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RESEARCH ARTICLE | Originally Published 11 December 2025

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Low-Field Portable MRI Detects Developmental Pattern of Ultraearly Cerebral Infarction in aSAH

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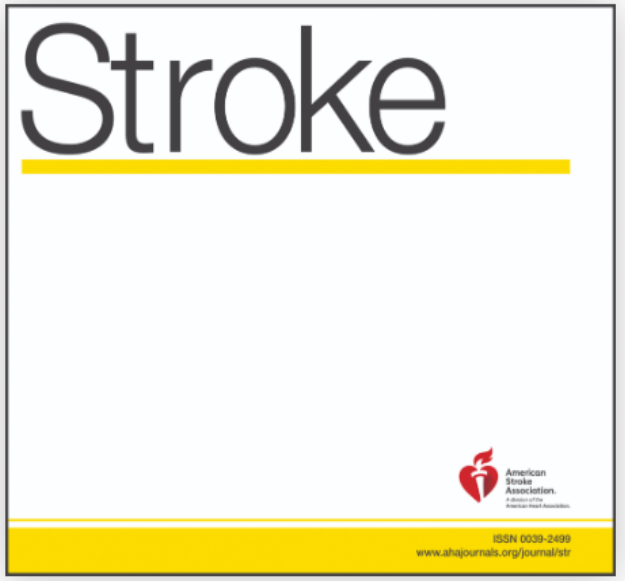
Stroke • New online • https://doi.org/10.1161/STROKEAHA.125.052474



Abstract

BACKGROUND:

Aneurysmal subarachnoid hemorrhage (aSAH) is an acute cerebrovascular disease. The concept of ultraearly infarction is gaining recognition. The advent of low-field portable magnetic resonance imaging (LF-pMRI) technology offers a promising new avenue for exploration. We aimed to identify the developmental pattern of ultraearly infarction after aSAH with LF-pMRI.

METHODS:

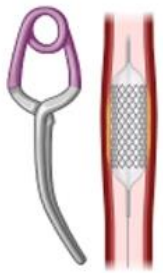
This was a prospective, observational, single-center cohort study. Data for patients with consecutive subarachnoid hemorrhage admitted to Beijing Tiantan Hospital from January 1, 2024 to July 31, 2024 were obtained. The inclusion criteria were as follows: (1) diagnosis as subarachnoid hemorrhage, (2) patients who underwent computed tomography, computed tomography angiography, and LF-pMRI in the emergency room before treatment of the aneurysm. Patients with comorbidities, angiographically negative subarachnoid hemorrhage, onset time >72 hours, and Hunt-Hess scale score of 5 were excluded. All enrolled patients underwent LF-pMRI on admission to evaluate the occurrence of ultraearly infarction. Primary outcome was the occurrence of unfavorable functional outcomes (modified Rankin Scale score ≥2). Modified Rankin Scale score was identified by physicians at discharge. Patients who received emergency aneurysm treatment and received complete LF-pMRI process during hospitalization were further evaluated for the risk of delayed cerebral ischemia (DCI), which was the secondary outcome.

RESULTS:

Among 176 patients with aSAH, 14.20% (25/176) were observed ultraearly cerebral infarction, and 28.41% (50/176) developed DCI. Multivariate analyses showed that ultraearly infarction was an independent risk factor for unfavorable functional outcomes (odds ratio, 3.095 [95% CI, 1.304–7.345]; *P*=0.010). Subsequently, for the secondary outcome, among 92 patients who received emergency aneurysm treatment and underwent a complete LF-pMRI process, 16.30% (15/92) developed ultraearly cerebral infarction, and 54.35% (50/92) developed DCI. Multivariate analyses showed that for patients with aSAH presenting with DCI, ultraearly infarction (odds ratio, 7.027 [95% CI, 1.485–33.253]; *P*=0.014), modified Rankin Scale score before admission (odds ratio, 3.737 [95% CI, 1.117–12.508]; *P*=0.032) were independent risk factors.

