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Structured postmortem genetic counselling and testing in sudden unexpected death among young adults in the Netherlands: Results and implications regarding hereditary risk

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Abstract

Introduction: Sudden unexpected death (SUD) in young adults may result from hereditary cardiovascular disorders that remain undetected even after conventional autopsy. Routine genetic testing may enable identification of hereditary cardiovascular disorders and can offer important diagnostic opportunities and preventive benefits for surviving relatives. **Methods:** Between September 2022 and October 2025, 100 cases of sudden unexplained death in young adults in the Netherlands were investigated using the NODOV (Nader Onderzoek DoodsOorzaak Volwassenen) procedure. Genetic counselling was offered to the next of kin in 50 of these cases in whom conventional autopsy results pointed towards an inherited cardiovascular disorder. Conventional autopsy findings, medical history and family history guided phenotype-directed genetic testing using targeted gene panel analysis after whole-exome/genome sequencing.

Results: Genetic testing was successfully performed in 46 out of 50 SUD cases and identified pathogenic variants in six cases, involving the CACNA1C, BAG3, MYBPC3, LDL-R, KCNJ2, and PKD1 genes. Pretest genetic counselling proved to be of importance in guiding genetic testing. Pathogenic variants were identified in both structurally abnormal and autopsy-negative cases. Surviving families received tailored genetic counselling and recommendations for clinical follow-up based on predictive genetic testing. In 43 out of 44 families without a genetic diagnosis advice for clinical screening was given by a clinical geneticist.

Conclusion: This study highlights the importance of a structured multidisciplinary postmortem investigation, such as the NODOV procedure, which incorporates molecular autopsy combined with (pretest) genetic counselling. This approach may improve determination of cause of death (particularly in autopsy-negative cases), aid in the identification of hereditary cardiovascular disorders and facilitate preventive strategies for at-risk relatives.

Introduction

Sudden unexpected death (SUD) in young adults is a rare event that raises questions regarding the underlying cause of death, the presence of hereditary cardiovascular disorders, and opportunities for prevention.[1]Three overlapping terms are commonly used in literature on sudden death: sudden unexpected death (SUD), sudden cardiac death (SCD), and sudden arrhythmic death syndrome (SADS). These classifications reflect varying levels of diagnostic certainty regarding the underlying cause of death. SUD represents the broadest category, referring to a sudden and unexpected death in which no definitive cause is identified during the initial investigation, irrespective of whether a conventional autopsy has been performed. SCD refers to sudden death attributable to a cardiac cause, either confirmed by postmortem investigation or presumed on clinical grounds. Causes of SCD include structural cardiac abnormalities such as cardiomyopathies, coronary artery disease (CAD) and valvular heart disease.[2-4]SADS represents a subset of SCD and describes sudden death in individuals without evidence of a structural heart disease at conventional autopsy. In SADS cases at young age, death can

result from a concealed cardiomyopathy or an inherited arrhythmogenic disorder, such as long QT syndrome or Brugada syndrome.[5-7]

In clinical practice, cases are initially categorized as SUD before conventional autopsy. Classification may subsequently change to SCD when a structural cardiac abnormality is identified or strongly suspected. Conventional autopsy remains the gold standard for determining the cause of death; however, some cases remain unexplained despite conventional autopsy.[8] Cases may be classified as SADS when conventional autopsy findings are negative, and a hereditary arrhythmogenic disorder or concealed cardiomyopathy is either suspected or genetically confirmed.[7] Previous studies have reported an average incidence of SCD of approximately 1–2 deaths per 100,000 person-years among individuals younger than 40 years.[9]The cause of death remains unexplained in approximately 30–40% of SCD cases in individuals aged 29–48 years, despite the performance of a conventional autopsy.[4,10] Consequently, surviving relatives may be left without clear information regarding the cause of death or its implications, potentially resulting in uncertainty and emotional distress. In addition, surviving relatives may be at risk of carrying undetected hereditary disorders, potentially exposing them to health risks that could otherwise be identified and treated through timely genetic and medical assessment.

