Section: Cardiology

Original Research Article

SUDDEN CARDIAC DEATH IN THE YOUNG: UNMASKING SILENT CARDIOVASCULAR DISORDERS IN APPARENTLY HEALTHY INDIVIDUALS - A SYSTEMATIC REVIEW OF THE HIDDEN EPIDEMIC

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ABSTRACT

Background: Sudden cardiac death (SCD) in young individuals is a rare but devastating event, often occurring in apparently healthy individuals without prior diagnosis of cardiovascular disease. Unlike older populations, where coronary artery disease predominates, SCD in individuals under 35 years is primarily associated with inherited structural and electrical cardiac abnormalities.[1,2]

Materials and Methods: A systematic review was conducted in accordance with PRISMA guidelines. Electronic databases including PubMed/MEDLINE, Embase, Scopus, Web of Science, and the Cochrane Library were searched from inception to the present. Studies evaluating SCD in individuals under 35 years without known cardiac disease were included. Data extraction focused on incidence, etiology, clinical presentation, risk factors, and prevention strategies. Study quality was assessed using NIH tools for observational studies and AMSTAR criteria for systematic reviews.

Results: A total of 3,987 records were identified, of which 12 studies met inclusion criteria after screening. The incidence of SCD ranged from 0.3 to 3.6 per 100,000 individuals annually, with a marked male predominance[2,3]. Cardiomyopathies and inherited channelopathies were the leading causes,[1,4] while a significant proportion of cases remained unexplained after autopsy. Exercise and acute physiological stress were common triggers among athletes.[3,5] Screening strategies, particularly electrocardiography, demonstrated variable effectiveness across populations, with some studies showing a reduction in mortality following structured screening programs [6-8]. Emerging tools such as molecular autopsy and genetic testing showed promise in identifying underlying etiologies.[1,9]

Conclusion: SCD in young individuals is most commonly due to undiagnosed inherited cardiac disorders. Although incidence is low, the societal impact is substantial. Improved awareness, targeted screening strategies, and advances in genetic diagnostics are essential for early identification and prevention.[2,7] Further research is required to optimize screening protocols and reduce mortality in this population.

Keywords: sudden cardiac death, cardiomyopathies, screening, young adults, athletes, arrhythmia, exercise-induced, channelopathies.

INTRODUCTION

Sudden cardiac death (SCD) is a major global health concern, accounting for approximately 15–20% of all deaths worldwide.[1,2] Although predominantly associated with older adults, SCD in young healthy individuals—typically defined as those under 35 years of age—is particularly alarming due to its unexpected occurrence and devastating consequences.

Unlike older populations where coronary artery disease is the primary cause, SCD in younger

individuals is mainly attributed to inherited and structural cardiac abnormalities, including cardiomyopathies and channelopathies.[1,2] In many cases, SCD is the first manifestation of underlying disease, making early detection challenging.

Athletes represent a unique subgroup in whom the risk of SCD may be increased due to the interaction between underlying cardiac abnormalities and intense physical exertion.[3,4] Although the absolute incidence remains low, the visibility and societal impact of such events are substantial.

This systematic review aims to provide a comprehensive synthesis of current evidence on SCD in young healthy individuals, focusing on epidemiology, etiology, pathophysiology, clinical presentation, screening strategies, and prevention approaches, with an emphasis on improving early identification and reducing mortality.

Objective

This review aims to critically evaluate existing literature on sudden cardiac death in young healthy individuals, focusing on incidence, genetic and structural causes, clinical presentation, screening strategies, and emerging preventive interventions, to improve early identification and reduce mortality in this population.

MATERIALS AND METHODS

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.

Search Strategy

A comprehensive literature search was performed across multiple electronic databases, including PubMed/MEDLINE, Embase, Scopus, Web of Science, and the Cochrane Library, from database inception to March 2026; additional sources included gray literature (Google Scholar) and major cardiology society guidelines.

The search strategy incorporated combinations of Medical Subject Headings (MeSH) terms and keywords, including the following:

("sudden cardiac death" OR "SCD") AND ("young adults" OR "adolescents" OR "athletes") AND ("cardiomyopathy" OR "channelopathy" OR "arrhythmia" OR "screening" OR "genetics").

