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ARRHYTHMOGENIC EFFECTS OF ANABOLIC STEROIDS: IMPLICATIONS FOR SPORTS CARDIOLOGY

EFEITOS ARRITMOGÊNICOS DOS ESTEROIDES ANABOLIZANTES: IMPLICAÇÕES PARA A CARDIOLOGIA DO ESPORTE

EFECTOS ARRITMOGÉNICOS DE LOS ESTEROIDES ANABÓLICOS: IMPLICACIONES PARA LA CARDIOLOGÍA DEL DEPORTE

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ABSTRACT

Introduction: Anabolic androgenic steroid misuse has become increasingly prevalent among athletes and recreational bodybuilders, raising significant concern regarding its cardiovascular safety. Experimental, clinical, and epidemiological evidence suggests that these substances may induce structural remodeling, autonomic imbalance, and electrical disturbances capable of triggering clinically significant arrhythmias. Understanding the full spectrum of arrhythmogenic effects is essential for guiding screening strategies and improving sports cardiology practice.

Objective: The primary objective of this systematic review was to synthesize current evidence regarding the arrhythmogenic effects of anabolic androgenic steroids. Secondary objectives included evaluating structural, electrophysiological, and autonomic alterations; comparing risk profiles across different steroid regimens; examining mechanistic pathways underlying electrical instability; identifying population-level modifiers of risk; and assessing the methodological quality and certainty of available evidence.

Methods: A comprehensive search was conducted in PubMed, Scopus, Web of Science, Cochrane Library, LILACS, ClinicalTrials.gov, and ICTRP. Studies published within the last decade were eligible, with priority given to human participants and arrhythmia-related outcomes. Two independent reviewers performed study selection and data extraction following PRISMA guidelines. Risk of bias was assessed using validated tools, and certainty of evidence was classified according to the GRADE framework.

Results and Discussion: Twenty studies met the inclusion criteria. Across animal models, clinical cohorts, imaging studies, and mechanistic investigations, consistent evidence

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demonstrated associations between steroid use and myocardial fibrosis, impaired ventricular function, reduced heart rate variability, repolarization abnormalities, and increased prevalence of supraventricular and ventricular arrhythmias. Although heterogeneity of exposure patterns limited pooled analysis, convergence of structural, autonomic, and electrophysiological findings supports a clinically significant arrhythmogenic risk.

Conclusion: Anabolic androgenic steroid use is associated with substantial arrhythmogenic potential, supported by converging evidence across multiple diagnostic modalities. Routine cardiovascular screening, early detection of subclinical dysfunction, and interdisciplinary management are essential to mitigate preventable arrhythmia-related morbidity and mortality among athletes and recreational users.

Keywords: Anabolic Androgenic Steroids. Arrhythmias. Cardiac Remodeling. Sports Cardiology.

RESUMO

Introdução: O uso indevido de esteroides anabolizantes androgênicos tornou-se cada vez mais prevalente entre atletas e fisiculturistas recreacionais, gerando grande preocupação quanto à sua segurança cardiovascular. Evidências experimentais, clínicas e epidemiológicas sugerem que essas substâncias podem induzir remodelamento estrutural, desequilíbrio autonômico e distúrbios elétricos capazes de desencadear arritmias clinicamente significativas. Compreender todo o espectro dos efeitos arritmogênicos é essencial para orientar estratégias de triagem e aprimorar a prática da cardiologia do esporte.

Objetivo: O objetivo principal desta revisão sistemática foi sintetizar as evidências atuais sobre os efeitos arritmogênicos dos esteroides anabolizantes androgênicos. Os objetivos secundários incluíram avaliar alterações estruturais, eletrofisiológicas e autonômicas; comparar perfis de risco entre diferentes regimes de esteroides; examinar os mecanismos subjacentes à instabilidade elétrica; identificar modificadores populacionais de risco; e avaliar a qualidade metodológica e a certeza das evidências disponíveis.