Prior studies demonstrated that postmortem genetic testing identified pathogenic or likely pathogenic variants associated with the cause of death in approximately 22.1% of SUD cases (range, 20–23%).[11] Among cases classified as SADS, the overall pooled diagnostic yield of postmortem genetic testing was reported to be 11% when both channelopathy- and cardiomyopathy-associated genes were analysed.[7] Previous research showed that hereditary cardiomyopathies and channelopathies were more prevalent among younger individuals, whereas SCD in older individuals was predominantly attributed to CAD and myocardial infarction.[12] This age-dependent shift in etiology may suggest a greater contribution of environmental factors and gene–environment interactions in older individuals, particularly in the pathogenesis of CAD. Nevertheless, postmortem genetic testing remains clinically valuable across age groups, as the identification of pathogenic or likely pathogenic variants can facilitate the detection of at-risk relatives and enable targeted cascade screening, preventive interventions, and long-term clinical surveillance, thereby potentially reducing the incidence of subsequent sudden deaths within affected families.[8]

In 2022, a structured postmortem investigation procedure for sudden and unexpected deaths in young adults aged 18–45 years was implemented in the Netherlands. The procedure, known as NODOV (Nader Onderzoek DoodsOorzaak Volwassenen; in English translated to
further investigation into the cause of death amongst adults), integrates multiple postmortem diagnostic modalities, including toxicological analysis, computed tomography (CT) imaging, conventional autopsy, molecular autopsy (also known as postmortem genetic testing) and genetic counselling.[13]This study aimed to evaluate the outcomes of molecular autopsies performed within the structured postmortem examination procedure (NODOV) and to assess the implications for surviving relatives through genetic counselling.

Methods

Study Design

This multicentre prospective cohort study was conducted in the Netherlands between September 2022 and October 2025. During this study period, 100 individuals aged 18–45 years who experienced sudden and unexpected death presumed to be of natural origin were included. The indication for postmortem genetic testing was assessed following evaluation of the findings obtained from the other postmortem examination modalities incorporated within the NODOV procedure. The next of kin were referred for genetic counselling in cases showing a potential hereditary cause (e.g. cardiomyopathy signs or myocardial infarction) or where the cause of death remained insufficiently explained and/or a suspicion of a genetic predisposition persisted.

Genetic counselling and genetic testing

Genetic testing was preceded by pretest genetic counselling of a next of kin of the deceased. During genetic counselling, the genetic counsellor discussed information regarding the deceased's medical history, family history, potential heritable conditions, and the possible outcomes and limitations of genetic testing. During counselling, additional consent was requested to perform genetic testing. Mostly, this was a molecular autopsy, meaning genetic testing on freshly frozen or paraffin embedded tissue of the spleen obtained from the deceased during autopsy. Genetic testing was performed in one of the university-affiliated DNA laboratories. Guided by the pathological findings from the conventional autopsy, suspected underlying disease mechanisms, and patient and family history, targeted gene panels were selected to optimize diagnostic yield. Several gene panels were available: an arrhythmia gene panel, a cardiomyopathy gene panel, and a dyslipidaemia gene panel. Additional phenotype-directed panels were applied when other heritable disease categories were suspected. Also, in a select number of cases family history showed a known genetic cause. In these cases, genetic testing focused on the familial variant. Molecular analyses were predominantly performed using whole-exome sequencing (WES) with subsequent filtering on respective gene panels. As the study was conducted over multiple years and across different laboratories, different versions of specific gene panels were used, meaning gene content could vary mildly. Genetic variants were classified into pathogenicity classes according to the American College of Medical Genetics and Genomics (ACMG) guidelines.[14] Clinically relevant findings, including pathogenic and likely pathogenic variants, as well as variants of uncertain significance (VUS) with strong molecular or clinical suspicion, were documented for potential familial evaluation.

Outcomes

The primary outcome measures were a) the identification of pathogenic or likely pathogenic genetic variants associated with the cause of death, and b) cardiovascular screening advice for relatives of gene-elusive deceased patients.

Data Analysis

Descriptive analyses were performed. Data are presented as percentages, ranges, and qualitative summaries. No inferential statistical analyses were conducted.

