Sudden cardiac arrest in young adults: A comprehensive systematic review of epidemiology, aetiology and preventive strategies

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Background: Sudden cardiac arrest (SCA) in young adults (18–40 years) is rare but devastating, often occurring without prior symptoms. It often occurs in the absence of preceding symptoms or known structural heart disease. A comprehensive and systematic evaluation of its multifactorial aetiology, diagnostic challenges and preventive strategies is fundamental to reducing morbidity and mortality in this population. **Objective:** This systematic review aims to comprehensively evaluate the epidemiology, underlying aetiologies, associated risk factors, diagnostic approaches and preventive strategies for SCA in adults aged 18–40 years. **Methods:** PubMed, Scopus, Embase, Web of Science and Google Scholar were searched from January 2015 to June 2025. Eligible studies examined aetiologic mechanisms, screening strategies, clinical outcomes or public health interventions targeting the prevention, detection or management of SCA in individuals aged 18–40 years. Study quality was assessed using the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 checklist, the Risk of Bias in Nonrandomized Studies of Exposures (ROBINS-E), A Measurement Tool to Assess Systematic Reviews 2 (AMSTAR 2) and the Newcastle–Ottawa Scale (NOS). **Results:** Of 3,000 records identified through the literature search, 55 studies met the inclusion criteria. The reported incidence of SCA among young adults ranged from 1 to 2 per 100,000 person-years in Western countries, with higher incidence rates observed in Asian populations. Structural cardiomyopathies were the predominant aetiologies, most notably hypertrophic cardiomyopathy and arrhythmogenic right ventricular cardiomyopathy. These were followed by inherited channelopathies, including long QT syndrome, Brugada syndrome and catecholaminergic polymorphic ventricular tachycardia, as well as acquired conditions such as myocarditis and substance-related cardiac toxicity involving cocaine, amphetamines and anabolic steroids. Additional risk factors included systemic comorbidities, particularly sarcoidosis, chronic kidney disease and autonomic dysfunction. Diagnostic evaluation most frequently incorporated electrocardiography, transthoracic echocardiography, cardiac magnetic resonance imaging and genetic testing. Survival following out-of-hospital cardiac arrest was significantly improved in settings with prompt bystander cardiopulmonary resuscitation, widespread availability of automated external defibrillators and the implementation of community-based education initiatives. **Conclusion:** Targeted screening strategies, improved access to advanced diagnostic modalities and population-level community interventions are essential for reducing the burden of SCA among young adult individuals. Further prospective research is warranted to enhance risk stratification and optimize prevention strategies in this high-impact population.

Keywords

Cardiomyopathy, cardiopulmonary resuscitation, channelopathy, substance abuse, sudden cardiac arrest, sudden cardiac death, young adults

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Sudden cardiac arrest (SCA) in young adults (typically defined as individuals younger than 40 years) has emerged as an important yet under-recognized public health concern. Recent estimates suggest an incidence of approximately 1–4 cases per 100,000 person-years, underscoring the clinical and epidemiological relevance of SCA in this younger population.[1]

SCA is a life-threatening medical emergency characterized by the abrupt cessation of effective cardiac activity, resulting in haemodynamic collapse, apnoea and loss of consciousness. Without immediate intervention, SCA rapidly progresses to sudden cardiac death (SCD).[2] Despite substantial advances in resuscitation strategies and cardiovascular care, SCD remains a leading cause of mortality worldwide. It affects individuals across all age groups and exhibits pronounced regional and socioeconomic disparities in incidence, prevention and survival outcomes.[2,3] These persistent inequalities underscore the urgent need for integrated, multidisciplinary strategies and robust system-level interventions to meaningfully reduce the global burden of SCD.[3]

In the USA, more than 356,000 cases of out-of-hospital cardiac arrest (OHCA) occur annually.[4,5] Although advances in emergency cardiovascular care and widespread cardiopulmonary resuscitation (CPR) training have been achieved, survival to hospital discharge remains below 10%. While the incidence of SCA among young adults is lower than that in older populations, its consequences are disproportionately devastating, as it affects individuals in their most productive and physically active years who are often otherwise healthy. Recent data from South Korea report SCA incidence rates of 12.3 per 100,000 among those aged 20–29 and 17.7 per 100,000 among those aged 30–39, including both in-hospital and community cases.[6] Population-based registries from China and Japan have also demonstrated growing recognition of SCA in this age group, with higher rates in males and urban residents; however, the true incidence may be underestimated because of incomplete reporting.[7,8] In Iran, a multicentre analysis showed that 8.5% of cardiac arrests occurred in individuals under 40 years of age, a situation worsened by delayed response times and limited bystander CPR.[9] Similarly, studies from India have attributed over 40% of premature cardiovascular mortality due to myocardial infarction (MI) and stroke to delayed hospital presentation, limited access to specialized emergency care and low rates of CPR by laypersons.[10]

These findings underscore that SCA in young adults represents a global health challenge extending beyond Western countries. The aetiologic spectrum differs substantially from that in older adults, among whom coronary artery disease (CAD) predominates. In younger individuals, SCA is more often associated with inherited or structural cardiac disorders, such as hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy (ARVC), congenital coronary artery anomalies and ion channelopathies, including long QT syndrome (LQTS), Brugada syndrome (BrS) and catecholaminergic polymorphic ventricular tachycardia (CPVT).[11–13] Additionally, external factors such as substance abuse (e.g. cocaine, amphetamines) and metabolic conditions, which are also emerging risk factors for heart failure (HF) in younger populations, contribute significantly to SCA risk.[14] A prospective 4-year study in the USA identified previously unrecognized inherited cardiac diseases as major causes of SCA among young athletes, many of which could be detected through structured preparticipation cardiovascular screening.[15] However, systematic screening of asymptomatic individuals remains inconsistent and controversial across regions, resulting in missed opportunities for early prevention. Socioeconomic disparities and limited emergency infrastructure further influence outcomes; the US data show poorer survival in rural areas due to delayed defibrillation and restricted access to automated external defibrillators (AEDs).[16] Similar challenges are observed across Asia, where limited AED availability and insufficient public CPR training contribute to low post-arrest survival.[17]

Advances in diagnostic and risk stratification strategies, including electrocardiography (ECG), genetic testing and cardiac imaging, have enhanced the early identification of individuals at increased risk. Recent major guidelines, notably the 2022 European Society of Cardiology (ESC) Guidelines on the management of ventricular arrhythmias (VAs) and the prevention of SCD, together with relevant American Heart Association (AHA) scientific statements, strongly endorse the systematic use of these tools to reduce the incidence of SCA, particularly among athletes and individuals with a positive family history (FH).[2,18]

Emerging biomarkers, including proteinuria and myocardial fibrosis, have demonstrated potential utility for identifying subclinical cardiovascular risk in asymptomatic individuals.[19] The coronavirus disease 2019 (COVID-19) pandemic has illustrated the vulnerability of young adults to sudden cardiac events. Current evidence does not indicate a causal association between COVID-19 vaccination and an increased incidence of SCA; however, indirect factors such as reduced physical activity, psychological stress and delayed healthcare access may have contributed to elevated cardiovascular risk.[20] This systematic review aims to synthesize contemporary evidence on the epidemiology, aetiological mechanisms, risk factors, diagnostic modalities and preventive strategies for SCA in adults aged 18–40 years, with the goal of informing both clinical practice and public health policy.

