Sudden death of a champion athlete

Autopsy findings

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Summary

We report the autopsy findings after the sudden death of a champion swimmer. The athlete was asymptomatic and had only recently retired from a sport in which he had been the South African captain for 4 years and the national record holder in the 400 m medley event. He died suddenly shortly after returning from a 5 km run. At autopsy there was histological evidence of hypertrophic cardiomyopathy.

While there is growing evidence that regular exercise may reduce the risk of sudden death from ischaemic heart disease,1-4 apparently healthy individuals still die suddenly and unexpectedly during exercise.5,6 When death occurs in a highly trained athlete, concern is frequently expressed because vigorous exercise not only failed to prevent heart disease, but might even have accelerated the fatal event.

Recently, much press publicity was attracted by the sudden death after exercise of a 25-year-old athlete who had been a national champion, record holder and captain of the South African swimming team for 5 years. To place his death in perspective, we report the autopsy findings.

Case report

The deceased, aged 25 years, had been a competitive swimmer for 17 years before his retirement in 1982. During the first 5 years of his career he swam about 8 km a day, 6 days a week for the 7-month swimming season (a total of approximately 1440 km/yr). For the next 5 years he swam about 10 km a day (a total of approximately 1 800 km/yr), and for the last 7 years he swam about 15 km a day (a total of approximately 3 150 km/yr).

His competitive achievements were particularly impressive: he represented his province for 10 consecutive years, for 7 of which he was team captain. He set provincial records, which still stand, in 8 different events from 100 m to 1 500 m in both freestyle and breaststroke and at one time held 5 national titles. He gained his first Springbok cap in 1977 and became Springbok captain in 1978. He won the 400 m medley at the National Championship 6 years in a row — a record that still stands. In addition, he represented his province in both surf lifesaving and water polo, and was selected to represent South Africa in the modern pentathlon and still-water lifesaving.

After he retired from competitive sport, he continued to swim and to run regularly. One month before his death he competed in the South African Surf Lifesaving Championships. On the day of his death he had been for an early morning run of about 5 km. He was discovered later that day collapsed against the inside of his door, still dressed in his running clothes.

Past medical history

The patient was a non-smoker and essentially a teetotaller. A malignant melanoma on the lower part of one of his legs had been removed 2 years before his death. There was no apparent recurrence and the patient was asymptomatic.

There was no relevant family history. The first-born son of the family had died at the age of 3 months from an unspecified cardiac abnormality. Two remaining brothers, aged 30 and 19 years, are well and have no symptoms. Both were examined clinically. The eldest brother had a normal blood pressure (110/70 mmHg) and there were no abnormal findings on either clinical examination or on the resting ECG. The echocardiogram showed that the interventricular septum (1,6 cm) and posterior wall (1,2 cm) were of normal size, giving a septal: free wall ratio of 1,3. The end-diastolic diameter was 4,3 cm and there was no abnormal movement of the mitral valve.

Clinical examination of the younger brother, a marathon runner, showed his blood pressure to be normal (110/70 mmHg) and there was no evidence of ventricular hypertrophy. A third heart sound was heard at the apex. The resting ECG showed left ventricular hypertrophy by voltage criteria and no abnormality was detected on maximal exercise testing up to a heart rate of 175/min. On echocardiography there was no asymmetrical septal hypertrophy (left ventricular posterior wall thickness 0,9 cm, interventricular septal thickness 1,04 cm, giving a septal: free wall ratio of 1,1), the mitral valve moved normally and the end-diastolic diameter was 4,8 cm.

Autopsy findings

There were no macroscopic abnormalities in the brain, thorax or abdominal cavity that might have explained sudden death. In particular, there was no evidence of metastatic melanoma.

The heart weighed 445 g and showed symmetrical left ventricular hypertrophy. The left ventricular free-wall thickness was 1,7 cm and that of the interventricular septum 1,8 cm. All the valves were normal as was the subaortic area. The coronary arteries showed no significant narrowing.

On histological examination the epicardial coronary arteries were normal although prominent intimal cushions were seen in the proximal portion of the left anterior descending coronary artery. The left ventricular free wall showed no evidence of acute ischaemic damage, and the small coronary arteries were normal. However, myofibre disarray diagnostic of hypertrophic cardiomyopathy involving about 81% of the longitudinally sectioned myofibres was present in the interventricular septum (Fig. 1).
The right ventricle showed minimal disarray with some nuclear changes of hypertrophy. The myocardium of the left and right ventricles showed areas of intra-alveolar oedema and fresh haemorrhage.

Discussion

Sudden death during exercise in athletes over 40 years of age is usually due to coronary heart disease, whereas similar deaths reported in younger athletes were more usually due to hypertrophic cardiomyopathy. Recently, cases have been described in which both conditions coexisted in athletes dying suddenly.

This patient therefore fits the more usual pattern of hypertrophic cardiomyopathy being the cause of sudden death in a young athlete. The absence of premonitory symptoms and the capacity for superior athletic performance also fit the previous descriptions of this condition.

It must be stressed that the diagnosis of hypertrophic cardiomyopathy in this patient was made on the basis of histological10 rather than macroscopic criteria. The heart showed symmetrical left ventricular hypertrophy which is the form of hypertrophy encountered in 31%-40% of patients with hypertrophic cardiomyopathy.

The clinical question that arises is: had the patient developed symptoms, could the condition have been diagnosed antemortem? The resting ECG may be normal or be considered to illustrate the 'normal athletic heart' in people who subsequently die from hypertrophic cardiomyopathy.6,11 and at autopsy there may be no asymmetrical septal hypertrophy in between 31% and 40% of cases with hypertrophic cardiomyopathy.10,12 Thus a normal resting ECG and the absence of asymmetrical septal hypertrophy on echocardiography (as found in the deceased's brothers) does not exclude the possibility of hypertrophic cardiomyopathy.

Further research should therefore aim to distinguish the usual hypertrophy of the 'athlete's heart' from the pre-decompensated hypertrophy of hypertrophic cardiomyopathy. One pointer may be differences in heart weight. In our previous reports of autopsy findings in trained runners dying suddenly, all 3 who died from coronary heart disease had heart weights ranging from 344 to 357 g (median 317 g), whereas the 3 with hypertrophic cardiomyopathy had heart weights ranging from 405 to 460 g (median 420 g).1,12 Maron et al. have also reported heart weights ranging from 360 to 630 g (mean 475 g) in athletes dying suddenly from hypertrophic cardiomyopathy. This evidence, incomplete as it is, suggests that the heart weight of trained runners may normally be less than 400 g, whereas in hypertrophic cardiomyopathy heart weights in excess of 400 g may be more common. If this is so, non-invasive measurement of heart weight may be helpful in identifying people with hypertrophic cardiomyopathy but without asymmetrical septal hypertrophy.

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REFERENCES