Reference lists of relevant articles were manually screened to identify additional eligible studies.

Eligibility Criteria

Studies were included if they met the following criteria:

- Population: Individuals aged <35 years
- Condition: Sudden cardiac death or aborted cardiac arrest
- Focus: Structural or inherited cardiac causes in previously undiagnosed or apparently healthy individuals
- Study design: Prospective or retrospective cohorts, registries, systematic reviews, and clinical guidelines

Exclusion criteria included

- Studies involving individuals ≥35 years
- Non-cardiac or traumatic causes of sudden death
- Animal studies
- Case reports with insufficient clinical detail

No language restrictions were applied.

Study Selection

A total of 3,987 records were identified through database and manual searches. After removal of duplicates (n = 1,014), 2,973 records were screened based on title and abstract. Of these, 312 full-text articles were assessed for eligibility. Ultimately, 12 studies met inclusion criteria and were included in the qualitative synthesis.

Two reviewers independently performed study selection, with disagreements resolved through consensus.

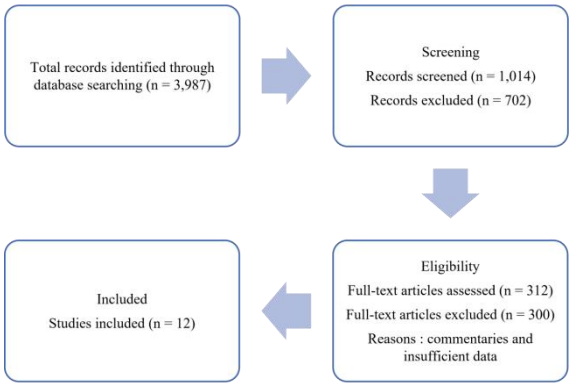

Figure 1: PRISMA flowchart of study selection. Database and gray-literature searches identified several thousand records. After duplicate removal and screening, 12 studies met the inclusion criteria for qualitative analysis

Data Extraction

Data were extracted using a standardized form, including:

- Study design and setting
- Population characteristics
- Sample size
- Definitions of SCD
- Incidence rates
- Etiology and risk factors
- Key outcomes and conclusions

Quality Assessment and Risk of Bias

The methodological quality of included studies was assessed using the National Institutes of Health (NIH) quality assessment tools for observational cohort and cross-sectional studies. Systematic reviews were evaluated using the AMSTAR (A Measurement Tool to Assess Systematic Reviews) criteria.

Studies were categorized as high, moderate, or low quality based on risk of bias domains, including selection bias, confounding, and outcome assessment.

Due to heterogeneity in study design, populations, and outcome definitions, a meta-analysis was not performed, and findings were synthesized qualitatively.

Study (Author, Year)	Study Design	Bias Tool Used	Selection Bias	Confounding	Outcome Assessment	Overall Quality
Bagnall et al., 2016	Prospective cohort	NIH	Low	Moderate	Low	High
Couper et al. (2020)	Systematic review	AMSTAR	Low	Moderate	Moderate	High
Ha et al. (2020)	Nationwide cohort	NIH	Low	Moderate	Low	High
Harmon et al., 2015	Registry	NIH	Moderate	Moderate	Moderate	Moderate
Corrado et al., 2006	Cohort	NIH	Low	Moderate	Low	High
Maron et al., 2009	Cohort	NIH	Moderate	Moderate	Moderate	Moderate
Finocchiaro et al., 2016	Cohort	NIH	Low	Moderate	Moderate	High
Eckart et al., 2011	Cohort	NIH	Moderate	Moderate	Moderate	Moderate
Winkel et al., 2011	Nationwide cohort	NIH	Low	Moderate	Moderate	High
Behr et al. (2007)	Cohort	NIH	Moderate	Moderate	High	Moderate
Semsarian et al., 2015	Review	AMSTAR	Moderate	Moderate	Moderate	Moderate
Ackerman et al. (2016)	Review	AMSTAR	Moderate	Moderate	Moderate	Moderate

Most studies were assessed using the NIH quality assessment tool, while systematic reviews were evaluated using AMSTAR criteria. Overall, the included studies demonstrated moderate to high methodological quality, with stronger evidence derived from large prospective and nationwide cohort studies.