Materials and methods

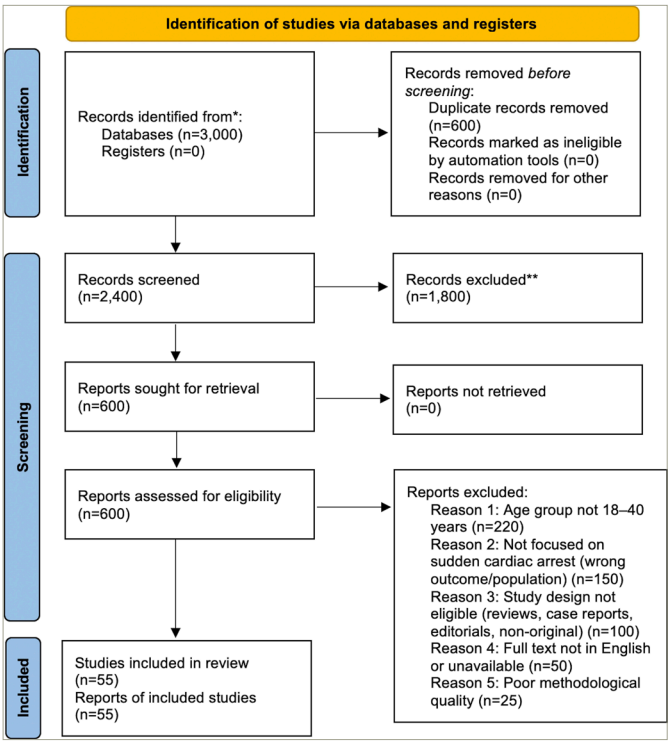
Search strategy and data sources

A comprehensive and systematic literature search was conducted across five major electronic databases: PubMed (MEDLINE), Scopus, Embase, Web of Science and Google Scholar. The search spanned the period from January 2015 to June 2025 and aimed to identify studies examining the epidemiology, aetiological mechanisms, risk factors, diagnostic approaches and preventive strategies for SCA in adults aged 18–40 years. A combination of Medical Subject Headings (MeSH) and free-text keywords was employed to maximize search sensitivity. Primary search terms included 'sudden cardiac arrest', 'sudden cardiac death', 'adults aged 18–40 years', 'cardiomyopathy', 'channelopathies', 'arrhythmia', 'out-of-hospital cardiac arrest', 'screening', 'athletes' and 'genetic predisposition'. Boolean operators (AND/OR) were applied to combine and refine search queries within each database. The search was conducted independently by two reviewers to ensure accuracy, reproducibility and minimize selection bias. Discrepancies in search results or study eligibility were resolved through discussion and consensus. In addition, the reference lists of all included full-text articles, relevant systematic reviews and major international clinical guidelines were manually screened to identify additional studies not captured during the initial search. The full PubMed search strategy is provided in Appendix S1, and the strategies for the other databases were adapted accordingly.

Eligibility criteria

Study selection was conducted according to predefined inclusion and exclusion criteria to ensure methodological rigour and relevance to the research objectives.

Figure 1: Study selection process



**Records were identified from five electronic databases: PubMed, Scopus, Web of Science, Embase and Google Scholar (n=3,000). The number of records retrieved from each database was not documented, and no records were obtained from registers. **Records excluded at title/abstract screening (n=1,800) due to irrelevance to sudden cardiac arrest in adults aged 18–40 years (e.g. focus on populations outside this age range or unrelated cardiovascular conditions, n=800), non-peer-reviewed sources (e.g. conference abstracts, editorials, n=400), studies published outside the 2015–2025 timeframe (n=200) and other reasons, such as insufficient detail in abstracts (n=200). No automation tools were used; all exclusions were performed independently by two reviewers. Data sourced from: Page et al., 2021.[21] This work is licensed under CC BY 4.0. To view a copy of this licence, visit https://creativecommons.org/licenses/by/4.0/*

Inclusion criteria

Studies were included if they were published between January 2015 and June 2025, investigated SCA or SCD in adults aged 18–40 years or provided extractable data for this age group, reported original findings on incidence, aetiological mechanisms, risk factors, diagnostic approaches or preventive strategies and employed rigorous study designs, including cohort studies, case–control studies, population-based registries, randomized controlled trials (RCTs), systematic reviews or meta-analyses. All included studies were selected for the availability of extractable data specific to individuals aged 18–40 years, in accordance with the predefined study objective. This included studies with broader age ranges, provided that relevant subgroup data for the 18- to 40-year-old cohort were available.

Exclusion criteria

Studies involving populations outside the 18–40 age range without extractable data for this group, non-peer-reviewed material (including case reports, editorials, letters to the editor and conference abstracts), studies not reporting outcomes relevant to SCA, those published in languages other than English or those unavailable in full text were all excluded.

Study selection process

Following the initial database search (n=3,000), 600 duplicates were removed using EndNote (version 23), resulting in 2,400 unique records for screening. Two independent reviewers screened the titles and abstracts for relevance according to predefined eligibility criteria. Following this initial screening, 600 articles were selected for full-text review. Comprehensive evaluation of these articles resulted in 55 studies meeting both the inclusion and quality criteria, which were included in the final qualitative synthesis. The study selection process is illustrated in the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 flow diagram (*Figure 1*), in accordance with PRISMA guidelines.[21]

An internal review protocol was developed prior to study initiation, detailing the objectives, eligibility criteria, search strategy, quality assessment methods and planned synthesis. Although the protocol was not prospectively registered in the International Prospective Register of Systematic Reviews (PROSPERO), all predefined procedures were rigorously followed to ensure methodological transparency and reproducibility.

Quality assessment

A standardized data extraction form was employed to collect relevant data from eligible studies, including study design, population characteristics, outcomes and key findings. Subsequently, the methodological quality of the included studies was appraised using established tools specific to each design: the Newcastle–Ottawa Scale (NOS) for observational studies and the Risk of Bias in Nonrandomized Studies of Exposures (ROBINS-E) tool.[22,23] For RCTs, the Cochrane Risk of Bias (RoB) tool was applied to evaluate internal validity.[24] The quality of included systematic reviews was appraised using the updated 'A Measurement Tool to Assess Systematic Reviews 2' (AMSTAR 2). All quality and RoB assessments were performed independently by two reviewers. Any discrepancies were resolved through discussion until consensus was reached. Discrepancies in search results, eligibility, data extraction or quality ratings were resolved through discussion and consensus between the two reviewers; a third reviewer was available if needed. The results of these assessments are summarized in *Table 1*. The table stratifies the findings by study design and assessment tool, reporting the number of studies, quality ratings (including mean NOS scores and ranges) and key methodological observations. The limited number of RCTs included reflects the predominantly observational nature of the current evidence on SCA in young adults.

Data extraction and analysis

Data from the included studies were extracted using a standardized template to ensure consistency. Extracted variables included study design, sample size, participant demographics, primary outcomes and key findings. Due to substantial heterogeneity in study designs, populations, outcome definitions and measurement methods, a narrative synthesis approach was adopted. Studies were thematically categorized according to their focus, including SCA incidence and prevalence, aetiological factors, associated risk predictors, diagnostic evaluation and preventive interventions. Any discrepancies in data extraction or interpretation were resolved through discussion between the two reviewers until consensus was reached.

Results

A total of 55 studies met the predefined inclusion and methodological quality criteria for this systematic review. The majority were observational in design, including 22 cohort or registry-based studies. The remaining studies comprised nine randomized or quasi-RCTs (primarily addressing resuscitation or preventive interventions), 15 systematic reviews or meta-analyses and several non-randomized investigations or case series with extractable data.

Table 1: Risk-of-bias assessment summary of included studies

Study type	Assessment tool	Number of studies	Quality scores/ratings	Key observations
Systematic reviews/ meta-analyses	AMSTAR 2	15	Moderate to high (mean score: 22/27; range: 20–25)	Most adhered to PRISMA; minor issues in protocol registration and funding reporting. Low-quality reviews excluded
Observational/cohort/ registry studies	NOS	22	Low to moderate (mean score: 7.2/9; range: 6–8)	High-quality population-based registries scored higher; limitations in confounding and follow-up in low-resource settings
Non-randomized studies of exposures	ROBINS-E	~10	Moderate (majority)	Confounding moderate–high (e.g. substance use, myocarditis); no high-risk studies were retained
Randomized or quasi-RCTs	Cochrane RoB/ RoB 2	9	Low to moderate (low risk: majority)	Limited RCTs in SCA topic; most were low risk for primary outcomes (e.g. interventions); some concerns in randomization/ blinding
Overall across all included studies	Multiple tools	55 total	Predominantly moderate	No high-risk studies retained; assessments conducted independently by two reviewers with consensus

AMSTAR 2 = A Measurement Tool to Assess Systematic Reviews 2; NOS = Newcastle–Ottawa Scale; PRISMA = Preferred Reporting Items for Systematic Reviews and Meta-Analyses; RCTs = randomized controlled trials; RoB 2 = Cochrane Risk of Bias 2 tool; ROBINS-E = Risk of Bias in Nonrandomized Studies of Exposures; SCA = sudden cardiac arrest.