Results

Study population

In 50 out of 100 SUD cases included in the NODOV study, either no cause of death was established through the other postmortem examination modalities (excluding molecular autopsy) within the NODOV procedure, or the cause of death was suggestive for an underlying hereditary disorder. Consequently, the next of kin in these 50 cases were offered pre-test genetic counselling. *See* Fig. 1 *for further details.* The study population was comprised of 10 females (20%) and 40 males (80%), with an age range of 19–45 years.

Conventional autopsy findings

Conventional autopsy was performed in 48 out of 50 cases. In two out of 50 cases postmortem CT imaging showed a clear cause of death, making conventional autopsy redundant. In 13 out of 48 cases no cause of death could be established during conventional autopsy, and these were classified as autopsy-negative. There were (relevant) findings in 35 out of 48 conventional autopsy cases: CAD was the most common observation in sudden young deaths, with evidence of myocardial infarction in 14 cases. Myocardial infarction due to CAD was most common in the older age groups (37–45 years). CAD without certain evidence of myocardial infarction (negative lactate dehydrogenase staining) was described in seven cases. Cardiomyopathy was described in eleven cases. In two of these cases both CAD and cardiomyopathy were described. Other vascular disorders (type-A-dissection and brain aneurysms) were described in three cases and pulmonary embolism in one case. Myocardial infarction with nonobstructive coronary arteries (MINOCA), and multiorgan failure (MOF) were each described in one case. *See Appendix 1 for further details.*

Molecular autopsy findings

Of the 50 families receiving genetic counselling, genetic testing was successfully performed in 46 families. Among the 46 tested cases, a pathogenic variant was identified in 6 cases (13%). Pathogenic variants were identified in the CACNA1C, BAG3, MYBPC3, LDL-R, KCNJ2 and PKD1 genes. The conventional autopsy findings were consistent with the detected genetic cause: The two cases with a pathogenic variant in a gene related to channelopathies (CACNA1C, KCNJ2) had negative conventional autopsies. The two cases with a pathogenic variant in a cardiomyopathy gene (BAG3, MYBPC3) had visible signs of cardiomyopathy at autopsy. The one case with a pathogenic variant in a gene related to

familial hypercholesterolemia (LDL-R) had significant CAD. The one case with a pathogenic gene related to autosomal dominant polycystic kidney disease (PKD1) had polycystic kidneys, a polycystic liver, and a brain aneurysm. In three additional cases a VUS was found.

Genetic counselling and family recommendations

Pretest genetic counselling resulted in tailored genetic testing or family counselling in six cases. In one family, the genetic cause had been identified in their extended family, leading to targeted variant testing in the deceased. In two families with CAD, genetic testing was performed in one or more affected surviving relatives, in whom genetic testing could include copy number variant analyses of the gene panel, which is not possible on DNA derived from tissue. In another family, the initial genetic test on the deceased's tissue was negative, however relatives showing cardiac abnormalities resulted in genetic testing in one of these relatives. As a result, the detected pathogenic variant could later be confirmed in a different tissue specimen of the deceased.

The six families of the SUD victims in which a pathogenic variant was found were advised to undergo genetic testing and/or further examination. Cascade genetic testing was recommended to identify surviving relatives at risk for heritable disease. In one case in which a pathogenic variant was found, the family decided not to go through with genetic testing.

Forty-three out of 44 families of deceased individuals with a negative genetic test result were mostly given recommendations based on clinical guidelines. Families received a family letter with instructions at which age testing should start (and finish) and what type of testing was recommended. In cases with a stronger suspicion of a genetic cause, e.g. when a cardiomyopathy was detected at autopsy, periodic follow-up with a medical specialist was recommended. In gene-elusive CAD cases cardiovascular risk management (CVRM) was advised. In gene-elusive autopsy-negative cases mostly a one-time extensive cardiac and/or vascular evaluation was recommended. In one case pharmaceutical advice was given to female family members. In another case family members were advised to undergo a medication provocation test. Finally, in one family no recommendations were given as the deceased was gene-elusive and a potential toxicological cause of death was also present. *See* Fig. 1*for further details.* The highest level of advice given to the families by the clinical geneticist is recorded in the flowchart, irrespective of whether families decided to follow through, or multiple recommendations were given within one family.

