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Letter to the Editor

Are Exertional Syncope Episodes Actually Caused by Myocardial Crypts?

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Letter to the Editor

We read with interest the article by Allen *et al.* about a 20-year-old female patient with exertional syncope attributed to myocardial crypts in the interventricular septum ^[1]. Her troponin level was markedly elevated, which was attributed to cardiac arrest ^[1]. Genetic testing revealed a mutation in a sarcomere gene associated with hypertrophic cardiomyopathy ^[1]. She received an implantable cardioverter-defibrillator (ICD) ^[1]. Although the study is interesting, several points require further discussion.

First, alternative causes for the exertional syncope in this patient were not sufficiently ruled out ^[1]. In addition to malignant ventricular arrhythmias and arrhythmogenic or hypertrophic cardiomyopathy, exertional syncope can also be caused by coronary anomalies, exercise-induced collapse (blood pooling in the legs upon abrupt cessation of activity), dehydration or heat stress with resulting hypovolemia, vasovagal syncope due to a sudden drop in heart rate or blood pressure, subclavian steal syndrome, or seizures triggered by hyperventilation. The results of coronary angiography, exercise testing, and MIBI-SPECT should be reported.

Second, no neurological examinations were performed. To determine whether the exertion-induced syncope was neurological or cardiac in origin, a cerebral MRI, carotid ultrasound to rule out subclavian steal syndrome, an electroencephalogram to rule out increased excitability, and nerve conduction velocity measurements to rule out autonomic neuropathy, which can lead to vasovagal syncope, would have been essential. The family history regarding epilepsy or hereditary neuropathy should also be investigated. Did the patient experience urinary or fecal incontinence during the syncope, and was tongue biting observed afterward?

Third, the type and duration of the physical exertion that triggered the syncope were not specified. To assess the etiology and determine whether the syncope was indeed related to physical exertion, it is crucial to know the type and duration of the exertion the patient performed prior to fainting. It should also be stated whether the syncope was observed or not, and whether it was a convulsive or non-convulsive syncope.

The fourth point is that no long-term outcomes have been reported ^[1]. Of particular interest is whether the ICD has recorded any malignant arrhythmias since implantation, whether the ICD has triggered an alarm, and whether the patient has experienced another syncope.

The fifth point is that the identified mutation made responsible for the myocardial crypts has not been described ^[1]. Knowledge of the gene, the mutation, and the inheritance pattern is crucial, as the patient is of reproductive age and requires genetic counseling. This information is also important for predicting the course and outcome of the disease. It should also be reported whether the mutation has been detected in other family members, as there is a known family history of sudden cardiac death ^[1]. Finally, it should be considered that the myocardial crypts are not related to the syncope and are more likely a benign phenomenon than a cause of disease. The long latency period between the first and second syncopal episodes, as well as the otherwise normal morphology and kinetics of the heart, argue against a causal relationship. Is it conceivable that the elevated troponin level is due to kidney failure rather than heart muscle failure?

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