The most common limitation across studies was moderate confounding, reflecting variability in population characteristics and incomplete adjustment for risk factors. Outcome assessment was generally reliable in prospective cohorts but more variable in retrospective and autopsy-based studies.

RESULTS

Table 1: Summary of Selected Key Studies on SCD in Youth

No.	Author (Year)	Country	Study Type	Population	Sample Size	Key Findings	Quality
1	Bagnall et al. (2016)	Australia/New Zealand	Prospective cohort	1–35 yrs	490 cases	Incidence 1.3/100,000; strong genetic contribution	High
2	Couper et al. (2020)	Global	Systematic review	Young population	NA	Incidence ~1–2/100,000; variability across regions	High
3	Ha et al. (2020)	Korea	Nationwide cohort	Young adults	>10,000	Incidence 0.9–1.5/100,000; male predominance	High
4	Maron et al. (2009)	USA	Cohort	Athletes	1,866 deaths	HCM leading cause of athlete SCD	Moderate
5	Harmon et al. (2015)	USA	Registry	NCAA athletes	4,242,519 athlete-years	Higher incidence in male athletes	Moderate
6	Corrado et al. (2006)	Italy	Cohort	Athletes	Large national cohort	ECG screening reduced SCD by ~89%	High
7	Semsarian et al. (2015)	Australia	Review	Young individuals	NA	Genetic basis significant in SCD	Moderate
8	Ackerman et al. (2016)	USA	Review	Channelopathies	NA	Ion channel disorders major cause	Moderate
9	Behr et al. (2007)	UK	Cohort	Unexplained SCD	117 cases	High rate of SADS	Moderate
10	Winkel et al. (2011)	Denmark	Nationwide cohort	Young population	6,629 deaths	High unexplained SCD proportion	High
11	Finocchiaro et al. (2016)	UK	Cohort	Athletes	357 cases	Cardiomyopathies predominant	High
12	Eckart et al. (2011)	USA	Cohort	Military recruits	6.3 million	Higher incidence in males	Moderate

DISCUSSION

Epidemiology: Sudden cardiac death (SCD) in young individuals is an uncommon but catastrophic event, with reported incidence ranging from approximately 0.3 to 3.6 per 100,000 persons annually across different populations [2,3]. Variability in incidence reflects differences in study design, case definitions, and population characteristics.

A large prospective study by Bagnall et al. reported an incidence of 1.3 per 100,000 individuals aged 1–35 years, with a marked male predominance

(~70%).[2] Similar findings have been observed in other cohort and registry-based studies, consistently demonstrating a higher risk in males compared to females.[3]

Among athletes, the incidence is estimated at approximately 0.5–1.0 per 100,000 athlete-years, although certain subgroups, such as male collegiate basketball players, exhibit substantially higher rates.[3,4] While physical exertion is recognized as an important trigger, a considerable proportion of SCD events also occur at rest or during sleep.[4]

Etiology and Pathophysiology

The etiology of SCD in the young is multifactorial and can be broadly categorized into structural heart diseases, primary electrical disorders, and acquired conditions.

Structural Heart Disease

Inherited cardiomyopathies are among the leading causes. Hypertrophic cardiomyopathy (HCM) is often cited; in athletic-field autopsy series, HCM accounts for ~36% of SCD in U.S. athletes.[3,4] Arrhythmogenic right ventricular cardiomyopathy (ARVC) and dilated cardiomyopathy (DCM) are also implicated.[4] Congenital coronary anomalies (e.g., anomalous origin of a coronary artery) are important, especially under age 35.[4] Connective-tissue diseases (e.g., Marfan) and myocarditis (often viral or immune-mediated) can cause fatal events.[4]

Importantly, SADS (sudden arrhythmic death syndrome)—defined as SCD with a structurally normal heart – is very common. Bagnall et al. found 40% of young SCDs were "unexplained" after complete autopsy. In other cohorts, the majority of SCD victims under 35 have no gross pathology. Studies have shown that a large proportion of young SCD cases remain unexplained even after comprehensive autopsy.[1,2]