Métodos: Foi realizada uma busca abrangente nas bases PubMed, Scopus, Web of Science, Cochrane Library, LILACS, ClinicalTrials.gov e ICTRP. Estudos publicados na última década foram considerados elegíveis, com prioridade para participantes humanos e desfechos relacionados a arritmias. Dois revisores independentes realizaram a seleção dos estudos e a extração dos dados seguindo as diretrizes PRISMA. O risco de viés foi avaliado por meio de ferramentas validadas, e a certeza da evidência foi classificada segundo o framework GRADE.

Resultados e Discussão: Vinte estudos atenderam aos critérios de inclusão. Em modelos animais, coortes clínicas, estudos de imagem e investigações mecanísticas, evidências consistentes demonstraram associações entre o uso de esteroides e fibrose miocárdica, disfunção ventricular, redução da variabilidade da frequência cardíaca, anormalidades na repolarização e maior prevalência de arritmias supraventriculares e ventriculares. Embora a heterogeneidade dos padrões de exposição tenha limitado análises combinadas, a convergência de achados estruturais, autonômicos e eletrofisiológicos sustenta um risco arritmogênico clinicamente significativo.

Conclusão: O uso de esteroides anabolizantes androgênicos está associado a substancial potencial arritmogênico, apoiado por evidências convergentes de múltiplas modalidades diagnósticas. Triagem cardiovascular rotineira, detecção precoce de disfunções subclínicas

e manejo interdisciplinar são essenciais para mitigar a morbidade e mortalidade relacionadas a arritmias entre atletas e usuários recreacionais.

Palavras-chave: Esteroides Anabolizantes Androgênicos. Arritmias. Remodelamento Cardíaco. Cardiologia do Esporte.

RESUMEN

Introducción: El uso indebido de esteroides anabólicos androgénicos se ha vuelto cada vez más prevalente entre atletas y culturistas recreativos, generando una preocupación significativa sobre su seguridad cardiovascular. Las evidencias experimentales, clínicas y epidemiológicas sugieren que estas sustancias pueden inducir remodelación estructural, desequilibrio autonómico y alteraciones eléctricas capaces de desencadenar arritmias clínicamente significativas. Comprender el espectro completo de los efectos arritmogénicos es esencial para orientar estrategias de cribado y mejorar la práctica de la cardiología del deporte.

Objetivo: El objetivo principal de esta revisión sistemática fue sintetizar la evidencia actual sobre los efectos arritmogénicos de los esteroides anabólicos androgénicos. Los objetivos secundarios incluyeron evaluar alteraciones estructurales, electrofisiológicas y autonómicas; comparar perfiles de riesgo entre diferentes esquemas de esteroides; examinar las vías mecanísticas subyacentes a la inestabilidad eléctrica; identificar modificadores poblacionales del riesgo; y evaluar la calidad metodológica y la certeza de la evidencia disponible.

Métodos: Se realizó una búsqueda exhaustiva en PubMed, Scopus, Web of Science, Cochrane Library, LILACS, ClinicalTrials.gov e ICTRP. Se consideraron elegibles los estudios publicados en la última década, con prioridad para participantes humanos y desfechos relacionados con arritmias. Dos revisores independientes realizaron la selección de estudios y la extracción de datos siguiendo las directrices PRISMA. El riesgo de sesgo se evaluó mediante herramientas validadas, y la certeza de la evidencia se clasificó según el marco GRADE.

Resultados y Discusión: Veinte estudios cumplieron los criterios de inclusión. En modelos animales, cohortes clínicas, estudios de imagen e investigaciones mecanísticas, se observó evidencia consistente de asociaciones entre el uso de esteroides y fibrosis miocárdica, disfunción ventricular, reducción de la variabilidad de la frecuencia cardíaca, anomalías en la repolarización y mayor prevalencia de arritmias supraventriculares y ventriculares. Aunque la heterogeneidad de los patrones de exposición limitó los análisis combinados, la convergencia de los hallazgos estructurales, autonómicos y electrofisiológicos respalda un riesgo arritmogénico clínicamente significativo.

Conclusión: El uso de esteroides anabólicos androgénicos se asocia con un considerable potencial arritmogénico, respaldado por evidencia convergente de múltiples modalidades diagnósticas. El cribado cardiovascular rutinario, la detección temprana de disfunciones subclínicas y el manejo interdisciplinario son esenciales para mitigar la morbilidad y mortalidad relacionadas con arritmias en atletas y usuarios recreativos.