Lf-pMRI detects ultra-early cerebral infarction in aSAH

First Lf-pMRI
14.2% ultra-early infarction



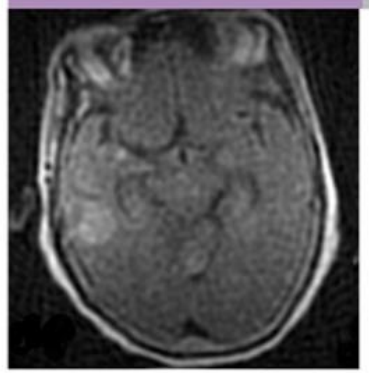
Clipping or Embolization

Second Lf-pMRI

Third Lf-pMRI

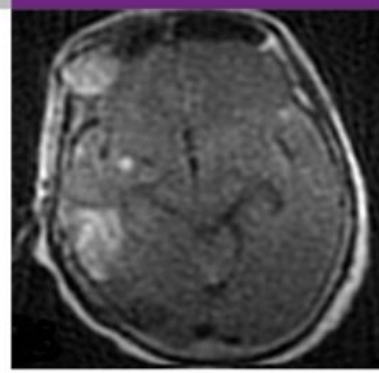
aSAH
n = 176

Onset ≤ 72h



Surgery

Day 1



Day 3-7

Primary outcome: MRS≥2 56.0%vs29.1%

Ultra-early infarction (Group 2) n=25	No ultra-early infarction (Group 1) n=151

Secondary outcome: DCI 52.0%vs24.5%

Ultra-early infarction (Group 4) n=15	No ultra-early infarction (Group 3) n=77

Lf-pMRI: low field portable MRI; aSAH: aneurysmal subarachnoid hemorrhage; MRS: modified Rankin scale; DCI: delayed cerebral infarction

CONCLUSIONS:

LF-pMRI can be utilized to detect the occurrence of ultraearly cerebral infarction in patients with aSAH. Those patients with aSAH who experience ultraearly cerebral infarction have a higher risk of DCI and a greater incidence of unfavorable functional outcomes.

Urinary Exosomal miRNAs as Biomarkers in Chronic Kidney Disease Progression

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Keywords: Chronic Kidney Disease, Urinary Exosomes, Microrna Biomarkers, Mir-192, Gut-Kidney Axis, Uremic Toxins

PDF

Published

2025-06-30

Issue

Vol. 2 No. 01 (2025): Advances in Biosciences Research

Section

Articles

Journal Information

Journal Name: Advances in Biosciences Research

Short Name: ABR

ISSN(Print) : 3080-3438

ISSN (Online) : 3080-3446

Frequency : Semi-Annual

Nature: Print and Online

Submission: Via OJS System

Languages of Publication: English

Review Type: Double Blind Peer Review

Publisher: METROPOLE RESEARCH AND TECHNOLOGY HUB (SMC-PRIVATE) LIMITED

Publisher Address: Office No. 21, 3rd Floor, Pakland Business Center, I-8 Markaz, Islamabad, Islamabad Capital Territory 44000, Pakistan.

Abstract

Chronic Kidney Disease (CKD) remains a global health burden, necessitating novel biomarkers for early detection and progression monitoring. This study investigated the utility of urinary exosomal microRNAs (miRNAs) as non-invasive biomarkers in CKD by profiling their expression across varying disease stages. A cohort comprising 60 CKD patients (stages 1–5) and 30 healthy controls was analyzed. Urinary exosomes were isolated and characterized using electron microscopy and nanoparticle tracking, followed by RNA extraction and qRT-PCR to quantify selected miRNAs, including miR-21, miR-29a, miR-192, miR-200c, and miR-223. The results demonstrated significant upregulation of miR-192 (4.8-fold), miR-21 (3.5-fold), and miR-200c (3.0-fold) in stage 5 CKD patients compared to controls ($p < 0.01$). These miRNAs were strongly correlated with decreased eGFR (r = –0.72 for miR-192) and increased serum creatinine, indicating their potential in reflecting renal dysfunction. Further, the gut-derived uremic toxins that were extremely high in stage 5 patients included indoxyl sulfate and p-cresyl sulfate which showed positive correlation (r= 0.68 and 0.60) with miR-192 expression and systemic inflammation (CRP levels). The findings from these studies emphasize the crosstalk between the gut microbiome, toxic uremic build-up and the miRNA derangement at the gut-kidney axis. The research also revealed that urinary exosomal miRNAs provide a robust, reliable, accessible and sensitive platform for post-CKD progression and metabolic problems screening. Due to their association with the existing renal function markers and inflammatory mediators with good correlations, these miRNAs can contribute to risk stratification and timely clinical interventions. In conclusion, urinary exosomal miRNAs, and particularly miR-192, become promising non-invasive biomarkers with diagnostic and prognostic significance in CKD, which needs further validation in bigger and more diverse cohorts.