Discussion

Forty-six out of 50 SUD cases (92%) successfully underwent postmortem genetic testing. Six out of these 46 cases (13.0%) had a positive genetic test result; four out of 33 autopsy-positive cases (12.1%) and two out of thirteen autopsy-negative cases (15.4%).

Previous research demonstrated that postmortem genetic testing could identify pathogenic or likely pathogenic variants associated with the cause of death in approximately 22.1% of SUD cases (range,

20–23%).[11] No distinction was made between the different categories, such as SCD and SADS, or positive and negative autopsies. It is therefore not possible to explain with certainty why overall diagnostic yield was lower in this study versus previous research (13% versus 22.1%). This study solely offered genetic counselling and postmortem genetic testing (including only relevant cardiovascular-related gene panels) in cases suspicious of a heritable cardiovascular disorder, possibly resulting in a lower number of included cases and more precision genetic testing. Furthermore, SUD cases were not excluded from this study after comprehensive postmortem investigation was performed, despite an underlying toxicological cause; further resulting in a lower diagnostic yield.

However, previous research reported that among SADS cases (i.e. autopsy-negative cases or autopsy findings with unknown significance), the overall pooled diagnostic yield of postmortem genetic testing was reported to be between 6–11%, depending on whether channelopathy- and cardiomyopathy-associated genes were analysed separately or combined.[7] The diagnostic yield in autopsy-negative cases was higher in this study (15.4%). This may be due to the small sample size. Collectively, these findings support the additional diagnostic value of molecular autopsy in the evaluation of young sudden unexplained death.

A major strength of this study is the novel standardised offer of comprehensive postmortem investigations, provided free of charge to families since 2022 through the NODOV procedure.[13] Transportation, conventional autopsy, toxicology analyses, and postmortem CT imaging were provided without financial burden to surviving relatives, while genetic testing and counselling were largely reimbursed through health insurance (except the deductible excess). By reducing socioeconomic barriers to participation, this approach likely enhanced the representativeness of the study cohort. In addition, participation was entirely voluntary, and surviving relatives retained full autonomy regarding consent for conventional and molecular autopsy, strengthening the ethical integrity of the study. The study focused on young adults in whom the likelihood of identifying hereditary causes of death is higher, as acquired cardiovascular disease due to lifestyle and/or environmental factors becomes increasingly more prevalent after age 45.[12]

The genetic testing strategy was tailored rather than protocolized. Gene selection was guided by conventional autopsy findings, family history, and known familial variants. In selected families, testing of affected surviving relatives enabled better testing properties and decreased false negative findings in tissue samples of the deceased, thereby increasing diagnostic yield. An additional strength is the integration of genetic counselling into the postmortem investigation. Besides the mentioned effects on testing strategy, pre-test genetic counselling is essential when hereditary disease is suspected, as it informs surviving relatives about inheritance patterns, clinical implications, psychosocial consequences, and options for cascade genetic testing in case of positive results. Identification of a pathogenic variant allows early detection and timely management of asymptomatic carriers at risk of arrhythmias, cardiomyopathies, premature CAD, heart failure, myocardial infarction, or sudden death, thereby reducing morbidity and mortality. Surviving relatives were offered tailored follow-up recommendations based on conventional autopsy, genetic testing and family history. Depending on the estimated hereditary risk,

family recommendations ranged from one-time diagnostic testing, periodic cardiovascular risk management (CVRM) by a general practitioner, one-time extensive follow-up with a medical specialist (most often cardiological evaluation, including electrocardiography, echocardiography and exercise testing) to repeated periodic follow-up with a medical specialist. Such tailored follow-up strategies account for age-dependent disease expression and may reduce delayed or missed diagnoses. Families were respected in their decision to decline or defer recommended follow-up actions. The highest level of advice provided was recorded irrespective of uptake. *See* Fig. 1 *and Appendix 1*.