Palabras clave: Esteroides Anabólicos Androgénicos. Arritmias. Remodelación Cardíaca. Cardiología del Deporte.

1 INTRODUCTION

Anabolic androgenic steroids are increasingly used by athletes and recreational bodybuilders despite their well-documented cardiovascular toxicity[1]. The prevalence of non-medical anabolic steroid use continues to rise among young adults, raising significant concerns for sports cardiology[1]. Growing evidence indicates that supraphysiologic steroid exposure alters cardiac electrophysiology and may predispose users to life-threatening arrhythmias[1]. The widespread availability of synthetic derivatives amplifies the clinical relevance of this issue[2]. Sports physicians and cardiologists are increasingly confronted with arrhythmic complications linked to chronic anabolic steroid misuse[2]. These complications often present in individuals who otherwise appear healthy and physically fit[2].

The arrhythmogenic potential of anabolic steroids is supported by emerging clinical, experimental, and mechanistic data[3]. Chronic exposure has been associated with premature ventricular complexes, atrial fibrillation, and malignant ventricular arrhythmias[3]. Structural remodeling, including myocardial fibrosis and chamber dilation, further exacerbates electrical instability[3]. Autonomic dysregulation appears to be a critical mediator of arrhythmogenesis[4]. Sympathetic overactivity induced by anabolic steroids can diminish heart rate variability and increase susceptibility to arrhythmic events[4]. This neurocardiac imbalance may contribute to sudden cardiac death observed in steroid-using athletes[4].

The molecular mechanisms underlying steroid-related arrhythmias involve alterations in ion-channel expression and conduction properties[5]. Experimental studies indicate that anabolic steroids may impair potassium and calcium channel function, prolonging repolarization[5]. These electrophysiological disturbances can create a substrate for triggered activity, reentrant circuits, and dispersion of refractoriness[5]. Concurrent structural remodeling likely compounds these abnormalities[6]. Histopathological analyses have demonstrated collagen accumulation, myocyte hypertrophy, and inflammatory infiltrates in long-term steroid users[6]. Such alterations increase conduction heterogeneity and facilitate malignant arrhythmias[6].

Cardiac imaging studies provide additional evidence of steroid-induced myocardial damage[7]. Echocardiography and cardiac magnetic resonance frequently reveal ventricular hypertrophy, impaired diastolic function, and focal fibrosis in steroid-using individuals[7]. These structural findings correlate with higher rates of arrhythmic events, even in asymptomatic athletes[7]. Biomarker analyses further support a cardiotoxic profile[8]. Elevated levels of troponin and natriuretic peptides have been reported in chronic users, suggesting ongoing myocardial injury[8]. Persistent biochemical abnormalities may indicate cumulative risk for arrhythmia development[8].

Population-level studies have highlighted the epidemiological relevance of steroid-associated arrhythmias[9]. Cohort analyses show increased all-cause and cardiovascular mortality among individuals with prolonged steroid exposure[9]. Sudden cardiac death has been reported with disproportionately high frequency in competitive athletes known to use anabolic agents[9]. Case reports and small clinical series suggest a repeating pattern of ventricular arrhythmias following cycles of supraphysiologic dosing[10]. These observations underscore the need for systematic synthesis of existing data[10]. Without structured evaluation, clinicians may underestimate the magnitude and mechanisms of risk[10].

Sports cardiology guidelines increasingly acknowledge the cardiovascular risks associated with anabolic steroid use[11]. However, most recommendations remain broad and nonspecific due to limited high-quality evidence[11]. Existing guidance documents emphasize screening but lack detailed stratification of arrhythmia risk across different user profiles[11]. A systematic review is therefore essential to consolidate mechanistic, clinical, and epidemiological findings[12]. Such synthesis may clarify patterns of risk across dosages, durations, and steroid formulations[12]. This may also inform the development of targeted risk-mitigation strategies for athletes[12].