JAMA Network

JAMA

Original Investigation

Efficacy and Safety of Oral PCSK9 Inhibitor Enlicitide in Adults With Heterozygous Familial Hypercholesterolemia

A Randomized Clinical Trial

Christie M. Ballantyne, MD[1]; Laura Gellis, MD, MPH[2]; Jean-Claude Tardif, MD[3] ; et al

Author Affiliations | Article Information

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JAMA
Published Online: November 9, 2025
2026;335;(2):129-139. doi:10.1001/jama.2025.20620

Key Points

Question Does treatment with the oral proprotein convertase subtilisin/kexin type 9 inhibitor enlicitide lead to clinically meaningful reductions in low-density lipoprotein cholesterol in adults with heterozygous familial hypercholesterolemia?

Findings In this phase 3, randomized clinical trial including 303 adults with heterozygous familial hypercholesterolemia and stable background statin use, the percentage change in the level of low-density lipoprotein cholesterol at 24 weeks was clinically meaningful and significant in those treated with enlicitide (−58.2%) vs placebo (2.6%) (between-group difference, −59.4%).

Meaning In adults with heterozygous familial hypercholesterolemia, enlicitide is an effective and well-tolerated treatment for lowering the level of low-density lipoprotein cholesterol.

Abstract

Importance Persons with heterozygous familial hypercholesterolemia (HeFH) are at increased risk of atherosclerotic cardiovascular disease due to lifelong elevated levels of low-density lipoprotein cholesterol (LDL-C). Many patients with HeFH do not achieve guideline-recommended LDL-C goals with the currently available lipid-lowering therapies.

Objective To evaluate the efficacy of enlicitide decanoate (an oral proprotein convertase subtilisin/kexin type 9 inhibitor) vs placebo in adults with HeFH requiring further lowering of LDL-C levels despite use of statin therapy.

Design, Setting, and Participants This phase 3, randomized clinical trial included persons aged 18 years or older with HeFH currently using lipid-lowering therapy (taking at least a moderate- or high-intensity statin) and either an LDL-C level of 55 mg/dL or greater and a history of major atherosclerotic cardiovascular disease or an LDL-C level of 70 mg/dL or greater without a history of major atherosclerotic cardiovascular disease. The trial was conducted at 59 sites across 17 countries; the first participant was screened on August 8, 2023, and the last follow-up visit occurred on April 7, 2025.

Interventions Participants were randomized (2:1) to 20 mg of enlicitide (n = 202) or placebo (n = 101) once daily for 52 weeks.

Main Outcomes and Measures The primary outcome was the mean percentage change in LDL-C level at week 24. The secondary outcomes included the mean percentage change in LDL-C level at week 52, the mean percentage change at week 24 in levels of non–high-density lipoprotein cholesterol (non–HDL-C) and apolipoprotein B, and the median percentage change at week 24 in lipoprotein(a).