The included studies examined a broad spectrum of research topics related to SCA in adults aged 18–40 years. Specifically, 22 studies investigated epidemiological trends and incidence rates across diverse populations. Another 14 focused on aetiological factors, with particular emphasis on inherited cardiomyopathies and primary arrhythmia syndromes, such as ion channelopathies. Ten studies evaluated screening modalities and risk stratification strategies, while nine assessed public health interventions, including community-based preventive measures and survival outcomes following SCA events. The key characteristics, findings and implications of all included studies are summarized in *Table 2*.[11,14,15,19,20,25–43]

Epidemiology and incidence of sudden cardiac death in young adults

SCA among individuals under 40 years of age is relatively rare compared with older populations; however, it remains a clinically significant cause of mortality, with notable regional and demographic disparities. Most population-based studies estimate the annual incidence of SCA in individuals aged 1–35 years at 1–2 cases per 100,000 person-years.[40,44,45] A large prospective study in Australia and New Zealand reported an incidence of 1.3 per 100,000 person-years in this age group, with approximately 72% of cases occurring in males, indicating a pronounced sex disparity.[26] Consistent findings have been reported in Europe. For example, a nationwide Danish cohort study covering 2000–2019 found an overall SCD incidence of 2.2 per 100,000 person-years among individuals aged 1–35 years, with males accounting for 69% of cases.[40] In contrast, a meta-analysis of multiple sources reported a broader incidence range of 1.1–8.7 per 100,000 person-years, reflecting differences in age definitions, case ascertainment and registry coverage.[31]

In East Asia, recent registry data demonstrate comparable epidemiological trends; for instance, a South Korean national registry reported SCA rates of 12.3 and 17.7 per 100,000 person-years in the 20–29 and 30–39 age groups, respectively, which encompassed both in-hospital cardiac arrest and OHCA.[19] Studies from China and Japan likewise indicate growing recognition of SCA among younger urban populations, although underreporting and limitations in data infrastructure remain significant challenges.[7,46] Data from South Asia, although limited, suggest a considerable burden of SCA among young adults. Reports from India and Iran indicate that 8–10% of all cardiac arrests occur in adults aged 18–40 years, especially in urban areas, where delayed emergency response and low bystander CPR rates contribute to poorer outcomes.[47,48] A multicentre Iranian study illustrated a substantial proportion of cardiac arrests in this age group, emphasizing disparities in emergency medical services (EMS) access and insufficient public knowledge of resuscitation practices.[27]

Sport-related SCA exhibits distinct epidemiological characteristics. A systematic review and meta-analysis estimated the incidence of sport-related SCA at 1.46 events per 100,000 athlete-years in individuals aged ≤35 years, with males accounting for nearly 87% of cases.[49] Recent, large-scale registries have established key patterns in sports-related SCA among children, adolescents and young adults. These events occur predominantly during recreational, non-competitive activities, with soccer and running being the most frequently involved. Although the overall incidence is low (approximately 0.25 per million children per year in paediatric cohorts), survival is critically dependent on immediate bystander CPR and the prompt use of a public-access AED.[36]

Evidence from contemporary registries indicates that the incidence of SCA is not significantly higher in competitive athletes than in the general population. In young adults (aged 18–40 years), most sports-related SCA events occur in non-elite male participants. CAD, notably acute coronary syndrome (ACS), represents a predominant aetiology in this group. Bystander AED application remains low (~7.5%); however, when paired with prompt CPR, survival can exceed 90%.[50] These findings are supported by analyses of the Danish Cardiac Arrest Registry and National Collegiate Athletic Association (NCAA) data, which collectively affirm that athletic participation itself is not an independent risk factor for SCA.[51,52]

Sex differences in SCA incidence among young adults are consistently reported. A Danish study of individuals aged 1–35 years reported an incidence of 3.6 per 100,000 person-years in males versus 1.8 in females, yielding a male-to-female incidence rate ratio (IRR) of 2.0.[53] In parallel, a meta-analysis in competitive athletes reported a male incidence of 1.42 per 100,000 athlete-years compared with 0.32 in females, reflecting an approximate 5.5-fold difference.[49] These disparities likely arise from a combination of biological, behavioural and environmental factors.

Several factors contribute to the heterogeneity in reported SCA incidence. Variability in study design, case definitions (e.g. inclusion of in-hospital versus out-of-hospital events) and data collection methods significantly affect reported rates. Socioeconomic and geographic disparities further influence both SCA incidence and outcomes. A meta-analysis found that individuals from lower socioeconomic backgrounds were 33–40% less likely to receive bystander CPR and had approximately 24% lower

Table 2: Summary of key included studies on sudden cardiac arrest in young adults (aged 18–40 years)[11,14,15,19,20,25–43]

Author (year)	Country	Study type	Population/age focus	Sample size/ setting	Focus	Main findings	Clinical implications
Marijon et al. (2015)[25]	France	Registry-based	Young adults (18–40) during sports	Large national registry	SCA during physical exertion	SCA more prevalent in midlife but significant in young	Emergency preparedness in athletic settings
Bagnall et al. (2016)[26]	Australia	Prospective cohort	Young adults (18–35)	490 SCD cases	Genetic causes of SCD	Genetic causes in 27% of cases	Support for molecular autopsy in young SCD
Mawani et al. (2016)[27]	Pakistan	Multicentre cohort	OHCA patients (18–40 subgroup)	Multicentre	OHCA outcomes in low-resource settings	Survival limited by inadequate EMS	Urges investment in EMS infrastructure
Mellor et al. (2017)[28]	Canada/UK	Registry (CASPER)	Young adults (18–40) with unexplained arrest	Large registry	Genetic testing after arrest	Pathogenic variants in ~20%	Reinforces genetic workup in survivors
Tester et al. (2020)[29]	USA	Genetic study	Young adults (18–40) with exertion-related events	Amish cohort	RYR2 mutations	Novel duplication identified	Value of genetic testing in arrhythmia syndromes
Harris and Lubitz (2020)[30]	USA	Registry-based	SCA survivors (18–40) with preserved EF	Registry cohort	Genetic evaluation after arrest	High yield of genetic testing	Supports cascade screening in families
Couper et al. (2020)[31]	UK	Systematic review	Young adults (18–40)	Review of global data	SCD incidence	Global incidence 1.3–2.5 per 100,000	Suggests population-wide screening strategies
Niederseer et al. (2021)[32]	Europe	Review	Young athletes (18–40)	Review	Echocardiography in screening	Role in distinguishing athlete's heart	Guides pre-participation evaluation
Semeraro et al. (2021)[33]	Europe	Guideline/RCT-informed	Young adults (18–40) community	Consensus + trial data	Systems saving lives	CPR training + AED deployment	Emphasizes community CPR and AED
Peterson et al. (2021)[15]	USA	Prospective cohort	Young competitive athletes (18–35)	4-year study	SCA in athletes	Inherited diseases major cause	Structured preparticipation screening
Uzendu et al. (2021)[34]	USA	RCT	Young adults (18–25) minority youth	School-based RCT	Virtual CPR training	Improved knowledge and readiness	Novel virtual CPR programmes for youth
D'Ascenzi et al. (2022)[11]	Italy	Meta-analysis	Young athletes (18–40)	Pooled data	SCD aetiology	Cardiomyopathies most common	Targeted screening in athletes
Brooks et al. (2022)[43]	International	Scientific statement/ RCT-informed	Young adults (18–40)	Consensus + trial	Public-access defibrillation	Innovative AED approaches	Optimizes outcomes after OHCA
Paratz et al. (2023)[20]	Australia	Registry	Young adults (18–40)	Large cohort	Vaccination and SCA	No association with COVID-19 vaccine	Reassures vaccination safety
Trytell et al. (2023)[35]	Australia	Registry	Young SCD (18–40 subgroup)	523 cases	Illicit drug use	32.5% positive toxicology	Routine toxicological evaluation
Bohm et al. (2023)[36]	Multi	Registry	Young adults (18–40) sports-related	Large cohort	Sports-related SCA	Survival 90.9% with CPR + AED	Promote bystander AED use
Palermi et al. (2023)[37]	Italy	Multimodality review	Young athletes (18–40)	Review	Athlete's heart imaging	Step-by-step multimodality approach	Improves diagnostic accuracy
Fovaeus et al. (2024)[38]	Sweden	RCT-informed cohort	Young adults (18–40)	Nationwide registry	OHCA survival trends	Survival from 7% to 20%	Benefits of early bystander CPR
Finocchiaro et al. (2024)[39]	UK	Narrative review	Young adults (18–40) athletes	Review	SCD mechanisms	Structural and arrhythmogenic dominate	Refine screening protocols
Bohm et al. (2024)[36]	Multi	RCT-informed registry	Young adults (18–40) sports	Large cohort	Sports SCA in young	Low incidence, high survival with CPR/AED	Immediate CPR and AED critical
Parizad et al. (2025)[14]	Multi	Review	Young adults (18–40)	Review	Emerging HF risks	Substance abuse as risk	Public health education on stimulants

Continued

Table 2: Continued

Author (year)	Country	Study type	Population/age focus	Sample size/ setting	Focus	Main findings	Clinical implications
Hansen et al. (2025)[40]	Denmark	Nationwide registry	Young adults (18–35)	Nationwide	SCD trends	Incidence declined 49%; survival improved	Progress via bystander CPR and ICD
Srivats et al. (2025)[41]	USA	Review	Young adults (18–40)	Review	AI in SCA prediction	AI models outperform EF alone	Endorses AI-wearable integration
Cho et al. (2025)[19]	USA	Review	Young adults (18–40)	Review	SCA prevention	AI-enabled multiparametric models	Recommends wearable-based detection
Astley et al. (2025)[42]	USA	Registry	Young athletes (18–40)	NCAA data	SCA during pandemic	No increase during COVID-19	Continue screening protocols