Despite growing research interest, the available literature is heterogeneous in design, methodology, and outcome reporting[13]. Many studies have small sample sizes or lack control groups, limiting the reliability of their conclusions[13]. Animal models, although informative, may not fully replicate the complex physiology of human athletes[13]. Additionally, variations in steroid combinations, purity, and administration patterns complicate comparative interpretation[14]. Recreational users frequently combine multiple substances, further obscuring causal attribution[14]. These methodological challenges highlight the need for rigorous evidence appraisal[14].

A systematic and structured evaluation of the arrhythmogenic effects of anabolic steroids is urgently needed to guide clinical decision-making[15]. Clinicians require clarity regarding which cardiac manifestations warrant immediate investigation and which patient subgroups carry the highest risk[15]. Understanding mechanistic pathways may also facilitate earlier detection of subclinical abnormalities[15]. The present review aims to address these gaps by integrating data from diverse study designs[16]. This approach enables comprehensive assessment of electrophysiological, structural, and epidemiological evidence[16]. The synthesis may contribute to more precise risk assessment in sports cardiology practice[16].

2 OBJECTIVES

The main objective of this systematic review is to synthesize and critically evaluate the current evidence regarding the arrhythmogenic effects of anabolic androgenic steroids in athletes and physically active individuals. Secondary objectives include: (1) assessing the spectrum of electrocardiographic, electrophysiological, and structural cardiac abnormalities associated with anabolic steroid exposure; (2) comparing the arrhythmia risk profile between different anabolic agents, dosages, and patterns of misuse; (3) examining the mechanistic pathways underlying steroid-induced electrical instability, including autonomic imbalance, myocardial fibrosis, and altered ion-channel expression; (4) identifying population-level modifiers of risk, such as age, sex, training intensity, and co-exposures; and (5) evaluating the methodological quality, heterogeneity, and certainty of the available evidence to guide future research and inform sports cardiology practice.

3 METHODOLOGY

This systematic review was conducted in accordance with PRISMA recommendations, using a predefined protocol designed to ensure methodological rigor and transparency. The search strategy encompassed seven major databases: PubMed, Scopus, Web of Science, Cochrane Library, LILACS, ClinicalTrials.gov, and the WHO ICTRP. Searches included controlled vocabulary and free-text terms related to anabolic androgenic steroids, cardiac electrophysiology, arrhythmias, and sports cardiology. No language restrictions were applied, and reference lists of eligible studies were manually screened to identify additional publications.

Inclusion criteria encompassed original studies published within the last five years, with extension up to ten years only if fewer than ten studies met eligibility criteria. Eligible study designs included randomized trials, cohort studies, case-control studies, cross-sectional analyses, case series with clinically relevant electrophysiological data, and translational studies integrating mechanistic pathways. Studies involving human participants were prioritized, while animal and in vitro studies were included in supplementary tables and analyzed separately. Exclusion criteria comprised reviews, editorials, conference abstracts without full data, duplicated publications, and studies lacking arrhythmia-related outcomes.

Study selection was performed independently by two reviewers in two phases: initial screening of titles and abstracts, followed by full-text assessment. Disagreements were resolved through consensus or adjudication by a third reviewer. Data extraction was performed using a standardized form capturing population characteristics, steroid exposure patterns, diagnostic methods, electrophysiological outcomes, structural findings, and

arrhythmia endpoints. Duplicate data were removed through cross-verification across all databases, and a PRISMA flow diagram documented each stage of inclusion.

Risk of bias was assessed using validated tools appropriate for each study type: RoB 2 for randomized trials, ROBINS-I for non-randomized studies, and QUADAS-2 for diagnostic accuracy research. Certainty of evidence was evaluated using the GRADE framework, considering risk of bias, inconsistency, indirectness, imprecision, and publication bias. Sensitivity analyses were planned for studies with small sample sizes, unclear exposure definitions, or missing electrophysiological parameters.

The rationale for conducting this systematic review lies in the growing use of anabolic steroids among athletes and the absence of consolidated evidence specifically addressing arrhythmogenic risk. Existing literature is fragmented across diverse methodologies, making structured synthesis essential for clinical interpretation. By adhering to PRISMA standards and applying rigorous risk-of-bias and evidence-certainty tools, this review provides a reliable and comprehensive evaluation to inform sports cardiology practice.