Results Of the 303 participants (mean age, 52.4 [SD, 13.5] years; 51% were female) randomized, 293 (96.7%) completed the trial. The mean LDL-C level was 119.0 mg/dL (SD, 41.0 mg/dL) at baseline, all had statin current use (81.5% were taking a high-intensity statin), and 64.4% were taking ezetimibe. The mean percentage change in LDL-C level at week 24 was −58.2% in the enlicitide group vs 2.6% in the placebo group (between-group difference, −59.4% [95% CI, −65.6% to −53.2%]; $P < .001$). The mean percentage change in LDL-C level at week 52 was −55.3% in the enlicitide group vs 8.7% in the placebo group (between-group difference, −61.5% [95% CI, −69.4% to −53.7%]; $P < .001$). At week 24, the mean percentage change in non–HDL-C level was −52.3% in the enlicitide group vs 2.1% in the placebo group (between-group difference, −53.0% [95% CI, −58.5% to −47.4%]; $P < .001$), the mean percentage change in apolipoprotein B level was −48.2% vs 1.8%, respectively (between-group difference, −49.1% [95% CI, −54.0% to −44.3%]; $P < .001$), and the median percentage change in lipoprotein(a) level was −24.7% vs −1.6% (between-group difference, −27.5% [95% CI, −34.3% to −20.6%]; $P < .001$). The incidence of adverse events, serious adverse events, and study discontinuation due to adverse events was similar between groups.

Conclusions Among adults with HeFH, treatment with enlicitide was well tolerated and significantly reduced levels of LDL-C, apolipoprotein B, non–HDL-C, and lipoprotein(a).

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Clinica Chimica Acta

Volume 583, 1 March 2026, 120839

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Review

GDF-15 as an integrative cardiometabolic biomarker

Obaid Afzal [a], Muhammad Afzal [b], Nawaid Hussain Khan [c], Ali Altharawi [a], Mubarak A. Alamri [a], Manal A. Alossaimi [a], Abdulmalik S.A. Altamimi [a], Pavan Goud [d]

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Highlights

- GDF-15 integrates metabolic stress, inflammation, and tissue injury signals.
- Analytical advances: immunoassays and MS improve precision of GDF-15 testing.
- Biological variation (age, sex, comorbidity) demands context-specific ranges.
- High GDF-15 predicts poor outcomes in diabetes, obesity, HF and atherosclerosis.
- Prognostic cut-offs support CV, renal and mortality risk stratification.

Abstract

Growth Differentiation Factor 15 (GDF-15) is a stress-inducible cytokine that is abundantly expressed in cardiomyocytes, adipose tissue, macrophages, endothelial cells, and vascular smooth muscle cells. The levels of this biomarker increase in response to tissue injury, inflammation, and metabolic stress and are elevated in the presence of primary cardiometabolic diseases, such as type 2 diabetes, obesity, hypertension, atherosclerosis, heart failure, and chronic kidney disease. This is articulated in a clinical chemistry narrative review, which elucidates why GDF-15 can be characterized as an integrative biomarker. This characterization is due to the shared pathophysiological processes of metabolic stress, inflammation, oxidative/mitochondrial stress, and tissue injury across these conditions, as well as its clinical applications in risk assessment, prognosis, and monitoring. We also briefly present modern methods to measure circulating GDF-15 (primarily immunoassays, with new mass-spectrometry techniques being developed) and note that inter-assay variability and assay standardization are considered to be the major impediments to clinical application. The most significant determinants of biological variability (age, sex, renal function, genetic variation, and comorbidities), by which reference intervals and longitudinal interpretation are affected, are also highlighted. Increased GDF-15 is consistently associated with poor outcomes in diabetes, obesity, hypertension, heart failure, and atherosclerosis, such as cardiovascular events, kidney failure, and death. Nonetheless, prognostic cutoffs have been proposed variably across studies, populations, and measurement platforms. Therefore, these thresholds should be approached with caution until harmonized assays and clinically validated decision limits established through prospective studies are available. GDF-15 is a powerful integrative biomarker of metabolic stress, inflammation, and tissue injury in cardiometabolic diseases. Its introduction into clinical practice may enhance early risk stratification, personalize therapeutic interventions and monitor disease progression. Future studies should focus on optimizing the standardization of assays, developing evidence-based reference intervals (and reference change values), and elucidating clinically practical decision limits and therapeutic implications of the GDF-15 pathway in the treatment of cardiometabolic diseases.

