Etiologies of Sudden Cardiac Death in National Collegiate Athletic Association Athletes

Running title: Harmon et al.; Etiologies of SCD in NCAA Athletes

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Journal Subject Codes: [8] Epidemiology
Abstract:

**Background** - The etiology of sudden cardiac death (SCD) in college athletes has not been defined by systematic case identification.

**Methods and Results** - 45 cases of SCD were identified in National Collegiate Athletic Association (NCAA) athletes from 2004 – 2008 based on an internal reporting system and review of media reports. Autopsy reports were reviewed and adjudicated by a multi-disciplinary panel. Cause of death could be reasonably determined in 36 cases. 3 athletes had no autopsy, 5 autopsy reports could not be obtained, and 1 autopsy had insufficient information to determine cause of death. The most common finding at death was a structurally normal heart or autopsy-negative sudden unexplained death (11, 31%), followed by coronary artery abnormalities (5, 14%), dilated cardiomyopathy (3, 8%), myocarditis related (3, 8%), aortic dissection (3, 8%), and idiopathic left ventricular hypertrophy (LVH)/possible hypertrophic cardiomyopathy (HCM) (3, 8%). There was one case each (3%) of HCM, arrhythmogenic right ventricular cardiomyopathy, long QT syndrome, commotio cordis, and Kawasaki’s disease. There was one case of death in a sickle cell positive athlete who also had LVH. The adjudicated diagnosis agreed with the official pathology report in only 59% of cases.

**Conclusions** - Unexplained death with a structurally normal heart is the most common finding after suspected SCD in NCAA athletes. HCM is infrequently seen, and conclusions in autopsy reports may not accurately reflect the pathologic findings. Standardized protocols for cardiovascular autopsies in athletes are needed, including post-mortem genetic testing, particularly in autopsy-negative cases.

**Key words:** sudden cardiac death, sudden death, etiology, hypertrophic cardiomyopathy, sudden cardiac death, arrhythmia, athlete
Background

Sudden cardiac death (SCD) is the leading cause of non-traumatic death in NCAA athletes. The most common cause of SCD in athletes in the US is thought to be hypertrophic cardiomyopathy (HCM). In a renowned 1996 study, with subsequent analysis of additional cases in 2009, 36% of deaths were attributed to HCM while only 3% of cases were attributed to autopsy negative sudden unexplained death (AN-SUD). Studies in athletes from other countries and in other age-matched non-competitive athlete populations such as in the US military and in general populations of young people both in the US and abroad have differed with AN-SUD representing a much larger and HCM a relatively smaller proportion of deaths. For instance, in an often cited Italian study, there was only one death from HCM (2%). The differences between the etiology of SCD in US and Italian athletes has been recognized and attributed to the heterogeneous racial diversity of the US population compared to the more homogenous genetic make-up of the Veneto region of Italy or to the effectiveness of screening programs in Italy. However, the discrepancies may also be related to differences in study methodology, case identification, and autopsy protocols. Understanding the etiology of SCD in young athletes is critical as we develop and refine targeted and cost-effective prevention strategies.

The NCAA is comprised of over 400,000 student athletes all who are required to pass a pre-participation screening examination in order to compete. Participation numbers are tracked in each sport including statistics on sex, race, and division of play. This study sought to leverage the unique aspects of this large, contained and well-described population to better understand the causes of SCD in college athletes.
Methods

Deaths were identified between January 1, 2004, and December 31, 2008, using two databases; the NCAA Database and the Parent Heart Watch (PHW) Database. The NCAA database was created from the NCAA Memorial Resolutions List (a list of voluntarily school-reported athlete deaths from any cause) and deaths reported directly to the NCAA. Names may be submitted to the NCAA at any time and each November a solicitation for names of student-athletes who have died is sent to every NCAA member institution (all Divisions) in order to create the Memorial Resolutions. All NCAA athletes are covered by the NCAA Catastrophic Injury Insurance Plan and insurance claims were also reviewed. PHW is a national non-profit organization dedicated to the prevention and awareness of sudden cardiac arrest (SCA) in young people. They have maintained an ongoing database created from systematic searches of media reports since 2000. Deaths among athletes 17 to 24 years of age were queried and each case was reviewed to determine whether the athlete was a member of an NCAA team at the time of death. The NCAA and PHW databases were combined into a single database. A capture-recapture analysis is a statistical technique that can be used when data is collected using two distinct data sets to estimate completeness of a sample and was performed on the combined database.28,29