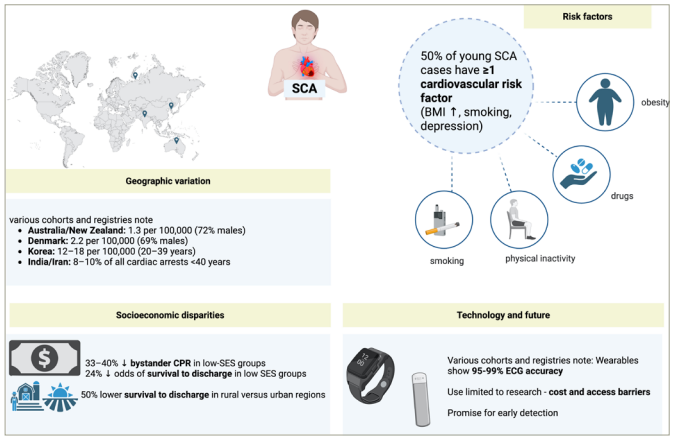
All studies included in this table report data specific to or extractable for individuals aged 18–40 years. Studies with broader age ranges were included only when relevant subgroup data for 18–40 years were available
AED = automated external defibrillator; AI = artificial intelligence; CASPER = Cardiac Arrest Survivors With Preserved Ejection Fraction Registry; COVID-19 = coronavirus disease 2019; CPR = cardiopulmonary resuscitation; EF = ejection fraction; EMS = emergency medical services; HF = heart failure; ICD = implantable cardioverter-defibrillator; NCAA = National Collegiate Athletic Association; OHCA = out-of-hospital cardiac arrest; RCT = randomized controlled trial; RYR2 = ryanodine receptor 2 gene; SCA = sudden cardiac arrest; SCD = sudden cardiac death.

odds of survival to hospital discharge compared with those from higher socioeconomic strata.[54]

Geographic differences are also evident between rural and urban settings. A systematic review across 13 countries demonstrated that although OHCA incidence was similar, survival to discharge was nearly 50% lower in rural areas (odds ratio [OR]≈0.52).[55] Corroborating this, a Danish cohort study (2016–2020) reported higher OHCA incidence in rural municipalities (IRR≈1.54), along with significantly reduced 30-day survival compared with urban regions.[56]

Individual lifestyle factors further influence SCA risk. Tobacco use, obesity, illicit substance use and physical inactivity are major contributors among adults aged 18–40 years. A large Danish cohort study reported that over 50% of SCA cases had at least one conventional cardiovascular risk factor, such as elevated body mass index or active smoking.[40] A nationwide cohort demonstrated that individuals aged 20–39 years with multiple behavioural risk factors, including smoking, obesity and depression, had a 2–3-fold increased risk of cardiovascular disease (CVD), with likely implications for SCA risk.[57]

Figure 2: Epidemiology of sudden cardiac arrest in adults aged 18–40 years



The schematic illustration summarizes key findings, including higher incidence in males, urban–rural disparities, geographic variations (with higher rates in Asian populations) and sport-related cases. Created with BioRender.com
BMI = body mass index; CPR = cardiopulmonary resuscitation; ECG = electrocardiography; NZ: New Zealand; SCA = sudden cardiac arrest; SES: socio economic status.

Technological advances offer new avenues for improving SCA detection and characterization. Wearable devices, including smartwatch-based four-lead electrocardiographic monitors, have shown high accuracy (95–99%) in ambulatory monitoring, although their use remains largely confined to research settings.[58] Despite these innovations, widespread deployment remains constrained by infrastructure, regulatory and economic challenges, particularly in low-resource regions. *Figure 2* illustrates the current epidemiological trends of SCA among adults aged 18–40 years.

Aetiological mechanisms and risk factors of sudden cardiac arrest in young adults

The primary aetiologies of SCA in young adults were identified as structural cardiac diseases, inherited channelopathies, myocarditis, coronary artery abnormalities and substance use.

Structural cardiac diseases

HCM, ARVC and coronary artery anomalies (CAAs) are the predominant structural causes of SCA in young adults. Advances in molecular autopsy and postmortem imaging have significantly improved elucidation of the genetic and pathological substrates underlying these conditions. Although they frequently follow an asymptomatic clinical course, structural cardiomyopathies account for a substantial proportion of SCDs among the young. This evidence underscores the value of systematic postmortem evaluations, incorporating both genetic testing and imaging, to inform preventive strategies and enable early identification of high-risk adults.[59,60]

Inherited channelopathies

Channelopathies are inherited disorders caused by dysfunction in cardiac ion channels, typically occurring in structurally normal hearts. Major syndromes include LQTS, BrS, CPVT and short QT syndrome. Collectively, these conditions account for an estimated 10–15% of SCA cases in this population.[59,61]

Pathogenic variants in genes encoding key cardiac ion channel subunits, including SCN5A (sodium), KCNQ1 (potassium) and RYR2 (calcium), are frequently implicated in this population. These genetic disturbances translate into identifiable clinical risk, particularly in BrS. Notably, patients with BrS who display a spontaneous type 1 ECG pattern face a substantially elevated risk of arrhythmic events, with an adjusted hazard ratio of 2.05 compared with those without this pattern.[62]

Broader genetic predispositions involving sarcomeric proteins, desmosomal components and ion channel modulators have also been associated with elevated risk of SCA. Pathogenic or probably pathogenic mutations are identified in 20–35% of autopsy-confirmed SCDs, while cascade family screening often reveals additional high-risk relatives. An FH of premature cardiac death remains a strong predictor of underlying heritable cardiac disease.[59]

Myocarditis

Myocarditis, defined as inflammatory infiltration of the myocardium, is an important but often under-recognized cause of SCA in this population, notably among athletes. Viral infections, such as enteroviruses, adenovirus and severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), can trigger myocardial inflammation and fibrosis, predisposing to malignant arrhythmias even when left ventricular systolic function (LVSF) is preserved.[63]

A nationwide registry including 14,294 sudden deaths in individuals aged 1–49 years identified myocarditis in approximately 6% of autopsy-confirmed SCA cases.[64] Furthermore, an Australian population-based registry reported myocarditis in 0.6% of OHCA cases in this population.[65] This inflammatory aetiology, especially when associated with viral infections, parallels emerging HF risk factors in younger populations.[14] Cardiac magnetic resonance (CMR) imaging, specifically with late gadolinium enhancement (LGE), is crucial for detecting subclinical myocarditis. Updated Lake Louise Criteria further enhance diagnostic accuracy and support arrhythmic risk stratification.[66,67]

Coronary artery abnormalities

CAAs, especially anomalous aortic origins of the coronary arteries, are rare but recognized contributors to exertion-related SCA in young individuals. These anomalies can cause MI or VAs due to dynamic compression or slit-like ostial narrowing during physical exertion.

A statewide prospective registry (2019–2021) of SCA in individuals aged 1–50 years identified CAAs in only 1% of autopsied cases, with none deemed the direct cause of arrest.[48–50] However, cohort studies and case series continue to suggest an association between CAAs and exercise-triggered SCA in select populations. Advanced imaging modalities, including coronary computed tomography angiography (CTA), CMR and CT-derived fractional flow reserve (CT-FFR), have enhanced both anatomical and functional evaluation. CT-FFR has been particularly useful for guiding unroofing procedures in adolescents, resulting in symptom resolution and restoration of coronary perfusion.[68–70]

Contemporary imaging modalities, including coronary CTA, CMR and CT-FFR, have markedly improved the anatomical and functional evaluation of coronary anomalies. For instance, CT-FFR has demonstrated clinical utility in guiding unroofing surgeries in adolescents, resulting in symptom resolution and restoration of coronary perfusion.[70] For individuals with high-risk anatomical features or significant ischaemic burden, surgical correction via unroofing, coronary reimplantation or neo-ostium formation is recommended.[71] Long-term follow-up indicates favourable outcomes, with most patients remaining asymptomatic and free from recurrent cardiac events.