Primary Electrical Disorders

Primary electrical (channelopathy) conditions account for many unexplained SCDs. Long QT syndrome, Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia, and Wolff-Parkinson-White syndrome are well-known genetic syndromes that cause ventricular arrhythmias in young individuals.[1] Molecular autopsy studies show that among autopsy-negative SCD cases, 25–30% have pathogenic variants in cardiac ion channel or cardiomyopathy genes.[1,9] For example, Bagnall et al. prospectively found clinically relevant gene mutations in 27% of 113 unexplained SCD cases. These inherited defects often have variable penetrance, so victims may have been asymptomatic until the fatal event.

Other Causes and Triggers

A portion of SCDs in young adults is due to acquired conditions: coronary artery disease (especially familial or early atherosclerosis) accounted for ~24% of SCDs in Bagnall's series, predominantly in the oldest subgroup (31–35 years).[2] Myocarditis (including from recent viral illness) can cause electrical instability.[4]

Acute triggers play a critical role in precipitating SCD in susceptible individuals. Strenuous physical exertion and emotional stress are well-recognized triggers.[4] Illicit drug use has also been implicated; registry-based data indicate that a significant proportion of young SCD victims have evidence of substance use, which may increase arrhythmic risk.[9] Additionally, medications that prolong the QT interval may unmask underlying susceptibility to arrhythmias. Commotio cordis, resulting from blunt chest trauma, represents a rare but recognized cause in athletic settings.[4]

In summary, most SCDs in the young stem from an underlying substrate (genetic or structural) that is often undiagnosed, with an acute trigger initiating a lethal ventricular arrhythmia.

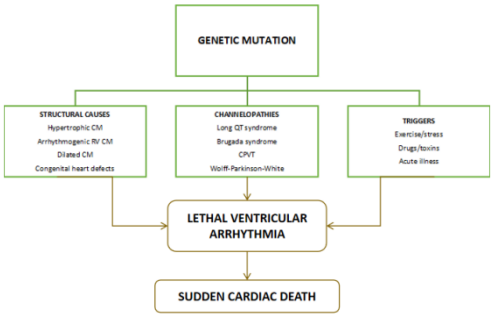

Figure 2: Mechanisms of sudden cardiac death in the young. Genetic substrates (cardiomyopathies and channelopathies) create vulnerability; acute triggers (exercise, drugs, illness) precipitate lethal arrhythmia, resulting in SCD

Clinical presentation

SCD often occurs without warning; however, up to half of affected individuals may experience preceding symptoms. Common warning signs include syncope particularly during exertion, palpitations, chest pain, dyspnea, and seizure-like episodes (often misdiagnosed).[2,4] Recognition of these symptoms is critical for early intervention.

Major risk factors include family history of SCD, known cardiomyopathy or channelopathy, previous unexplained syncope and documented arrhythmias.[1,2] Lifestyle and environmental factors such as intense physical exertion, recent viral illness, and substance use (including alcohol and illicit drugs) further contribute to increased risk.[4,9]

Sudden Cardiac Death in Athletes

Athletes are at increased relative risk of SCD due to the interaction between underlying cardiac abnormalities and physiological stress induced by exercise.[3,4] Physical exertion may act as a trigger for fatal ventricular arrhythmias in predisposed individuals. The most common underlying causes in athletes include hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy (ARVC), and congenital coronary artery anomalies.[3,4]

Pre-participation screening programs have been implemented in several countries with varying degrees of success, aiming to identify individuals at risk before participation in competitive sports.[5-7]

A substantial proportion of SCD cases remain unexplained after conventional autopsy. These cases are often attributed to inherited channelopathies.[1] Molecular autopsy, involving postmortem genetic testing, has emerged as a critical tool for identifying underlying genetic mutations, facilitating cascade screening of family members, and preventing further deaths in affected families.[1,9]

Screening and Prevention

Prevention of SCD hinges on early detection of at-risk individuals and mitigation of triggers. Pre-participation screening (PPS) is widely implemented for competitive athletes. All major cardiology and sports bodies (AHA/ACC, ESC, IOC, FIFA, etc.) recommend at least a focused personal/family history and physical exam before sports participation.[5,7] These include questions about syncope, chest pain, familial SCD, and examination for murmurs or physical stigmata (e.g., Marfan habitus).