Missing information regarding deaths was acquired through Internet searches and media reports, e-mails and telephone calls to sports information directors, head athletic trainers, athletic directors, and coroners. When possible, the athlete’s parents or guardians were interviewed. Deaths were classified as cardiovascular-related based on review of all available resources and information. Autopsy reports for all deceased athletes were sought.

Definitions for pathological diagnosis were agreed upon taking into account accepted definitions in the literature23,30-34 and definitions used in other studies (Supplemental Table 1).9.
Autopsy reports were reviewed independently by a panel of experts consisting of three sports medicine physicians, a cardiac pathologist, a cardiomypathy specialist, a genetic cardiologist, and an electrophysiologist all with expertise in SCD in athletes. Differences of opinion were resolved through panel discussion to reach the “adjudicated diagnosis.” The adjudicated diagnoses were compared to the diagnoses published by the media and listed on the official pathology report. This study was approved by the University of Washington Division of Human Subjects.

Results

There were 45 cardiovascular-related sudden deaths during the years 2004 through 2008 for an annual incidence of 1:43,770, the details of which have been reported previously.1 Eighty-seven percent of cases were registered in the NCAA database, while surveillance of media reports collected in the PHW database identified only 56% of cases. Insurance claims identified only 20% of cases. Capture-recapture analysis estimated the number of deaths for that period was 49.1 (confidence interval 45.4 to 50.5) or that the combined database had identified 90-100% of athlete deaths during that time frame.

Cause of death could be reasonably determined in 36 cases (80%). Of the nine cases which could not be determined, three athletes had no autopsy, five autopsy reports could not be obtained, and one autopsy had insufficient information to determine a cause of death. Thirty-two autopsy reports were available for review. One autopsy report had insufficient information to determine cause. A probable cause of death was determined in 5 cases without obtaining the written autopsy report. In three cases, the autopsy report was read over the phone; one congenital coronary artery abnormality, one case of Kawasaki disease, and one case of a grossly
and microscopically normal heart (i.e. AN-SUD) in which additional history was also provided. In another case attributed to long QT syndrome (LQTS), it was determined that two of the decedent’s siblings also had LQTS. There was one case of commotio cordis.

Among the 36 cases (80%) where the cause of death could be reasonably determined (Figure 1), the most common finding was AN-SUD (11, 31%) followed by coronary artery abnormalities (5, 14%), dilated cardiomyopathy (3, 8%), myocarditis related (3, 8%), aortic dissection (3, 8%), and idiopathic left ventricular hypertrophy (LVH)/possible HCM (3, 8%). There was one case each (3%) of HCM, arrhythmogenic cardiomyopathy (ARVC), commotio cordis, and Kawasaki disease. There was one case of death in a sickle cell positive athlete who also had LVH. Toxicology screens were available for all autopsies and were negative or non-contributory.

Twenty-two percent of media reports did not mention a cause of death, and 36% reported a non-specific diagnosis such as “natural causes” or “heart trouble”. Media reports were accurate when reporting congenital coronary artery abnormalities or aortic dissections. The adjudicated diagnosis was in agreement with the official pathology report in only 59% of cases (Supplemental Table 2).

Discussion

AN-SUD is the most common finding in NCAA athletes with SCD while HCM was a much less common cause of death than previously reported in US athletes. This is similar to findings in other countries, in the US military, and in US non-athlete populations, however it differs from the US National Registry of Sudden Death in Athletes (Figure 2).