Substance use and stimulant-associated arrhythmias

Stimulant substances, including cocaine, amphetamines, anabolic-androgenic steroids (AASs) and high-caffeine energy drinks, constitute an independent risk factor for VTs and SCA in young adults, even in the absence of underlying structural heart disease.[72,73] Their cardiotoxicity is mediated through multiple pathophysiological mechanisms, principally by heightening sympathetic tone, increasing myocardial oxygen demand, inducing coronary vasospasm and exerting direct toxic effects on cardiomyocytes. The consumption of energy drinks has been shown to induce acute adverse changes in cardiovascular parameters, including elevated blood pressure (BP) and altered heart rate (HR), which underscores these mechanistic pathways.[73,74]

Cocaine

Cocaine is recognized as one of the most potent and preventable precipitants of ventricular tachyarrhythmias and SCA among young individuals. Its use is independently linked to a substantially increased risk of SCA, with the highest hazard occurring during the acute phase and among frequent users.[2,75]

Similarly, methamphetamine use carries an independent hazard ratio of 1.90 for incident VTs.[76] AAS use confers a 2.1-fold increase in SCA risk, a relationship exacerbated during intense exertion due to associated myocardial hypertrophy and fibrosis.[77] Furthermore, excessive consumption of high-caffeine energy drinks is associated with ECG abnormalities, including corrected QT interval (QTc) prolongation and early repolarization patterns, which predispose individuals to VTs in a dose-dependent manner.[77,78]

This risk profile is corroborated by postmortem toxicological data, which identify stimulant use in approximately 30–33% of young SCD cases. One large registry reported positive toxicology or regular use in 32.5% of cases, predominantly involving cannabis and polysubstance abuse.[35] These findings underscore the necessity of routine toxicological screening in young SCA cases and highlight the urgent need for targeted public health initiatives to raise awareness and mitigate these preventable risks.[35,79]

Methamphetamine

Methamphetamine use is strongly associated with an elevated risk of life-threatening VTs, including ventricular tachycardia (VT) and ventricular fibrillation. A large longitudinal cohort study found a hazard ratio of 1.90 for incident VTs among methamphetamine users compared with non-users.[76,80] A pooled analysis of amphetamine abuse further indicated a high prevalence of hypertension (HTN) and ischaemic heart disease among users, along with notable incidences of acute MI and arrhythmias. These results emphasize the need for integrated psychiatric and cardiovascular care to manage these high-risk individuals effectively.[81]

Anabolic steroids

Emerging data also implicate anabolic steroid use in adverse cardiac outcomes, particularly among physically active young adults. A cohort study demonstrated a 2.1-fold increased risk of SCA in anabolic steroid users, especially during intense physical exertion. The study identified steroid-induced myocardial hypertrophy and fibrosis as key pathological mechanisms contributing to arrhythmogenesis.[82] A comprehensive literature review further indicates that AAS use induces adverse myocardial remodelling. This process is characterized by hypertrophy, systolic and diastolic dysfunction (including impaired LVSF and left ventricular diastolic dysfunction), as well as disturbances in lipid metabolism. These alterations collectively elevate the risk of myocardial ischaemia, arrhythmias and sudden death.[83] Furthermore, a systematic review of SCD cases related to anabolic steroid abuse revealed consistent data of myocardial fibrosis, hypertrophy and necrosis, structural changes that facilitate reentrant arrhythmias, especially in the context of exertion.[84]

Energy drinks

Recent studies indicate that energy drinks, which contain high concentrations of caffeine and other stimulants, can exert significant acute cardiovascular effects in young adults. Excessive consumption has been associated with ECG abnormalities, including QT interval prolongation and early repolarization patterns, acute elevations in blood pressure and dose-dependent QTc prolongation. These alterations heighten proarrhythmic potential, increasing susceptibility to VAs and MI.[77,78,85]

Public health implications and toxicological screening

Toxicological screening underscores a significant public health concern, revealing that stimulant use is implicated in approximately 30–33% of SCD cases in young adults. This is exemplified by a large registry in which 32.5% of cases showed positive toxicology or reported regular use, predominantly involving cannabis and other substances within a pattern of polysubstance abuse.[35] These observations advocate for routine toxicological evaluation as part of the diagnostic workup in young SCA victims. In total, evidence indicates that stimulants such as cocaine, amphetamines, anabolic steroids and energy drinks increase the risk of SCA by promoting electrophysiologic instability and myocardial stress. These agents may exacerbate latent cardiac conditions or directly precipitate lethal arrhythmias in otherwise healthy individuals.[1,86]

To reduce the burden of SCA among young populations, public health interventions must prioritize education regarding the cardiovascular risks of stimulant use. Implementation of targeted screening programmes and behavioural health initiatives is vital to promote awareness and safer lifestyle choices in at-risk youth.[87]

Comorbidities and systemic factors in sudden cardiac arrest in young adults

While structural and genetic cardiac abnormalities are the predominant causes of SCA in young adults under 40 years, a range of systemic and metabolic comorbidities also play critical modulatory roles. These conditions contribute to a pro-arrhythmic milieu through mechanisms such as electrolyte disturbances, chronic inflammation, myocardial fibrosis and autonomic imbalance, thereby increasing the risk of malignant VTs.

Chronic kidney disease

Chronic kidney disease (CKD) significantly elevates the risk of SCA, notably in advanced stages, through electrolyte imbalances, including hyperkalaemia and hypocalcaemia, which disrupt myocardial ion channel function and electrophysiological stability. A recent study demonstrated that young adults with stages III–V CKD had a 2.8-fold higher risk of developing VTs compared with age-matched controls.[79]

Metabolic syndrome

Metabolic syndrome (MetS), defined by the coexistence of obesity, insulin resistance, HTN and dyslipidaemia, promotes systemic oxidative stress and low-grade inflammation, both of which impair cardiac repolarization. A meta-analysis reported that young individuals with MetS had a 1.7-fold increased risk of SCA, with central adiposity and hyperglycaemia as the most primary contributing factors.[88]

Inflammatory and infiltrative diseases

Cardiac sarcoidosis (CS), an infiltrative inflammatory condition, has emerged as a potent risk factor for SCA in young adults. CS leads to granulomatous infiltration, myocardial scarring and fibrosis, precipitating conduction abnormalities and VTs. One study revealed that young adults with CS and associated conduction disturbances on ECG, such as atrioventricular block or bundle branch block, had significantly elevated odds of experiencing SCA, with ORs ranging from 5.0 to 13.7.[89]

Autonomic dysfunction

Abnormalities in autonomic regulation, especially heightened sympathetic activation, contribute to myocardial electrical instability. Reduced heart rate variability, an established marker of autonomic dysfunction, is independently associated with a 2.1-fold increase in the risk of VTs among young adults.[90]

Liver–heart and brain–heart axis interactions

Accumulating evidence underscores the pivotal role of multi-organ crosstalk in shaping arrhythmic vulnerability. For instance, non-alcoholic fatty liver disease has been associated with subclinical myocardial fibrosis and altered electrophysiological properties, despite the absence of clinically evident CVD. These systemic interactions, especially between hepatic and cardiac tissues, are increasingly recognized as contributors to susceptibility to arrhythmias.[91]

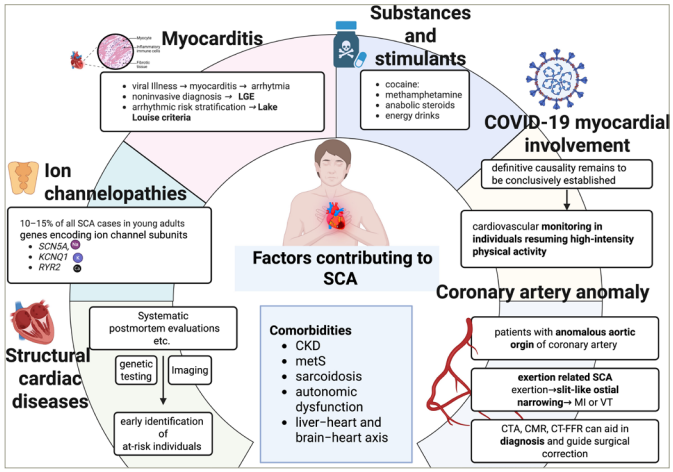
Beyond traditional cardiac aetiologies, systemic disorders such as CKD, MetS, CS, AD and inter-organ interactions markedly elevate the risk of SCA in young adults. A comprehensive risk stratification model incorporating both cardiac and systemic factors is essential for the early identification of individuals at elevated risk and the development of targeted preventive interventions.

Coronavirus disease 2019 and myocardial involvement

The COVID-19 pandemic raised concerns about potential myocardial injury and increased risk of SCA in young adults. Although myocarditis is a recognized complication of COVID-19, with a post-infection incidence approximately 2–4 times higher than after influenza in early studies (≈0.3–0.8 versus 0.1–0.3 per 1,000 person-years), large-scale registry and cohort studies have not shown a significant increase in SCA or SCD rates in this group during the pandemic or following vaccination. In major autopsy studies and national registries (2023–2025), myocarditis was found in approximately 1.5–4% of reviewed SCD cases, with no causal link established to COVID-19 vaccination. The benefits of vaccination continue to outweigh these rare risks.[20,42] Evidence indicates that COVID-19 may be a risk factor for myocardial remodelling and HF in younger populations, including those without pre-existing heart disease.[14] Therefore, structured cardiac assessment after infection, such as CMR imaging to detect LGE, is recommended for high-risk individuals before returning to intense exercise.[6,14,42] Current data do not indicate that SARS-CoV-2 infection or vaccination has increased the population-level burden of SCA.