The addition of a 12-lead electrocardiogram (ECG) to screening remains a subject of ongoing debate. European screening programs, particularly in Italy, have incorporated mandatory ECG screening and reported significant reductions in SCD incidence among athletes.[6] For example, Corrado et al. demonstrated a substantial decline in SCD following the implementation of a nationwide ECG-based screening program, largely through the detection of previously unrecognized cardiomyopathies.[6] These findings contributed to the endorsement of ECG-inclusive screening by several international bodies.[5] However, U.S. guidelines (AHA/ACC) have not required ECG; their consensus (1996–2015) has been that universal ECG screening lacks proven mortality benefit.[7,8] Notably, a large Israeli study found mandatory ECGs did not change SCD rates. Current ACC/AHA guidance (2025 update) suggests ECG may be considered (Class IIb) in select young adults with expert interpretation available. Studies evaluating ECG screening across different populations have shown variable outcomes, reflecting differences in methodology, interpretation criteria, and healthcare infrastructure.[7,8]

Evidence on ECG efficacy is mixed. Modern ECG criteria for athletes (Seattle, International) have improved specificity, reducing false positives to ~3%.[5] The Illinois and European screening programs routinely disqualify a small percentage of athletes (~2%) for cardiomyopathy or channelopathy based on ECG findings.[5] However, no randomized trials exist to prove screening reduces overall SCD mortality. The value of echocardiography or advanced imaging for universal screening is unproven and generally reserved for further evaluation of abnormal initial findings. Familial screening after a SCD event (cascade testing) is now standard, given that genetic findings in victims can identify at-risk relatives[1,9]

Table 2: Screening modalities and their limitations

Tool	Strength	Limitation
ECG	Detects electrical disorders	False positives
Echocardiography	Structural evaluation	Operator-dependent
MRI	High sensitivity	Expensive
Genetic testing	Identifies inherited risk	Ethical concerns
Holter monitor	Detects intermittent arrhythmias	Limited duration

Overall, structured screening (including history, physical examination, and electrocardiography) is widely endorsed; however, its full impact on mortality reduction remains uncertain.[5,7,8] Prevention strategies also include appropriate management of identified conditions, such as beta-blocker therapy in long QT syndrome, lifestyle counseling, and avoidance of QT-prolonging medications, along with ensuring safe environments through the availability of automated external defibrillators (AEDs) at sporting and public events.[1,5]

Management of Cardiac Arrest

Immediate emergency response is critical in cases of sudden cardiac arrest. Young SCD victims often have ventricular fibrillation as the initial rhythm. Survival depends on rapid CPR (cardiopulmonary resuscitation) and early defibrillation.[10] In community and school settings equipped with AEDs, survival after SCD in young people can exceed 50–85%.[10] Guidelines emphasize bystander training in CPR, public access to AEDs, and coordinated emergency medical services.[5]

Post-resuscitation care follows standard ACLS protocols. For survivors of cardiac arrest, comprehensive evaluation (including ECG, imaging, and often genetic testing) is mandated to identify the cause and guide family screening.[1,9] High-risk survivors (e.g., HCM with risk factors) may receive ICD implantation per standard guidelines (e.g., ACC/AHA/ESC recommendations on HCM, cardiomyopathies, LQTS, Brugada) to prevent recurrence.[1]

Medicolegal and Ethical Issues

Screening young people raises ethical and legal questions. Mandatory screening has led to debates over cost-effectiveness, potential anxiety, and false reassurance.[7,8] Some argue that detecting even one life-threatening condition justifies screening; others counter that many deaths may not be preventable by screening and resources could be better spent on AED programs and CPR training.[7]

Ethical issues include informed consent (especially in minors), confidentiality, and how to manage affected individuals. For example, an asymptomatic teen discovered to have HCM may face disqualification from sports, insurance issues, or emotional distress. There is also concern about genetic privacy: families might learn of inherited risks that impact insurability or employment.[1,9]