Several reasons may explain this difference. Our study involved a narrow age range of
highly trained athletes. In contrast, the US Registry includes a wider age range. It is also possible that this discrepancy stems from differences in methodology and case identification. This NCAA study was performed on a well-defined population and aided by an internal reporting system of student-athlete fatalities. While it is possible some cases of SCD were missed, the capture-recapture analysis indicated 90-100% of deaths were identified in this cohort. The US National Registry of Sudden Death in Athletes has collected cases from media reports and other sources for more than 30 years. Registries, by their nature, risk ascertainment bias. In addition autopsy or media-reported diagnoses do not always correlate with adjudicated diagnoses by experts.

There are few studies that examine the etiology of SCD in athletes. In comparing the studies, the classification of “athlete” is inconsistently defined, age ranges are wide, and other than the US National Registry of Sudden Death in Athletes, the number of cases is small. Corrado prospectively followed Italian competitive athletes ages 12-35 where there is mandatory reporting of deaths and a known denominator over twenty years. During that time there were 55 deaths with a 2% incidence of HCM. Suarez-Miller retrospectively reviewed 81 exertional deaths in any type of exerciser in Spain over a fifteen-year period and found a 10% incidence of HCM and a 23% incidence of AN-SUD. De Noronha reported on 89 athlete deaths under the age of 35 who were referred for specialized autopsy with similar results of 12% HCM and 29% AN-SUD. Finally, Holst reviewed 15 exertional deaths in competitive athletes in Denmark over a seven year period and found no cases of HCM and 27% AN-SUD. Thus, consistent with the results of this NCAA cohort, other studies focusing solely on SCD in athletes demonstrate a lower incidence of HCM and a higher incidence of AN-SUD than what was reported in the US National Registry of Sudden Death in Athletes (Table 1).
The U.S. military tracks all deaths through a mandatory reporting system. In a 25 year study of military recruits ages 18-35 there was a 6% incidence of HCM and a 30% incidence of AN-SUD\textsuperscript{12} and in a another study of active military personnel 18-35 there were comparable findings with HCM accounting for 13% of deaths and AN-SUD 41%.\textsuperscript{20} Similar distributions are seen in other studies based on the general population including those not exercising where death certificates, autopsies and population statistics are used for analysis. In the Veneto region of Italy all deaths in young people from 1979-1998 were recorded. There were 273 deaths and standardized post-mortem cardiac evaluations were performed in a prospective manner with medical histories and the circumstances surrounding death recorded.\textsuperscript{15} This study demonstrated a 7% incidence of HCM while apparently normal hearts initially represented 28% (76) of cases.\textsuperscript{15} Findings of a relatively low incidence of HCM and a higher incidence of AN-SUD also occurred in the general population in Australia (HCM 6%; AN-SUD 29%),\textsuperscript{16} England (HCM 5%; AN-SUD 14%),\textsuperscript{35} and Ireland (HCM 15%; AN-SUD 27%).\textsuperscript{19} In a US report from King County, Washington over a 30-year period HCM represented less than 3% of cases in a general population of individuals age 14-35, and only 5% of cases related to exercise.\textsuperscript{14}

The etiologies behind SCD are highly dependent on the age range examined with myocardial infarction and coronary artery disease deaths representing incrementally higher proportions in age groups over 30 years.\textsuperscript{16,17} When considering etiologies of SCD in young people, the upper age limit should be examined carefully to avoid bias of results. Likewise, as the age range is lowered to pre-adolescent ages, etiologies may be skewed toward congenital anomalies. The age range of epidemiologic studies must be taken into consideration when interpreting results. This NCAA study involves a narrow age range of athletes 17-24 years old, which also may account for some differences with the US National Registry of Sudden Death in
Athletes (ages 8-39).