Myocarditis represents the most frequently reported cardiac complication of COVID-19, most prominently in the early convalescent phase. A multicentre registry study and subsequent analyses found that approximately 8–15% of young patients recovering from SARS-CoV-2 infection exhibited myocardial abnormalities on cardiac magnetic resonance imaging, specifically LGE. These imaging findings were associated with an increased arrhythmic burden and QT interval prolongation.[92] Furthermore, emerging data on long-term cardiovascular sequelae indicate persistent myocardial involvement in some patients, irrespective of pre-existing HF. This sustained injury may contribute to a pro-arrhythmic substrate, underscoring the importance of continued clinical vigilance and monitoring for SCA risk in this population.[93]

Figure 3: Aetiological spectrum of sudden cardiac arrest in young adults (aged 18–40 years)



The graphic summarizes the primary causes identified, including structural cardiomyopathies (HCM, ARVC), inherited channelopathies (LQTS, BrS, CPVT), myocarditis, substance use, CAAs, systemic comorbidities and emerging factors, such as COVID-19-related myocardial involvement. Created with BioRender.com
ARVC = arrhythmogenic right ventricular cardiomyopathy; BrS = Brugada syndrome; CAAs = coronary artery anomalies; CKD = chronic kidney disease; CMR = cardiac magnetic resonance; COVID-19 = coronavirus disease 2019; CPVT = catecholaminergic polymorphic ventricular tachycardia; CTA = computed tomography angiography; CT-FFR = computed tomography-derived fractional flow reserve; HCM = hypertrophic cardiomyopathy; LGE = late gadolinium enhancement; LQTS = long QT syndrome; MetS = metabolic syndrome; MI = myocardial infarction; SCA = sudden cardiac arrest; VT = ventricular tachycardia.

Concerns have also emerged regarding the rare occurrence of vaccine-associated myocarditis, predominantly following messenger RNA (mRNA)-based COVID-19 vaccination. This phenomenon has been observed mainly in adolescent and young adult males, with a small but statistically significant elevation in risk. Despite this, the absolute incidence remains low, and current consensus indicates that the protective benefits of vaccination outweigh these risks.[94] Consequently, several cardiology and sports medicine societies have recommended updated post-COVID cardiac screening protocols for athletes and other high-risk populations to mitigate the potential for exercise-induced arrhythmic events.[95] While no definitive population-level increase in SCA attributable to COVID-19 or vaccination has been established, the systemic pro-inflammatory effects of the virus and the possibility of subclinical myocardial injury support on-going clinical vigilance and structured monitoring during recovery, especially in those returning to high-intensity physical activity. *Figure 3* illustrates the principal aetiological categories of SCA in this population.

Risk stratification and predictors of sudden cardiac arrest in young adults

Risk stratification for SCA in the young population represents a critical yet complex clinical challenge, as many affected individuals remain asymptomatic until the sentinel event. Effective identification of high-risk individuals requires an integrative approach incorporating clinical history, genetic predisposition and electrocardiographic findings.

Several clinical predictors have been consistently associated with increased SCA risk in this population. A history of unexplained syncope, notably during exertion or emotional stress, frequently precedes SCA and serves as an important warning sign.[52] A positive FH of SCD before the age of 50 is a significant indicator of inherited arrhythmogenic conditions, including cardiomyopathies and channelopathies.[75] In addition, factors such as male sex, low birth weight (LBW) and comorbidities like CKD and ACSs further refine individual risk profiles.[96] Additional factors, including male sex, LBW and comorbidities such as CKD and ACSs, further refine individual risk profiles.[96–98] Integration of these clinical and demographic variables facilitates earlier recognition of individuals at elevated risk.

ECG abnormalities remain a cornerstone of risk assessment. High-risk features include prolonged QTc (>480 ms), spontaneous type 1 Brugada pattern and inferolateral early repolarization, all of which have demonstrated predictive value in prospective cohort studies of young adults.[52,99] Additional markers, such as T-wave alternans, fragmented QRS complexes and signal-averaged ECG abnormalities, may provide additional insights, although their prognostic utility in younger populations requires further validation.[100]

Cardiac imaging plays a pivotal role in the detection of structural abnormalities that may not be evident on routine clinical evaluation. Transthoracic echocardiography remains the primary modality for diagnosing HCM, whereas CMR imaging is superior for detecting ARVC and myocarditis through LGE.[101,102] In cases with inconclusive findings, adjunctive testing, such as exercise stress testing or invasive electrophysiological studies, may reveal latent arrhythmogenic substrates.

Genetic testing represents an important tool for refining risk stratification, especially in individuals with a suggestive FH or phenotypic features of inherited arrhythmia syndromes. Variants in genes encoding cardiac ion channel proteins, including *SCN5A*, *KCNQ1* and *RYR2*, are commonly implicated in LQTS, BrS and CPVT.[103] Furthermore, molecular autopsy studies have demonstrated that up to 30–40% of unexplained SCA cases harbour pathogenic variants, underscoring the clinical relevance of postmortem genetic evaluation.[59]

Novel predictors, including biochemical biomarkers and autonomic indices, are being actively investigated to enhance contemporary risk prediction models. Elevated levels of high-sensitivity cardiac troponin and N-terminal pro-B-type natriuretic peptide have been associated with increased arrhythmic risk in specific cardiomyopathies.[59] Moreover, AD, assessed using measures such as heart rate (HR) variability and baroreflex sensitivity, is gaining recognition as a non-invasive marker of arrhythmic susceptibility in individuals with inherited arrhythmia syndromes.[104]

Notwithstanding these advances, a substantial proportion of SCA events occur in individuals previously classified as low risk, underscoring important limitations of current risk stratification frameworks. Accordingly, a comprehensive multiparametric approach incorporating clinical assessment, electrocardiographic findings, cardiac imaging, genetic testing and biomarker profiling is essential for accurate identification of individuals at elevated risk and for guiding targeted preventive interventions. The principal clinical, electrocardiographic, imaging, genetic and biochemical predictors of SCA risk are summarized in *Table 3*.[25,59,62,102,104–113]

Screening and prevention of sudden cardiac arrest in young adults

SCA in young adults remains a major public health concern, frequently occurring in the absence of preceding symptoms. This inherent unpredictability poses substantial challenges for early identification and prevention. Accordingly, the implementation of effective screening strategies is critical for the timely detection of individuals at increased risk and for the initiation of appropriate preventive interventions.

Table 3: Key risk stratification parameters for sudden cardiac arrest in young adults (18–40 years)[25,59,62,102,104–113]

Category	Key elements	Reference
Clinical predictors	History of unexplained syncope, family history of SCD before age 50, male sex, low birth weight, CKD and ACS	25,105,106
Electrocardiographic markers	Prolonged QTc (>480 ms), spontaneous type 1 Brugada pattern, inferolateral early repolarization, T-wave alternans, fragmented QRS complexes and signal-averaged ECG abnormalities	62,107,108
Cardiac imaging	Transthoracic echocardiography for HCM; cardiac MRI for ARVC and myocarditis (using late gadolinium enhancement); exercise stress testing and EPS for concealed arrhythmogenic substrates	102,109,110
Genetic testing	Pathogenic variants in *SCN5A*, *KCNQ1* and *RYR2* genes associated with LQTS, BrS and CPVT; 30–40% of unexplained SCA cases harbour actionable mutations	59,111
Biomarkers and autonomic indices	Elevated high-sensitivity troponin and NT-proBNP levels; HRV and baroreflex sensitivity as non-invasive predictors	104,112,113

ACS = acute coronary syndrome; ARVC = arrhythmogenic right ventricular cardiomyopathy; BrS = Brugada syndrome; CKD = chronic kidney disease; CPVT = catecholaminergic polymorphic ventricular tachycardia; ECG = electrocardiography; EPS = electrophysiological study; HCM = hypertrophic cardiomyopathy; HRV = heart rate variability; KCNQ1 = potassium voltage-gated channel subfamily KQT member 1; LQTS = long QT syndrome; MRI = magnetic resonance imaging; NT-proBNP = N-terminal pro–B-type natriuretic peptide; QTc = corrected QT interval; RYR2 = ryanodine receptor 2; SCA = sudden cardiac arrest; SCD = sudden cardiac death; SCN5A = sodium channel protein type 5 subunit alpha.

Current screening approaches

The effectiveness and scope of cardiovascular screening in asymptomatic young individuals, especially athletes, continue to be debated. Both the AHA and the ESC advocate a targeted screening approach based on a detailed personal and FH combined with physical examination.[2,18] In selected high-risk populations, the addition of a resting 12-lead ECG is recommended.[114,115] While ECG-based screening improves sensitivity for detecting inherited arrhythmogenic conditions such as LQTS and BrS, concerns persist regarding limited specificity, false-positive results and overall cost-effectiveness. These limitations may result in unnecessary downstream testing, psychological burden and potential restriction from athletic participation.[116]

Role of electrocardiography and imaging

ECG abnormalities, including prolonged QTc, Brugada-type patterns or early repolarization variants, often prompt further imaging evaluation. Transthoracic echocardiography remains the first-line imaging modality for the assessment of structural heart disease, whereas CMR imaging provides superior spatial resolution and tissue characterization. CMR is particularly valuable in cases with inconclusive echocardiographic findings or when cardiomyopathies such as HCM or ARVC are suspected.[32,37,117] The integration of ECG findings with advanced imaging techniques enables a more accurate and comprehensive diagnostic assessment, especially in athletic populations where physiological remodelling may mimic pathological conditions.