This study further highlights limitations of conventional autopsy alone. Channelopathies and other hereditary arrhythmia syndromes may leave no structural abnormalities detectable at conventional autopsy, emphasizing the complementary role of molecular investigations in unexpected sudden death.

The relatively small sample size limits generalizability. Diagnostic yield was possibly influenced by the composition of available gene panels and current limitations in variant interpretation. Variants of unknown significance may later prove clinically relevant as genomic knowledge evolves. The presence of any VUS were shared with next of kin.

Importantly, genetic testing within the NODOV study was contingent on consent for conventional autopsy. When surviving relatives declined conventional autopsy, genetic testing was not performed, potentially resulting in missed hereditary diagnoses. This requirement may also have reduced inclusiveness, as some surviving relatives may consider conventional autopsy too invasive while still accepting minimally invasive genetic testing approaches. Precise data regarding this matter falls outside the scope of this study. Future protocols should therefore evaluate whether postmortem genetic testing can be offered independently of full conventional autopsy.

Current gene panels also remain incomplete. Novel disease-associated variants continue to be identified, and future sequencing technologies may improve detection of currently unrecognized pathogenic mechanisms. Previous studies have shown that re-evaluation over time can yield additional diagnoses, with most new findings emerging within five years after initial investigation.[14] The recommendation for families with persistent suspicion of hereditary disease to seek re-evaluation after several years is therefore justified and consistent with existing evidence. The findings additionally reflect the increasing recognition that hereditary cardiomyopathies, channelopathies, and familial hypercholesterolemia are not always purely monogenic disorders with straightforward Mendelian inheritance patterns. Contemporary genetic research suggests that disease susceptibility may result from complex interactions between multiple genetic variants and environmental or acquired factors, including obesity, diabetes, medication exposure, and physiological stress.[12] Further research is therefore warranted to understand how genetic predisposition interacts with external modifiers in SUD.

Overall, these findings highlight the clinical and forensic value of the NODOV procedure as a structured multidisciplinary framework for investigating sudden unexpected death in young adults in the Netherlands. Beyond improved determination of cause of death, the structured NODOV procedure creates opportunities for prevention among surviving relatives through targeted cascade genetic testing

and individualised clinical follow-up. Future research should focus on larger prospective cohorts with standardized postmortem and genetic protocols, long-term follow-up of screened surviving relatives, and the evaluation of minimally invasive and cost-effective postmortem genetic testing strategies. At the policy level, broader implementation of standardized postmortem procedures incorporating genetic counselling and targeted genetic testing, such as the NODOV procedure, may improve identification and prevention of hereditary causes of sudden cardiac death.

Conclusion

This study demonstrates the added value of the structured NODOV procedure, which integrates conventional autopsy, postmortem CT imaging, toxicological analysis, genetic counselling, and targeted postmortem genetic testing in the investigation of young sudden unexplained death in the Netherlands. Pathogenic variants were identified in approximately 13% of successfully tested cases, including individuals with negative conventional autopsy findings, highlighting the complementary role of postmortem genetic testing in improving cause-of-death determination and identifying hereditary cardiovascular disorders. By combining comprehensive postmortem evaluation with family- and phenotype-directed genetic testing, the structured NODOV procedure extends the scope of postmortem investigation beyond establishing the cause of death, enabling targeted (pretest) genetic counselling, cascade screening, and individualized follow-up of at-risk relatives.

These findings support the inclusion of genetic counselling and postmortem genetic testing as an integral component of structured sudden unexplained death investigations in young adults, with the potential to improve forensic diagnosis and facilitate preventive management aimed at reducing adverse cardiovascular events in affected families.

Declarations

Ethical Approval

The study proposal was reviewed by the Medical Research Ethics Committees of Maastricht University (reference number 2022-3148), Isala Hospital Zwolle (reference number 220901) and the University of Groningen (reference number NODOV; 20818). All three committees issued statements confirming that this research does not fall under the Dutch Medical Research Involving Human Subjects Act.

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Conflict of interest

None declared.