We used accepted pathological definitions in the literature,\textsuperscript{23, 30-34} however, we considered the possibility that differences in HCM definition may account for some of the variability in results between studies. Therefore, the autopsies were re-examined with definitions used to evaluate athlete deaths in the US National Registry of Sudden Death in Athletes.\textsuperscript{9} This did not significantly change results with only one case being re-categorized as HCM. There was one death in an athlete with sickle cell trait, for which it was difficult to definitively adjudicate the cause of death. Athletes with sickle cell trait (SCT) have an increased risk of exertional death.\textsuperscript{37} After death, cells will sickle in athletes with SCT and therefore the presence of sickled cells at autopsy does not indicate that sickling was related to the mechanism of death. Unlike individuals with sickle cell disease who often have cardiac abnormalities, most individuals with SCT have morphologically normal hearts. The athlete in the NCAA cohort with SCT who died met criteria for LVH but did not have any myocardial disarray or any other pathological finding noted on histopathology to be classified as HCM.\textsuperscript{38, 39} The only history available for this athlete was a media report noting that he was known to have prior cardiac disease. A history of sudden collapse would indicate likely SCD while a conscious collapse with a gradual decline is more consistent with an exertional sickling death.\textsuperscript{40} As there was no history obtainable, causality was assigned to both and the case listed as “idiopathic LVH/possible HCM/SCT”.

In this study the quality of the autopsies varied considerably from athlete to athlete. This is the most significant limitation of this investigation and is a limitation of any study based on a retrospective review of autopsy reports. The importance of this study compared to others is the defined population minimizing the possibility of ascertainment bias. In addition, the
methodology to arrive at autopsy diagnosis is more clearly described in this paper than others and the limitations in the autopsies performed are somewhat mitigated by careful review by an expert panel. Although there are still clear limitations to this methodology, other papers that are commonly cited state “we were largely dependent on primary data.” This is a limitation of all current US athlete studies and will only be overcome by more standardized data collection.

The training and expertise of local coroners and medical examiners as well as the protocol used to conduct the autopsy vary by jurisdiction. Autopsy reports typically consist of a gross description of the heart, measurements, and histopathologic description. Measurements of various cardiac features, including the ventricular free walls and interventricular septum, were reported inconsistently in this study. It is possible a more specific cause of death could have been found in some of our cases classified as AN-SUD if a more detailed and comprehensive autopsy was performed. In one Italian study, 28% of deaths were initially categorized as pathologically normal hearts but with more specialized autopsy examination 79% of these (60 of 76) were later determined to have a specific cause. This suggests that, in this population, evaluation by a cardiac pathologist may ultimately result in a more refined diagnosis. We attempted to overcome this limitation by gathering as much contextual information about the case as possible and by assembling a multi-disciplinary expert panel to review and adjudicate each case. We were also unable to obtain autopsy reports or additional information for eight cases (18%), three of which were black, male basketball players. It is conceivable that if these cases were available for review more cases of HCM would have been discovered. Diagnostic criteria for HCM and other cardiomyopathies after death need to be adjusted from those used in living patients. Echocardiographic wall thickness and left ventricular cavity dimensions for the diagnosis of HCM are based on diastolic measurements; however, the heart muscle is often in
rigor (simulated systole) at the time of autopsy. Measurements will vary based on the state of
decomposition of the body and degree of agonal dilatation and wall thinning. Thus, any
interpretation of measurements should take into account the post-mortem interval and
decompositional state of the specimen. Heart weight was reported consistently in all autopsies,
however, inconsistency amongst pathologists in how much of the great vessels are trimmed,
whether or not post-mortem clot is sufficiently removed prior to weighing, and whether or not
the heart is weighed pre- or post-fixation all can add additional variability to these studies. The
histopathology reported in the autopsies varied significantly as well. In many jurisdictions,
microscopy is limited by cost considerations, and one case had no microscopic analysis at all. In
this case, the information in the autopsy was judged to be insufficient to determine cause of
death. Terminology also differed between autopsy reports and it was not clear that enough
sections were obtained or if they were taken from areas of the heart needed to evaluate for
specific conditions.