Genetic and family-based screening

Cascade genetic screening of first-degree relatives of individuals affected by SCA represents a powerful strategy for identifying asymptomatic carriers of pathogenic variants. This approach is crucially important when conventional diagnostic tools, including ECG and imaging, fail to yield definitive results. Genetic testing facilitates early risk stratification, implementation of prophylactic measures and individualized surveillance strategies. Recent studies indicate that a substantial proportion of affected relatives may remain clinically silent despite harbouring actionable genetic variants, underscoring the importance of family-based screening for SCA prevention.[28–30]

Preventive measures and lifestyle modifications

Lifestyle-based preventive strategies are integral to risk reduction in individuals with inherited arrhythmia syndromes or cardiomyopathies. In patients with LQTS or ARVC, avoidance of high-intensity physical activity, particularly swimming in LQTS type 1, as well as stress management, plays a critical role in minimizing arrhythmic triggers. Nutritional optimization, including maintenance of adequate potassium and magnesium levels, contributes to myocardial electrical stability. These non-pharmacological interventions, when combined with pharmacological therapies such as beta-adrenergic blockers, have demonstrated efficacy in reducing arrhythmic events and improving survival outcomes.[12,118]

In addition, structured cardiovascular counselling improves adherence to follow-up and promotes sustained risk-reducing behaviours. Personalized lifestyle interventions tailored to individual risk profiles show promise in optimizing long-term management. When integrated with pharmacological therapies, antiarrhythmic drugs or implantable cardioverter-defibrillators in selected cases, these measures form a comprehensive and individualized prevention strategy.[12,119]

Public health and community-based strategies

Population-level interventions play a crucial role in reducing the burden of OHCA among young adults. The deployment of AEDs in high-risk public settings, including schools and athletic facilities, has been associated with improved survival outcomes. Despite proven effectiveness, AEDs remain underutilized, highlighting a gap between availability and effective implementation.[43] Furthermore, widespread community training in CPR is essential. Evidence indicates that early bystander-initiated CPR and AED use can increase survival rates by up to fourfold. Programmes promoting dispatcher-assisted CPR and targeted public education initiatives have further enhanced bystander response. Early recognition of warning symptoms, such as exertional syncope or palpitations, also facilitates timely intervention, emphasizing the importance of public awareness in cardiac safety strategies.[33,120] A comprehensive overview of the principal screening and prevention strategies for SCA in young adults is summarized in *Table 4*.[12,25,28–30,32,33,38,114–118,120]

Outcomes and survival after sudden cardiac arrest in young adults

Survival after OHCA in young adults has gradually improved over recent decades, largely attributable to increased rates of bystander CPR and advances in EMS systems. Data from the Swedish National Registry spanning 1990–2020 demonstrate that 30-day survival increased from approximately 7% to nearly 20%, paralleling a marked reduction in the median time to CPR initiation from 14 to 2 min.[38] Several factors have been consistently associated with improved survival and favourable neurological outcomes in this population, including younger age, male sex, a public location of cardiac arrest, witnessed events and the presence of an initial shockable rhythm. In contrast, unwitnessed arrests

Table 4: Strategic framework for the screening and prevention of sudden cardiac arrest in young adults (18–40 years)[12,25,28–30,32,33,38,114–118,120]

Strategy	Target population	Key components	Evidence level
Preparticipation screening	Competitive athletes, high-risk families	History, physical exam, 12-lead ECG (selective)	High[114–116]
Advanced diagnostic imaging	Suspected cardiomyopathy or anomaly	Echocardiography, CMR, CT angiography	High[32,38,117]
Genetic evaluation	SCA survivors, first-degree relatives	Cascade screening, molecular autopsy	Moderate–high[28–30]
Lifestyle modification	LQTS, ARVC, high-risk profiles	Avoid competitive sports, triggers; optimize electrolytes	Moderate[118]
Pharmacotherapy	Confirmed channelopathies, HCM	Beta-blockers, antiarrhythmics, anticoagulation	High[12]
Device therapy	High-risk survivors (EF <35%, VT)	ICD implantation	High[12]
Public access defibrillation	General population, high-traffic venues	AED placement in schools, gyms, public spaces	High[35]
Bystander CPR Training	School students, community members	Mandatory school programmes, dispatcher-assisted CPR	High[33,120]

AED = automated external defibrillator; ARVC = arrhythmogenic right ventricular cardiomyopathy; CMR = cardiac magnetic resonance; CPR = cardiopulmonary resuscitation; CT = computed tomography; ECG = electrocardiography; EF = ejection fraction; HCM = hypertrophic cardiomyopathy; ICD = implantable cardioverter-defibrillator; LQTS = long QT syndrome; SCA = sudden cardiac arrest; VT = ventricular tachycardia.

and prolonged delays in resuscitation are strong predictors of poor outcomes and increased mortality.[121]

Early initiation of bystander CPR and prompt defibrillation remain central determinants of survival. Accumulating evidence indicates that the combined effects of dispatcher-assisted CPR, layperson intervention and rapid AED deployment substantially improve both survival and neurological recovery.[122] Notwithstanding these advances, pronounced geographic disparities persist. A nationwide study from South Korea reported roughly 1.5-fold higher survival rates among patients in urban regions compared with rural areas, primarily driven by shorter EMS response times and higher rates of bystander intervention.[122] Similarly, population-based data from Minnesota demonstrated that rural OHCA cases were associated with longer EMS response intervals (12.9 versus 10.6 min) and lower survival-to-discharge rates (9.3% versus 13.1%) compared with urban counterparts.[123]

Neurological recovery among young adult survivors is generally favourable but remains heterogeneous. Observational studies suggest that 60–70% of survivors discharged from hospital achieve good neurological outcomes. Nevertheless, a substantial subset experiences persistent cognitive deficits, emotional disturbances or psychological sequelae, underscoring the importance of structured post-resuscitation and rehabilitation programmes.[124] Overall, optimizing outcomes following OHCA in young adults requires a multifaceted approach encompassing early event recognition, immediate bystander CPR, rapid AED deployment and minimization of prehospital delays, particularly in underserved or rural settings. Neurologically intact survival remains the most meaningful benchmark of resuscitation success and long-term recovery in this population.

Public health strategies and prevention

Community-based public health interventions, particularly widespread CPR education and strategic deployment of AEDs, have demonstrated substantial effectiveness in improving survival outcomes following SCA among young populations. Systematic reviews consistently indicate that school-based and community-wide training initiatives significantly enhance bystander CPR rates, thereby improving outcomes in OHCA scenarios.[120] A notable example comes from Busca, Italy, where a municipality-wide initiative resulted in 90% of residents, including students, reporting willingness to perform CPR. Moreover, the majority of public spaces had access to an AED within 4 min, highlighting the impact of proactive, localized preparedness strategies.[125] The benefits of early bystander-initiated defibrillation are well documented. In a multicentre study, implementation of evidence-based resuscitation protocols, combined with increased rates of bystander CPR and AED use, nearly doubled survival rates from 4.8% in 2006 to 9.4% in 2013, accompanied by marked improvements in neurological outcomes.[126] Another investigation illustrated that timely prehospital interventions, particularly rapid CPR and defibrillation, were associated with a 76% relative increase in survival with favourable neurological function.[127] Patients presenting with an initial shockable rhythm who received early defibrillation exhibited significantly higher survival rates and superior neurological recovery.[128]

Recent educational interventions further reinforce these findings. A pilot study utilizing virtual CPR and AED instruction during the COVID-19 pandemic reported substantial improvements in adolescents' knowledge, confidence and readiness to respond during cardiac emergencies.[34] In addition, a nationwide study in the USA found that mandatory CPR and AED training in schools achieved high student participation (~86%). However, self-reported confidence in AED use remained limited, emphasizing the need for enhanced hands-on training.[129]

Even with these advancements, practical challenges remain. A recent review identified that about 15% of AEDs were non-functional or inaccessible during actual emergencies, underscoring the necessity of routine equipment maintenance and comprehensive deployment audits to ensure operational readiness.[130] Collectively, this body of evidence underscores the multifactorial and complex nature of SCA in young adults, encompassing diverse aetiologies, heterogeneous diagnostic approaches and multiple determinants of clinical outcomes.