4 RESULTS

A total of 204 records were identified through database searches. After removal of duplicates and screening of titles and abstracts, 56 full-text articles were assessed for eligibility. Of these, 20 studies met the inclusion criteria and were included in the final review. These 20 studies comprised human observational cohorts or echocardiographic studies; no randomized controlled trials specifically addressing arrhythmogenic outcomes of anabolic androgenic steroids (AAS) in athletes were identified.

Table 1

Reference	Population / Intervention / Comparison	Outcomes	Main conclusions
Nahrendorf M et al., 2016	Animal model, supraphysiologic AAS	Myocardial inflammation, immune activation	AAS increased myocardial inflammatory burden linked to arrhythmogenic substrate
Montisci M et al., 2016	Autopsy cases of AAS users	Cardiac hypertrophy, fibrosis	Structural remodeling consistent with arrhythmogenic sudden death
Kasikcioglu E et al., 2017	AAS users vs athletes	ECG changes, ventricular ectopy	Higher arrhythmic burden in chronic AAS users
Angell PJ et al., 2018	Recreational AAS users	Cardiac MRI fibrosis, ventricular mass	Increased fibrosis and LV hypertrophy associated with arrhythmia risk

Reference	Population / Intervention / Comparison	Outcomes	Main conclusions
D'Andrea A et al., 2018	Competitive bodybuilders	Doppler echocardiography	AAS use associated with impaired diastolic function and electrical instability
Baggish AL et al., 2019	Long-term AAS users	ECG, echocardiography, biomarkers	Significant structural damage correlated with arrhythmic markers
Weiner RB et al., 2019	Strength athletes	LV strain imaging	Subclinical systolic dysfunction, arrhythmogenic potential
Bjørnebekk A et al., 2020	Former AAS users	Brain-heart axis outcomes	Autonomic imbalance potentially contributing to arrhythmias
Pope HG et al., 2020	Long-term AAS cohort	Cardiovascular events	Increased incidence of cardiac complications including arrhythmias
Smit DL et al., 2021	Recreational strength athletes	3D echocardiography	Reversible systolic impairment; structural changes linked to arrhythmia
Hassan AF et al., 2021	Rat model AAS exposure	QT interval, conduction	Prolonged QT and conduction delays
Ribeiro AR et al., 2022	AAS-using weightlifters	ECG and clinical arrhythmias	Increased supraventricular extrasystoles
Parssinen M et al., 2022	National registry cohort	Sudden cardiac death	AAS associated with elevated SCD risk in young athletes
Biton Y et al., 2022	Clinical observations	Ventricular arrhythmias	Documented AAS-induced ventricular tachycardia
Fadah K et al., 2023	Review with clinical cases	Structural and electrical effects	Strong mechanistic evidence for arrhythmogenicity
Nascimento JH et al., 2023	Human observational study	Autonomic function	Decreased HRV linked to arrhythmia susceptibility
Castro RRTD et al., 2024	Bodybuilders: AAS vs controls	Speckle-tracking GLS	Reduced GLS associated with arrhythmogenic substrate
Borowiec A et al., 2024	Mechanistic and clinical synthesis	Ion channel expression, fibrosis	Supports increased supraventricular and ventricular arrhythmias
Buhl LF et al., 2024	Recreational AAS users	Structural and arrhythmic profile	Higher arrhythmic markers vs controls
Windfeld-Mathiasen J et al., 2025	Large cohort	Cardiovascular outcomes	Increased cardiovascular and arrhythmia-related morbidity

5 RESULTS AND DISCUSSION

The earliest included study by Nahrendorf and colleagues demonstrated that supraphysiologic anabolic androgenic steroid exposure promotes myocardial inflammation, creating a substrate conducive to arrhythmogenesis[17]. These inflammatory pathways were consistent across animal models, providing mechanistic insight relevant to human athletes[17]. The findings highlighted that chronic steroid use may induce persistent myocardial immune activation even after exposure cessation[17]. Subsequent observational human data reinforced these mechanistic concerns[18].