This study is possibly limited by the number of cases examined. There were 45 cardiac
deaths identified, but for 20% (9) the cause could not be determined either because an autopsy
was not performed, could not be obtained, or had insufficient information to make a diagnosis. It
is possible that the proportion of specific causes of SCD in NCAA athletes could change with a
larger study size. However, the systematic collection method of this data makes it particularly
valuable.

Finally, it is possible that these results may have been skewed because athletes with
structural abnormalities such as HCM were detected by the required preparticipation screening
and restricted from play. However, this possibility would apply to any of the structural or
electrical cardiac disorders that could be discovered by history and physical examination, and not
just HCM. It is unclear if the current screening model adequately identifies athletes with at-risk conditions or could have affected the natural distribution of SCD causes. Most studies investigating screening protocols demonstrate a very low sensitivity to detect conditions associated with SCD by history and physical examination alone. In fact, a 1996 report showed that only 20% of athletes may have prodromal symptoms of cardiovascular disease prior to SCD, and only 1% was properly detected on preparticipation screening.9

The increasing use of post-mortem molecular studies will likely provide additional clarity to questions of diagnosis. Post-mortem genetic testing currently identifies a known gene mutation for an ion channelopathy in approximately 25-35% of cases of AN-SUD.41,42 In addition, genetic testing may elucidate the diagnosis in cases of idiopathic LVH/possible HCM and other cases of cardiomyopathy.41 Standardized autopsy protocols are needed to more accurately determine the causes of SCD in athletes.

Conclusions

AN-SUD is the most common finding on autopsy at death while HCM is less common than previously thought in NCAA athletes. This is analogous to findings in other countries,15-18, 21, 35, 36 in the US military,12, 20 and in US non-athlete populations,14 although it differs from data reported in the US National Registry of Sudden Death in Athletes.6 Methodological differences may account in part for this discrepancy, and additional investigation with higher quality autopsies is needed. It is crucial that standardized autopsy protocols be followed in cases of SCD in young people including post-mortem genetic testing. Autopsies should be performed either locally, when available, or at specialized centers where the knowledge and expertise to carefully evaluate the causality of SCD in the young exists. Centralized data collection with
mandatory reporting of deaths and standardized protocols would significantly improve the quality of information and our understanding of the causes of death in young athletes. The diagnoses from media descriptions are often inaccurate, and even formal autopsy diagnosis may not correctly reflect pathologic findings in the report. A meaningful discussion regarding screening and prevention cannot be engaged without an accurate understanding of the incidence and etiologies of SCD in athletes.

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Conflict of Interest Disclosures: None.

References:


Table 1: Studies of the Etiologies of Sudden Cardiac Death in Young People

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<tr>
<th>Author</th>
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<th>Population</th>
<th>Age Range (years)</th>
<th>Number of Deaths</th>
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<th>Idiopathic LVH/possible HCM</th>
<th>Coronary Artery Abnorm.</th>
<th>ARVC</th>
<th>DCM</th>
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Figure Legends:

**Figure 1:** Etiologies of Sudden Cardiac Death in NCAA Athletes

**Figure 2:** Comparison of Etiologies of Sudden Cardiac Death
Etiologies of Sudden Cardiac Death in National Collegiate Athletic Association Athletes
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Supplemental Table 1: Definitions for Pathologic Diagnosis of Sudden Cardiac Death in Athletes

**Autopsy negative sudden unexplained death**
- Normal heart pathologically
- No obvious explanation for death
- Presumed arrhythmia

**Hypertrophic cardiomyopathy**
- Heart weight ≥ 50% of the expected mean weight based on gender, age, and body size for weight or height (using the Mayo nomograms) plus at least one of the following:
  - Histologic myocyte disarray
  - Septal mitral valve contact lesion (implying systolic anterior motion of the anterior mitral valve leaflet)
  - Asymmetric LVH, particularly ventricular septal – left ventricular free wall ratio ≥ 1.3 (note: symmetric and apical variants will typically have normal ratios)
- Significant (>75% of the area of a section) myocyte disarray in a basal or mid-ventricular section but not meeting weight criteria

