CLINICAL VIGNETTE

Syncope in a Competitive Athlete

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A 41-year-old elite male water polo player is referred to cardiac electrophysiology for recurrent syncope since high school. He reports years of intermittent loss of consciousness. Episodes occur when rising from a seated or recumbent position and result in loss of postural tone. Witnessed and self-reported descriptions of events report no prodrome, return to normal consciousness within seconds, and no residual symptoms. They specifically deny diaphoresis, pallor, and nausea. A head-up tilt table test was ordered by the patient's primary care provider before the EP consult. The tilt table test shows a resting heart rate of 43, within the normal range for the patient, and BP 103/58. After a 20-minute head-up position at 80 degrees, no significant change occurs, and the head-up position is repeated with adjuvant 400 mcg SL NTG spray, during which there are no symptoms, with the heart rate nadir of 33 bpm with BP 104/71. At EP visit, the patient was asymptomatic with heart rate of 79 bpm, with regular rhythm. Twelve-lead ECG revealed typical CCW atrial flutter with 4:1 ventricular response. A 30-day event monitor was placed, and identified episodes of atrial flutter and atrial fibrillation, including both slow ventricular response and RVR up to 190 bpm. Due to limitations of the type of event monitoring approved by his insurance, it did not report episode duration and arrhythmia burden. The patient was asymptomatic, without awareness of events nor effect on athletic performance. His CHADSVASC score is zero and he was offered rhythm control to prevent progression with either drugs or ablation, with pulmonary vein isolation and cavotricuspid isthmus linear ablation, requiring 90 days postoperative oral anticoagulation. He declined intervention for the arrhythmia and on follow-up, reports resolution of syncope after adhering to increased water and salt intake: 60 oz water/ day QID and 2 grams NaCl TID.

Atrial Fibrillation in Athletes

Atrial fibrillation is a very common fitness-related arrhythmia, especially among endurance athletes. For every 10 years of regular endurance exercise, it is estimated that the risk of atrial fibrillation increases by about 16%. A study of 49 swimmers compared to age-matched controls reported 26.5% of swimmers reported atrial fibrillation compared to 7% of controls.[1]

Factors that contribute to the development of atrial fibrillation in athletes include remodeling of the left atrium, elevated left atrial pressure, inflammation, myocardial fibrosis, and heightened vagal tone. The heightened vagal tone can also increase risk of vasovagal syncope in these patients.[2]

Syncope in Athletes

Syncope is common in athletes, especially swimmers. Data suggests sudden cardiac death can be directly related to cardiac symptoms like syncope.[3] Therefore, adequate screening for and evaluation of syncope is important. A high quality, detailed patient history is essential and may be adequate for diagnosis.[4] Syncope that may otherwise be benign, such as most vasovagal or orthostatic syncope, may be life threatening in swimmers due to drowning risk. Syncope can also be directly life-threatening when it arises from dysrhythmia.

Malignant syncope causes include ventricular fibrillation and long QT syndrome (LQT). Patients with malignant syncope often report loss of consciousness without any identifiable triggers or prodrome of dizziness. Electrocardiographic (ECG) screening may reveal signs of malignant syncope[5] such as: short QT syndrome (SQT); LQT; arrhythmogenic right ventricular cardiomyopathy (ARVC); premature ventricular contractions (PVC); and hypertrophic cardiomyopathy (HCM). These conditions can lead to sudden cardiac death and should be screened manually without relying only on automated ECG analysis.[6] There is a lack of consensus on whether athletes should be required to receive ECGs. Regarding younger athletes, the American Heart Association (AHA) believes that it is not necessary whereas the European Society of Cardiology (ESC) and the International Olympic Committee (IOC) differ. For high level competitive athletes of all ages, ECG screening is highly recommended by all three organizations.[7]

Additional testing may be considered to identify risk of malignant syncope. For example, cardiac MRIs with late gadolinium enhancement (LGE) may identify cardiomyopathy[8] not seen on routine echocardiography. Positron emission tomography (PET) computed tomography (CT) scans can identify active inflammatory and infiltrative processes that may be amenable to intervention.[9] Tilt table tests in combination with the history may identify a neurocardiogenic etiology.

Syncope patients need medical clearance before returning to athletics. Dehydration and electrolyte loss can contribute to exercise-induced syncope. Simple interventions such as increasing fluid and salt intake can be effective.[10] Some studies have recommended up to 10 grams of NaCl and 2-3 liters of water throughout the day.[11] AHA guidelines advise athletes with syncope of unknown cause should not participate in sports where the transient loss of consciousness can be hazardous. They also state that athletes with syncope should have their

medical history taken, a physical examination, an ECG, and use of other tests when there is suspicion of structural heart disease or primary electrical abnormalities. Depending on the results of these tests, athletes who have syncope that is caused by structural heart disease or primary electrical disorders should be restricted from athletic activities according to their condition.[12]

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