Cell Reports Medicine
A Cell Press journal

ARTICLE · Volume 6, Issue 11, 102455, November 18, 2025 · *Open Access*

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A combined genomic arrhythmia propensity score delineates cumulative risk

Tanner O. Monroe [1] · Megan J. Puckelwartz [1,2] · Lorenzo L. Pesce [1,2] · … ·
Alfred L. George, Jr. [2] · Gregory Webster [6] · Elizabeth M. McNally [1,7] … Show more

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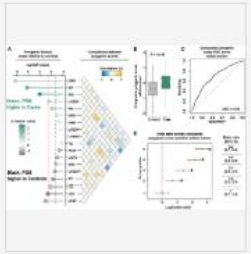
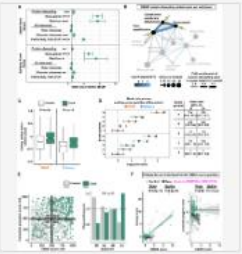
Highlights

- Ventricular arrhythmias are often unpredictable and are influenced by genetic risk
- Combining common, rare coding region and noncoding variation stratifies arrhythmia risk
- The cumulative risk, called GAPS, performs with or without primary pathogenic mutations
- This approach should be applicable to heritable complex disease

Figures (6)

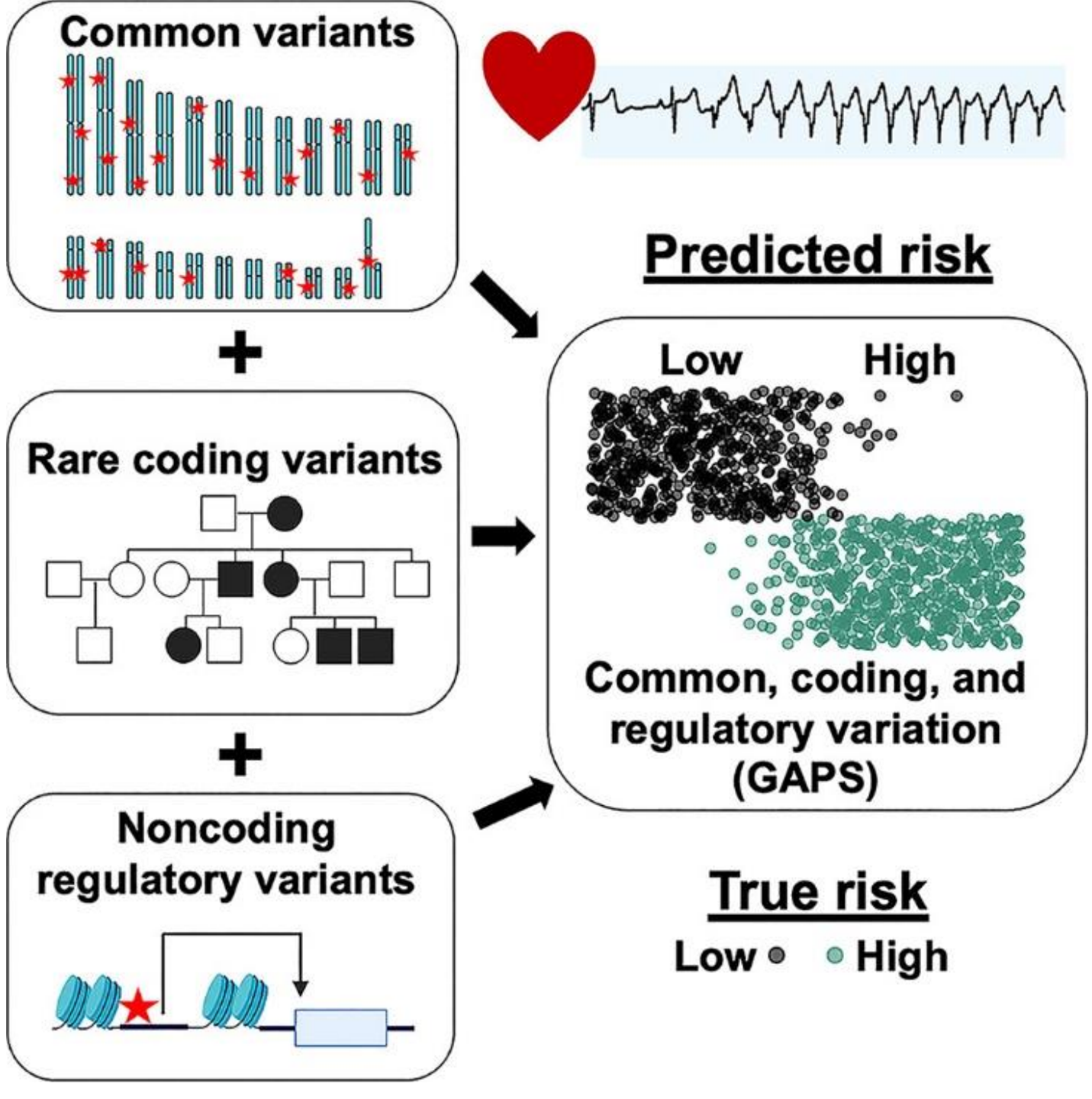
Figure Viewer





Summary

Cardiac ventricular arrhythmias can cause sudden death. Despite known genomic contributions, multigenic risk predictors are limited. The genetics of arrhythmias and cardiomyopathies overlap, with additional overlap with epilepsy. To improve genetic risk prediction, we assemble a cohort with non-ischemic ventricular arrhythmias and controls lacking cardiac diagnoses. Here, we integrate 18 polygenic scores; variants from clinical gene panels for coding regions of cardiomyopathy, arrhythmia, and epilepsy genes; and noncoding regulatory regions mapping to those genes. Polygenic scores alone hold prognostic value. Rare coding variants identify cumulative risk extending beyond known pathogenic/likely pathogenic variants. We also find enrichment of ultrarare regulatory variation. A risk predictor that combines all variant classes outperforms any single class or subset and replicates in a validation cohort. This combined genomic arrhythmia propensity score (GAPS) identifies high-risk individuals even among those who lack known primary pathogenic variants. This integrated approach serves as a model for other complex traits.