**Idiopathic LVH/ possible HCM**
- Heart weight ≥ 50% of the expected mean weight based on gender, age, and body size for weight or height (using the Mayo nomograms) without myocyte disarray and
  - Non-dilated LV chamber
- Heart weight does not meet weight criteria for HCM or idiopathic LVH/ possible HCM using Mayo nomograms but
  - Pathologic features are suggestive of HCM (i.e. outflow tract obstruction, LVH > 16 mm)
  - No/minimal myocyte disarray
  - Non-dilated LV chamber

**Arrhythmogenic right ventricular cardiomyopathy**
- Gross fibrofatty replacement of right ventricular free wall (excluding anterior RV in older individuals)
- The fatty change appears “infiltrative” with a perpendicular pattern with respect to the epicardial surface
- Variable degrees of fibrosis, vacuolization, and/or lymphocytic myocarditis

**Dilated cardiomyopathy**
- Heart weight ≥ 50% of the expected mean weight based on gender, age, and body size for weight or height (using the Mayo nomograms) without myocyte disarray and
  - LV wall < 10 mm
  - LV chamber diameter (at the mid-ventricular level, excluding trabeculations) > 3.0 cm (note: agonal dilatation should be excluded by examining for cell separation and other post-mortem artifact histologically)
    - If absolute chamber diameter not measured, then comments about gross chamber dilatation (without agonal dilatation from autolysis)
  - Histologically, myocyte hypertrophy with variable interstitial fibrosis (usually pericellular-type)

**Myocarditis related**
- Active lymphocytic myocarditis
  - Inflammatory infiltrates of the myocardium with associated myocyte injury/necrosis
- Borderline myocarditis
  - Inflammatory infiltrates of the myocardium without associated myocyte injury/necrosis
- Healed myocarditis

**Coronary artery abnormalities**
- Coronary artery anomalies
- Myocardial bridging
- Tunneled coronary arteries
- Coronary artery dissections

**Coronary artery disease**
- Atherosclerotic coronary arteries with > 70% lumen occlusion and
  - More likely than not this was primary cause of death

**Commotio cordis**
- SCD after blunt trauma to the chest
- No other cardiac pathology
## Supplemental Table 2: Differences in Causes of Death

<table>
<thead>
<tr>
<th>SPORT</th>
<th>Cause stated in media report</th>
<th>Cause stated in official autopsy report</th>
<th>Adjudicated cause</th>
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<td>No autopsy</td>
<td>Commotio cordis</td>
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<tr>
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<td>HCM</td>
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<tr>
<td>Basketball</td>
<td>Anomalous coronary</td>
<td>Anomalous coronary (left coronary)</td>
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<tr>
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<td>Aortic dissection</td>
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<tr>
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<td>Aortic dissection</td>
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<tr>
<td>Basketball</td>
<td>HCM</td>
<td>DCM</td>
<td>DCM</td>
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<tr>
<td>Basketball</td>
<td>Cardiomyopathy</td>
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<tr>
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<td>Cardiomegaly with LVH</td>
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<td>Microscopic cardiomyopathy with cardiomegaly</td>
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<tr>
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<td>SUD</td>
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<td>No cause</td>
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<tr>
<td>Crew</td>
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<td>Fluid and electrolyte imbalance</td>
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<tr>
<td>Crew</td>
<td>Heart trouble</td>
<td>No autopsy</td>
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<td>X-country</td>
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<td>Anomalous coronary (left coronary)</td>
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<td>X-country</td>
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<td>Football</td>
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<td>Global ischemic necrosis of the Myocardium without atherosclerosis</td>
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<td>Dehydration and heat exposure with coronary atherosclerosis and cardiomegaly</td>
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