Discussion

This systematic review provides a comprehensive synthesis of current evidence on the epidemiology, aetiology, diagnostic modalities, risk determinants and preventive strategies for SCA in young adults aged 18–40 years. The results underscore the multifactorial nature of SCA in this population, encompassing structural cardiac abnormalities, inherited electrical disorders, systemic and inflammatory conditions, environmental exposures and lifestyle-related triggers.

Structural heart diseases remain the most frequently implicated causes of SCA in young adults, with HCM and ARVC consistently identified as predominant aetiologies.[13] Among individuals with structurally normal hearts, inherited arrhythmogenic channelopathies, including LQTS, BrS and CPVT, play a pivotal role in precipitating malignant arrhythmias.[29,62]

In recent years, increasing attention has focused on systemic and inflammatory conditions, such as myocarditis, CS and CKD. Myocarditis, particularly following viral infections or COVID-19, has emerged as a notable contributor to SCA in young adults.[92] Moreover, AD and electrolyte imbalances, especially in the context of chronic illness, further heighten susceptibility to life-threatening ventricular tachyarrhythmias.[104]

Substance use and behavioural factors significantly influence the risk of SCA. Recreational exposure to stimulants, including cocaine, amphetamines and AAS, as well as excessive intake of energy drinks, has been linked to fatal arrhythmic events, even in structurally normal hearts.[81,82] These modifiable risk factors reflect patterns increasingly implicated in early-onset HF among younger populations.[14] In addition, non-genetic triggers, such as vigorous physical exertion, specifically in competitive athletes, and acute emotional stress, can precipitate malignant arrhythmias in predisposed individuals.[52]

Survival outcomes following OHCA in young adults have gradually improved over the past decade, largely owing to enhancements in EMSs, broader deployment of AEDs and increased public training in CPR.[38] Nevertheless, geographic disparities remain, with lower survival rates observed in rural areas due to delayed EMS response and limited AED accessibility compared with urban regions.[56]

Screening practices have evolved from population-wide initiatives towards more targeted, risk-based strategies. Current recommendations prioritize focused ECG screening in individuals presenting with concerning clinical features, a FH of SCD or known genetic predispositions.[115,116] Routine universal screening is generally discouraged due to high false-positive rates and associated healthcare costs. Advanced diagnostic tools, including CMR imaging and genetic testing, are increasingly employed to refine risk stratification in SCA survivors and their first-degree relatives.[102]

Evolving technologies, such as wearable biosensors and artificial intelligence-assisted ECG interpretation, are under investigation for real-time arrhythmia monitoring and early intervention.[29] When integrated within personalized care frameworks, these digital tools may complement traditional diagnostics and enhance predictive accuracy in identifying high-risk individuals.

Mitigating the burden of SCA in young adults necessitates a multidimensional strategy that integrates early risk identification, equitable access to advanced diagnostic modalities, comprehensive public education and the incorporation of innovative technologies for prevention, surveillance and timely intervention. Recommendations for targeted screening and preventive measures are primarily based on consistent observational data, registry findings and expert consensus from major guidelines (e.g. 2022 ESC, AHA statements), as high-level randomized trial evidence remains limited in this population.[2,18]

This integrative framework is consistent with contemporary international guidelines, notably the 2022 ESC Guidelines for the management of VTs and the prevention of SCD, as well as relevant scientific statements from the AHA on SCA prevention and resuscitation in younger populations.[2,18]

Limitations

The substantial heterogeneity across studies (in populations, methodologies and outcome definitions) precluded meta-analysis and limited the ability to draw a single quantitative conclusion; however, the narrative synthesis highlights consistent patterns in aetiology and prevention opportunities. Given the predominance of observational data and the scarcity of large-scale randomized trials, formal assessment using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) system for the assessment of screening efficacy was not feasible; recommendations, therefore, reflect the best available evidence and current expert consensus.

This systematic review has several limitations that should be acknowledged when interpreting the findings. Substantial heterogeneity in study designs, including cohort studies, registries and systematic reviews, as well as differences in populations, definitions of SCA and outcome reporting, precluded quantitative meta-analysis and may limit the consistency and precision of the synthesized results. Although validated tools, such as the NOS, ROBINS-E and AMSTAR 2, were used for quality assessment, residual confounding and methodological biases in the primary studies cannot be fully excluded. The majority of included studies were conducted in high-income countries (Europe, North America and East Asia), potentially reducing the generalizability of findings to low- and middle-income settings, where healthcare infrastructure, emergency response capabilities and data collection practices differ substantially. Additionally, some studies encompassed broader or overlapping age ranges, which may have introduced minor dilution of age-specific insights for the 18- to 40-year-old group.

The relatively modest number of included studies reflects the stringent inclusion criteria, which excluded case reports, case series and non-peer-reviewed publications to prioritize methodological rigour and higher levels of evidence. While this focused approach enhanced the reliability of the synthesis, it may have limited the overall volume of eligible studies. A further limitation is the incomplete availability of postmortem or autopsy-confirmed data in many reports, particularly for cases classified as unexplained sudden death, which may affect the accuracy of aetiological attributions. Publication bias is also a concern, as studies with null or negative results are less likely to be published.

Clinical implications

The findings carry important implications for clinicians, public health authorities and emergency response systems involved in the management of SCA in young adults. The heterogeneous aetiologies, including cardiomyopathies, channelopathies, myocarditis and substance-related triggers, require a comprehensive diagnostic strategy. Early recognition of warning signs, such as exertional syncope or palpitations in individuals with a FH of sudden death, is critical for timely evaluation.[114–116]

ECG and CMR imaging are central to identifying arrhythmogenic substrates, especially when echocardiographic findings are inconclusive. The integration of genetic testing and biomarker profiling further refines individualized risk assessment and facilitates early intervention.[28–30,117] Current clinical guidelines support multiparametric screening approaches to detect asymptomatic individuals at elevated risk.[12]

At the population level, widespread CPR training and strategic placement of AEDs, particularly in schools, gyms and rural settings, have been shown to improve survival following OHCA.[33,120] Mandatory school-based training programmes and regular AED maintenance represent cost-effective measures with proven impact.[43] Nevertheless, persistent gaps in device accessibility and functionality, especially in underserved regions, continue to constrain optimal outcomes.

Knowledge gaps and future directions

Although there is growing scientific interest, substantial gaps remain in understanding SCA in young adults aged 18–40 years. A key limitation

is the scarcity of large-scale, prospective registries and cohort studies specifically focused on this age group, which restricts accurate estimation of incidence, particularly in low- and middle-income countries. Furthermore, many cases remain unexplained even after thorough autopsy and molecular testing, reflecting incomplete insight into the underlying pathophysiological mechanisms.

Current screening and diagnostic tools face challenges, including interobserver variability in ECG interpretation, limited normative data for diverse ethnic groups and athletes, and difficulty distinguishing physiological adaptations from pathology. While numerous risk factors, such as structural heart disease, channelopathies, substance use and systemic comorbidities, have been identified, their relative contributions, interactions and predictive value in integrated models are not yet fully defined.

Future research should prioritize the establishment of multinational, age-stratified registries and longitudinal studies to generate robust epidemiological data. Development of validated, multiparametric risk scores incorporating clinical, electrocardiographic, imaging, genetic and biomarker information is necessary to improve identification of at-risk individuals. Routine implementation of postmortem molecular autopsy and advanced imaging in unexplained deaths would enhance aetiological classification.

Multi-omics approaches and validation of emerging technologies, such as wearable devices, artificial intelligence-assisted ECG analysis and remote monitoring in diverse populations, hold considerable promise for early detection. Public health initiatives should focus on equitable access to genetic counselling, mandatory school-based CPR training and optimized AED deployment, particularly in underserved areas. Ultimately, a precision medicine framework combining individualized screening, lifestyle interventions and targeted therapies will be essential to reduce the burden of SCA in young adults.

Conclusion

SCA in young adults remains a rare but devastating event, frequently occurring without prior warning signs. This systematic review emphasizes its complex, multifactorial aetiology, encompassing structural cardiomyopathies, inherited channelopathies, inflammatory conditions, substance-related triggers and systemic comorbidities, along with on-going challenges in early detection and emergency response. Despite notable advances in diagnostic tools, genetic evaluation, bystander intervention and public access defibrillation, significant disparities persist in screening access, survival rates and neurological outcomes, particularly in rural and resource-limited settings.

Reducing the burden of SCA in this population requires a coordinated, multifaceted approach, including targeted risk assessment and cascade family screening in high-risk individuals, widespread CPR education and strategic AED deployment in communities, enhanced clinical awareness, equitable access to advanced diagnostics, the establishment of age-specific registries and the integration of emerging technologies into precision prevention strategies. Through sustained multidisciplinary collaboration and public health efforts, meaningful improvements in early identification, survival and long-term quality of life for at-risk young adults can be achieved. ❑

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