

Should the ECG Be Excluded from Sports Certification Screening?

A Typical Case Supports Inclusion

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Afari and colleagues¹ have reported an illustrative case of sudden cardiac arrest (SCA) in an 18-year-old varsity athlete who collapsed during strenuous exercise on a basketball court. He had presumably passed routine precertification screening, which—as is usual in the United States—did not include an electrocardiogram (ECG). Indeed, the young man had never had a complete ECG.

Ventricular fibrillation was documented before his successful resuscitation. On his eventual admission to the emergency department, a resting ECG was diagnostic of apical hypertrophic cardiomyopathy (HCM). Further evaluation yielded another high-risk cardiovascular condition: anomalous origin of the right coronary artery from the opposite sinus of Valsalva (R-ACAOS). Either condition can be fatal; subclassification into “lower” and “very high” risk categories has been proposed in pilot studies.²

Had the patient undergone a precertification resting or exercise ECG, he could have been referred to a cardiologist. However, primary-practice cardiologists might not be immediately prepared to apply optimal treatments for rare and complex pathologic conditions: decision-making is frequently (and necessarily) improvised and empirical. Toward better universal knowledge, the Texas Heart Institute supports the creation of centers of excellence, involving multidisciplinary teams that are dedicated to the study of rare and serious conditions in well-equipped, technologically up-to-date clinical environments.²

The optimal approach to screening athletes for risk factors involves 3 considerations:

1) Identifying cardiovascular conditions that can lead to sudden cardiac death during sports activities.

2) Evaluating typical triggers for SCA. Pertinent to this case, the usual precipitant in both apical HCM and R-ACAOS is strenuous exercise—and competitive basketball is recognized as the highest-risk sporting activity in the U.S.^{3,4}

3) Evaluating functional severity, which differs in individual patients who have a given pathologic condition.⁵ During exercise, R-ACAOS causes SCA only by means of ischemia. Ischemia essentially occurs because of the intramural course of the proximal ectopic artery and phasic lateral compression.⁵ A nuclear adenosine stress test is unlikely to reproduce ischemia by inducing vasodilation. Only a maximal treadmill nuclear stress test can increase cardiac output, aortic dilation, and tachycardia—thereby augmenting the systolic time, during which stenosis worsens in such anomalies.⁵ Investigators have concluded that intravascular ultrasonographic imaging of the anomalous cross-sectional area is the most reliable test of R-ACAOS severity: in case of SCA, accurately determining the severity of a given defect is essential.⁵

This patient’s other cardiovascular risk factor was apical HCM, which is more likely than R-ACAOS to cause SCA, ventricular fibrillation, or both, during exertion. In apical HCM, evaluating individual case severity is even more complex and tentative than in R-ACAOS. The typical ECG changes by themselves—had an ECG been performed during precertification screening—should have been an exclusionary criterion for competitive sports in the present case. More severe forms of apical HCM are accompanied by an apical aneurysm, which is typically related to an obstructive muscular ridge in the midventricular cavity.⁶ In such cases, the pressure in the aneurysmal

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cavity can be extremely high because of midventricular stenosis.⁵ Scarring of the apical aneurysm can result from the combination of systolic coronary narrowing or spasm and a simultaneously increased oxygen demand. As Afari and co-authors¹ suggest, dedicated cardiac magnetic resonance studies can clarify details of the apical anatomy, including scarring; however, their published images do not enable such clarification. Selective catheterization might be useful to show patient-specific details of this variable pathologic entity.⁶

The patient apparently experienced no arrhythmias after the insertion of an automatic implantable cardioverter-defibrillator—except when he ignored medical advice to avoid athletic activity. This is evidence that the combination of avoiding competitive exercise, unroofing the anomalous right coronary artery, and prescribing β -blockers constituted adequate treatment. Nevertheless, vigilant follow-up is necessary.

In conclusion, at least in cases involving pathognomonic changes, a simple resting ECG can indeed be life-saving.

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