Medico-legal liability can arise if physicians fail to follow screening recommendations or if an identified at-risk individual later experiences SCD. Conversely, overscreening might lead to unnecessary restrictions. Current guidelines attempt to balance these by promoting shared decision-making and focusing on high-yield screening.[5,7]

Limitations

This systematic review has several limitations. First, significant heterogeneity existed among the included studies in terms of population characteristics, definitions of sudden cardiac death, and study methodologies, which limited direct comparability of findings.[2,7] Second, the absence of randomized controlled trials and reliance on observational and registry-based data may introduce inherent biases, including selection and reporting bias.[2]

Third, although a comprehensive search strategy was employed, the possibility of publication bias cannot be excluded, particularly given the tendency to report more severe or clinically significant cases.[7,8] Fourth, variability in autopsy practices and the underutilization of molecular autopsy in certain regions may have led to underestimation of genetic causes.[1,9]

Finally, due to heterogeneity in study design and outcome reporting, a quantitative meta-analysis was not performed, which limits the ability to derive pooled incidence estimates.[2,7] These factors should be considered when interpreting the findings of this review.

Gaps and Future Directions

Despite advances in understanding sudden cardiac death in the young, several important gaps remain. There is a lack of large-scale, prospective, and randomized studies evaluating the effectiveness of screening strategies, particularly the role of electrocardiography in reducing mortality.[7,8] Standardization of definitions and reporting criteria for SCD is also needed to improve comparability across studies.[2]

Molecular autopsy has emerged as a valuable tool in cases of unexplained sudden death, with studies demonstrating a diagnostic yield of up to 30% in identifying pathogenic genetic variants.[1,9] Wider implementation of postmortem genetic testing, along with structured family screening programs, may significantly improve detection of inherited cardiac conditions.[1]

Technological innovations offer promising avenues for early detection and prevention. Artificial intelligence (AI)-assisted electrocardiogram interpretation has shown potential in improving diagnostic accuracy and enhancing detection of arrhythmias using wearable devices.[5] Recent studies demonstrate that AI-enabled analysis of smartwatch ECG recordings can significantly improve arrhythmia detection compared to conventional methods.[5] Furthermore, wearable smartwatches have demonstrated high sensitivity and specificity in detecting cardiac arrhythmias, supporting their role as scalable screening tools in large populations.[5]

Additionally, wearable cardiac monitoring devices, including smartwatches and portable ECG systems, may facilitate continuous and early detection of subclinical arrhythmias in asymptomatic individuals, potentially enabling earlier intervention.[5]

Further research is needed to evaluate the cost-effectiveness, feasibility, and long-term outcomes of these emerging strategies, particularly in diverse populations and low-resource settings.[7] Development of personalized risk stratification models integrating clinical, genetic, and imaging data may represent a future direction in preventing sudden cardiac death in the young.[1,2]

CONCLUSION

Sudden cardiac death in young healthy individuals is most often due to inherited cardiac abnormalities unmasked by exercise or other stressors.[1,2] The overall incidence is low, but the clinical and societal impact is high. Clinicians should maintain a high index of suspicion: obtain thorough personal and family histories about syncope or early cardiac deaths, perform careful physical exams, and consider ECG screening in athletes or those with a family history of SCD.[5,7] When SCD occurs, immediate CPR and defibrillation are paramount for survival.[10] Guidelines recommend history and physical examination for all young athletes. The addition of ECG should be considered in high-risk settings, with awareness of false-positive findings.[5,7] Identifying a high-risk condition warrants appropriate restriction from high-intensity sports and initiation of therapy (e.g., beta-blockers, ICD).[1] Family members of a victim should undergo genetic counseling and testing when available, given the inherited nature of many underlying conditions.[1,9]

Integration of AI with wearable ECG platforms may represent a paradigm shift in population-level cardiovascular screening.

Finally, strengthening emergency preparedness through training of responders and widespread availability of automated external defibrillators (AEDs) in public and school settings is essential to improving survival outcomes.[10] While screening strategies continue to be debated, clinicians and policymakers must balance potential benefits and limitations, with emphasis on increasing awareness of SCD risk factors, ensuring rapid response systems, and supporting ongoing research to address existing knowledge gaps.[7,8]

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