Common variants
Rare coding variants
Noncoding regulatory variants
Predicted risk
Low
High
Common, coding, and regulatory variation (GAPS)
True risk
Low
High

Results

- Cohort
- As part of an American Heart Association Strategically Focused Research Network, we assembled 523 cases (median = 33 years, interquartile range [IQR], [19, 44]) who experienced SCD or carried high clinical SCD risk (Table 1). This early age enriches for genetic mediators.[39] Approximately 58% of cases had non-ischemic cardiomyopathy and 129 (25%) died from SCD. Nearly two-thirds (63%) of living participants had an implantable cardiac defibrillator (ICD) or were medically recommended for ICD. Of the ICD carriers, 34% received an ICD for primary prevention, 36% for secondary prevention, and 30% experienced an ICD discharge. The total case cohort expanded from two prior cohorts[11,12] (Figure S1). Cases included SCD (*n* = 129) events ascertained from a nationwide study.[11] We included individuals with nonischemic VAs with hypertrophic cardiomyopathy (HCM), dilated cardiomyopathy (DCM), or arrhythmogenic cardiomyopathy because of arrhythmia risk.[12] An additional 298 cases were recruited from cardiology clinics or referrals, and 36 cases were identified from NUgene biobank. Controls consisted of 596 individuals aged ≥40 years (median = 56, IQR, [48, 55]) with no known cardiac disease history (see STAR Methods). Controls did not undergo clinical cardiac screening, so they cannot be considered free of cardiac conditions. A portion was reserved for replication (*n* = 67 cases, *N* = 59 controls).

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for listening.