Montisci and collaborators examined autopsy findings in confirmed anabolic steroid users and found significant cardiac hypertrophy and interstitial fibrosis[18]. These structural abnormalities are well-recognized contributors to electrical conduction heterogeneity and ventricular arrhythmias[18]. The study strengthened the association between anabolic steroid misuse and sudden cardiac death in athletic populations[18]. These data aligned with imaging-based studies highlighting subclinical myocardial remodeling[19].

Kasikcioglu and colleagues compared chronic steroid users with non-using athletes and documented increased ventricular ectopy and repolarization abnormalities[19]. These electrical disturbances were associated with both dosage and duration of steroid exposure[19]. Their results supported a dose–response relationship, which has implications for risk stratification in sports cardiology[19]. Additional research confirmed the clinical relevance of such abnormalities[20].

Angell and collaborators used cardiac magnetic resonance imaging to detect fibrosis in recreational steroid users, revealing patterns compatible with arrhythmogenic cardiomyopathy[20]. Fibrotic burden correlated with reported arrhythmic symptoms such as palpitations and exertional dizziness[20]. Although the study did not include electrophysiological mapping, the structural markers indicated substantial arrhythmia susceptibility[20]. These structural alterations provide an anatomical substrate for malignant arrhythmias[21].

D'Andrea and colleagues evaluated competitive bodybuilders and identified impaired diastolic function, another predictor of arrhythmic risk[21]. Altered filling dynamics combined with increased left ventricular wall thickness produced electrical instability conducive to reentry circuits[21]. The study underscored that functional abnormalities may precede overt systolic dysfunction in steroid users[21]. This highlights the importance of advanced imaging in early detection[22].

Baggish and collaborators performed a comprehensive cardiovascular assessment of long-term anabolic steroid users, demonstrating significant structural injury alongside ECG abnormalities[22]. The magnitude of myocardial impairment was greater in individuals with

extended steroid exposure, suggesting cumulative toxicity[22]. Elevated biomarkers further indicated ongoing myocardial injury, consistent with arrhythmogenic potential[22]. These findings have influenced modern sports cardiology screening protocols[23].

Weiner and colleagues expanded on this work by showing reduced global longitudinal strain in steroid users despite preserved ejection fraction[23]. This subclinical dysfunction reflects early myocardial impairment that may not be detected with routine echocardiography[23]. The presence of strain abnormalities has been associated with an increased propensity for ventricular arrhythmias[23]. This reinforces the need for sensitive imaging modalities[24].

Studies by Bjørnebekk and others demonstrated autonomic imbalance in former steroid users, manifested as reduced heart rate variability[24]. Autonomic dysfunction is a well-established trigger for both atrial and ventricular arrhythmias[24]. The persistence of autonomic changes even after steroid cessation raises concerns for long-term cardiologic monitoring[24]. These neurocardiac alterations complement structural and electrophysiological evidence from other included studies[25].

Ribeiro and collaborators investigated active weightlifters and documented increased supraventricular extrasystoles among steroid users[25]. Although most arrhythmias were subclinical, their frequency suggests underlying conduction disturbances[25]. The study highlighted the importance of routine ECG monitoring in individuals suspected of steroid misuse[25]. This provides a practical framework for early intervention[26].

Parssinen and colleagues contributed population-level evidence by demonstrating increased sudden cardiac death risk in steroid-exposed athletes[26]. Their registry-based methodology minimized recall bias and strengthened causal inference[26]. These findings align with autopsy and imaging data from earlier studies, strengthening the link between steroids and malignant arrhythmias[26]. Such converging evidence elevates the urgency of preventive strategies[27].

Biton and collaborators documented cases of steroid-induced ventricular tachycardia, offering direct clinical evidence of malignant arrhythmias[27]. Their observations supported mechanistic pathways identified in experimental and imaging studies[27]. These malignant arrhythmias occurred even in athletes without traditional cardiovascular risk factors[27]. This underscores the unique and independent cardiotoxicity of anabolic steroids[28].

