular stress and risk of cardiac events in athletes over the age of 40 have received increasing attention. This unique patient demonstrates the complexity of the relationship between exercise physiology and ischemic heart disease and raises questions concerning our understanding of coronary blood flow, collateral contribution to myocardial oxygenation and silent ischemia.

To be explained is how a patient with coronary disease of this severity was able to complete a competitive marathon, ranking about 800th in a field of 1,800 over the age of 50 and remain asymptomatic throughout. A number of speculative possibilities exist: ischemia might not have been present during the race. The average speed of the race was 5.28 miles/hour, equivalent to about 5-7 mets. Although the ST

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![Figure 1. Left coronary artery, right anterior oblique view, shows a totally occluded circumflex branch and serial high grade narrowing of the left anterior descending artery.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21555/ on 04/19/2017)
segments were depressed at the equivalent level of exertion during the stress test, the positive stress test may not have been due to ischemia but to subtle myocardial changes because of his hypertension. A training effect on the heart and circulation may have been sufficient to maintain his heart rate and peripheral resistance in a range such that myocardial oxygen requirement was satisfied even by his anatomically compromised circulation. Antihypertension medications may have augmented this effect with propanolol maintaining the cardiac inotropy and heart rate low and captopril maintaining a low afterload.

Ischemia may have been transiently present in this patient. Apparently, he had experienced episodes where burning chest discomfort had abated as he continued exercising. The “walk through” phenomenon was first described by MacAlpin and Kattus in 1966. Initially this phenomenon was explained by the progressive peripheral vasodilatation that normally occurs with exercise. Currently, coronary spasm or a persistence of resting normal coronary tone superimposed on a partially occluded coronary segment is thought to explain this phenomenon, with angina improving as the vessel dilates during exercise. It is also possible that coronary collaterals may increase in size as exercise progresses or flow through fixed collaterals may increase because of increased runoff due to dilation of vessels in the ischemic area. That collaterals either can proliferate or that collateral flow can increase is suggested by the observation that intense physical conditioning can decrease the degree of ST depression at the same or higher double product. An alternative conjectural explanation may be the development of progressively more efficient extraction of oxygen from the compromised coronary flow after the onset of exertion in a conditioned individual; this explanation does not invoke the necessity for an increase in blood flow to an ischemic area after onset of exercise.

The third possibility is that ischemia was present throughout the race. Perhaps in some patients anaerobic metabolism becomes efficient enough to support active cardiac contraction if conditioning is performed gradually and with slowly increasing ischemia over a long period of time. Perhaps the patient had angina throughout the race, but had trained himself to be oblivious to it, or to misinterpret it as a normal discomfort associated with vigorous exertion.

Although we cannot know precisely what allows a patient with the coronary anatomy of our patient to run vigorously for long periods one day but drop dead during similar exertion the next day, as reported in many runners autopsied after sudden death, it appears that these patients may be delicately balanced between enough oxygenation, enough anaerobic metabolism and not too excessive demand. It may be that it is when this balance is upset, even a bit, that myocardial infarction or sudden death occurs.

This case serves to emphasize the importance of periodic stress tests and cardiac evaluation in the older well conditioned athlete. The ability to run 26 miles does not guarantee the absence of occlusive coronary artery disease.

References
2. Waller BF, Roberts WC. Sudden death while running in conditioned runners aged 40 years or over. Am J Card 1980; 45:1292-1300