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Figures

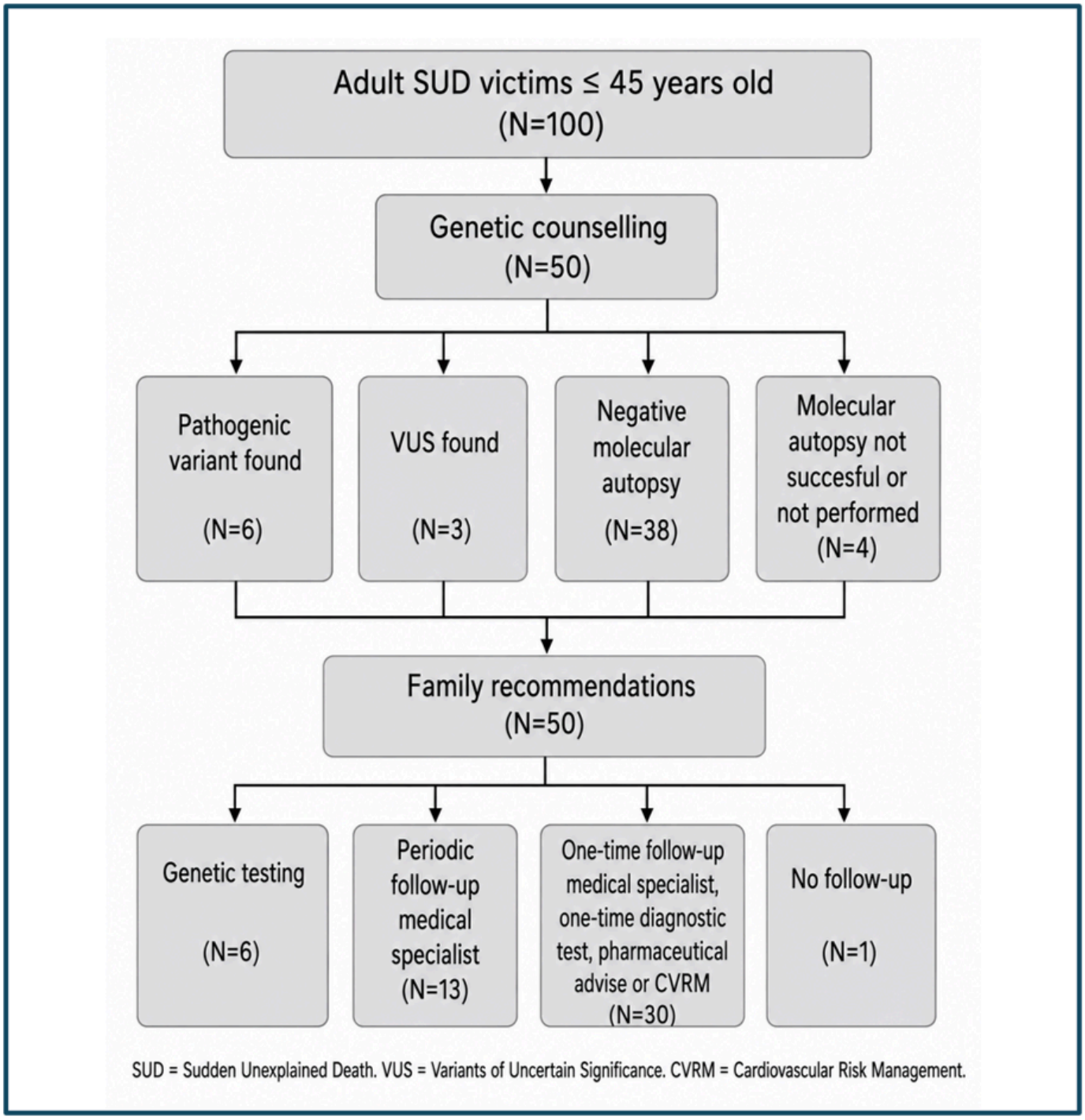


Figure 1

Flowchart of 50 selected cases that underwent genetic counselling and molecular autopsy: Test results and family recommendations given. The highest level of advice given by the clinical geneticist to the 50 families is recorded under family recommendations, irrespective of whether families decided to follow through or multiple recommendations were given within one family.

Supplementary Files

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