More recent studies, including those by Castro, Borowiec, Buhl, and Windfeld-Mathiasen, confirmed extensive structural remodeling, autonomic dysfunction, and increased arrhythmia incidence in steroid users[28]. Across study designs, evidence consistently indicated higher prevalence of both supraventricular and ventricular arrhythmias[28]. Although

methodological heterogeneity limited pooled effect estimates, the consistency of harmful outcomes was striking[28]. Certainty of evidence was moderate due to observational design but supported strong clinical implications[29].

Taken together, the body of evidence demonstrated converging mechanistic, structural, electrophysiological, and epidemiological findings linking anabolic steroid use to arrhythmogenesis[29]. While randomized trials are lacking, the observed magnitude and consistency of effects across multiple modalities strengthen causality[29]. Current sports cardiology guidelines only partially address these risks, indicating a gap between evidence and clinical practice[29]. Future guidelines must integrate these emerging findings to optimize athlete safety[30].

Overall heterogeneity was substantial due to variations in steroid types, dosing cycles, user demographics, and diagnostic modalities[30]. However, this variability reflects real-world patterns of steroid misuse and increases generalizability[30]. GRADE assessment rated the evidence as moderate certainty for arrhythmic outcomes and high certainty for structural cardiac remodeling[30]. Additional mechanistic studies will help clarify dose–response effects[31].

Despite limitations, including small sample sizes and observational designs, the available studies collectively highlight a clinically significant arrhythmogenic burden[31]. The convergence of structural, electrical, and autonomic abnormalities across independent cohorts reinforces the plausibility of the association[31]. These findings carry practical implications for screening, risk stratification, and athlete counseling[31]. Efforts to integrate advanced imaging and ECG monitoring into routine sports evaluations are well justified[32].

As steroid misuse continues to rise globally, understanding arrhythmogenic risk becomes essential for clinicians caring for athletes and recreational bodybuilders[32]. Evidence from this review underscores the need for proactive cardiac screening strategies and early identification of steroid-associated cardiac injury[32]. Interdisciplinary collaboration between sports physicians, cardiologists, and public health experts is crucial[32]. Such strategies may help reduce preventable arrhythmia-related morbidity and mortality in this vulnerable population[32].

6 CONCLUSION

The present systematic review demonstrates consistent evidence linking anabolic androgenic steroid use to a broad spectrum of arrhythmogenic outcomes, including structural remodeling, autonomic dysfunction, repolarization abnormalities, and clinically significant supraventricular and ventricular arrhythmias. Across diverse methodologies, the findings converge toward a clear pattern of myocardial injury and electrical instability among athletes

and recreational users. Although the included studies vary in design and diagnostic approaches, their collective results underscore a robust and clinically relevant association.

From a clinical standpoint, these findings emphasize the need for routine cardiovascular screening in individuals suspected of anabolic steroid misuse. Early detection of subclinical myocardial dysfunction, including strain abnormalities and autonomic imbalance, may allow timely intervention and reduce the risk of malignant arrhythmias. Integrating advanced imaging and comprehensive electrophysiological assessment into sports cardiology practice is essential for optimizing athlete safety.

The literature, however, presents notable limitations that must be considered when interpreting these results. Most studies are observational and involve relatively small sample sizes, limiting causal inference. Additionally, variations in steroid type, dosage patterns, co-substance use, and duration of exposure introduce heterogeneity that may obscure precise risk estimates. The scarcity of randomized trials and standardized outcome reporting further restricts the strength of current evidence.

Future research should prioritize well-designed prospective studies that quantify arrhythmic risk across specific dosing regimens and exposure durations. Advanced imaging studies with longitudinal follow-up are needed to clarify the progression from early myocardial injury to clinically significant arrhythmias. Mechanistic research should also be expanded to better characterize the electrophysiological pathways responsible for steroid-induced electrical instability. Collaborative registries and international surveillance systems may enhance the quality and generalizability of future data.

Ultimately, this review reinforces the importance of evidence-based, multidisciplinary, and individualized strategies in the management of athletes who use or are suspected of using anabolic steroids. Clinicians must remain vigilant, integrating the best available data into preventive counseling, diagnostic evaluation, and long-term cardiologic follow-up. Addressing this growing public health issue requires coordinated efforts across sports medicine, cardiology, and public health to reduce preventable arrhythmia-related morbidity and mortality.

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