REVIEW PAPER

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The impact of anabolic-androgenic steroids used by athletes on the cardiovascular system

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ABSTRACT

Introduction. In many countries, the use of anabolic-androgenic steroids (AAS) has become increasingly common among individuals who engage in physical activity and wish to improve their body composition with particular influence on increasing muscle mass. This phenomenon affects not only professional sports competitors but also amateur athletes and recreational exercisers. Easy access to these preparations, both through online sales and illegal trade, encourages the widespread use of AAS, despite their well-documented adverse health effects. With regard to the cardiovascular system, these substances may cause many pathological changes leading to the development of hypertension, myocardial ischemia, heart failure or the occurrence of sudden cardiac death. The mechanisms underlying these processes are multidirectional, on the one hand resulting from direct structural damage to the heart and blood vessels, and on the other hand indirectly inducing lipid disorders or prothrombotic processes.

Material and methods. A comprehensive literature review was conductes usuing PubMed and Google Scholar databases to identify studies investigating the impact of AAS on the cardiovascular system Keywords covered "anabolic-androgenic steroids", "cardiovascular risk", "myocardial hypertrophy", and "AAS abuse".
Results. This review synthesizes current knowledge on the multidirectional pathophysiological mechanisms of AAS. Although the available scientific evidence alearly indicates a significant cardiovascular risk associated with these substances, the detailed molecular pathways have not yet been explained.
Conclusions. The aim of the study was to inform clinicians and healthcare providers about the significant cardiovascular risks associated with AAS use, which is becoming an increasing health problem in clinical practice.

Introduction

Anabolic-androgenic steroids (AAS) constitute a broad group of substances, including endogenous hormones such as testosterone (T) and dihydrotestosterone (DHT), as well as synthetic derivatives such as nandrolone and methandienone (see **Figure 1**). These agents possess both anabolic (tissue-building) and androgenic (masculinising) properties.

Despite well-documented harmful effects on multiple organ systems, the non-medical use of AAS is prevalent among both professional and amateur athletes, representing a growing global public health issue. In 2010, it was estimated that nearly 3,000,000 people in the United States used these substances [1]. Globally, the prevalence of AAS use among amateur and professional athletes is reported to be approximately 6.4% in males and 1.6% in females [2]. Other data suggest that up to 98% of users are male, with this proportion predominantly observed in amateur sports [3]. The reported incidence of adverse effects is high, with estimates suggesting that 90% of users experience them [4]. Despite the gravity of the issue, public awareness of the dangerous consequences of AAS use remains low, and healthcare professionals often lack sufficient knowledge.

The problem is exacerbated by the easy and largely uncontrolled access to AAS through

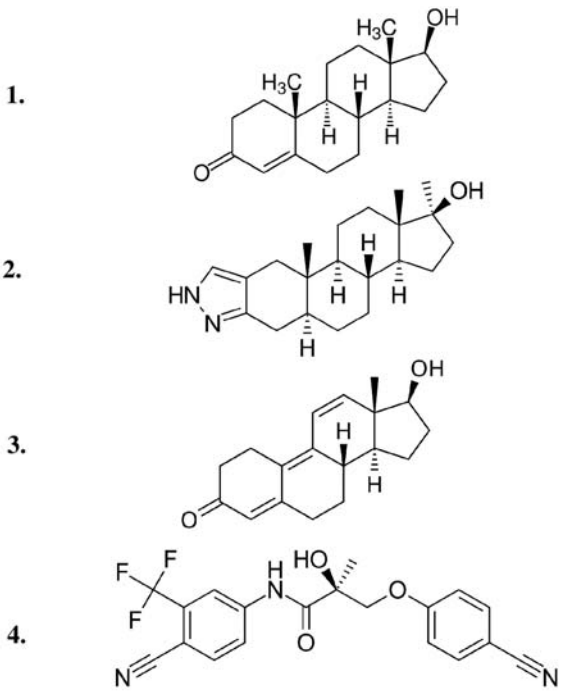

Figure 1. Structural formulas of compounds belonging to specific AAS classifications. 1 – Testosterone; 2 – Stanozolol; 3 – Trenbolone; 4 – Enobosarm.

online sources and illegal private trade, often in sports clubs. Studies conducted in the United States indicate a considerable, sustained annual growth in AAS sales, ranging from over 10% to nearly 20% [5].

The legal status of AAS varies by country. For instance, under Polish law, the possession of small quantities of AAS for personal use is not criminalised. Such regulations, however, may contribute to the widespread use of these substances among amateur exercisers [6]. Conversely, at the competitive sports level, AAS are completely banned by the World Anti-Doping Agency, being classified under group S1 on the list of prohibited substances and methods in sports. The use of AAS constitutes a doping violation and results in an athlete's disqualification [7]. Therapeutic use of T requires a prior Therapeutic Use Exemption, which is restricted to cases of hypogonadism of organic aetiology (e.g., genetic or developmental abnormalities, bilateral testicular trauma or torsion, and orchitis), not functional states [8].

Despite the risk of disqualification from sports competitions, the development of new synthetic agents continues, often involving modification of the basic steroid structure, primarily through esterification at the 17β-position [9]. This modification aims to maximise anabolic potential while reducing androgenic effects. The potency of AAS is often compared to T, which has an anabolic-to-androgenic ratio of 100:100. Compounds such as methandienone (1000:200) and certain selective androgen receptor modulators (SARMs) (up to 2000:100) demonstrate superior anabolic activity [10]. The actual effect on the body, however, is also dependent on the dose, the duration of the cycle, and individual predispositions. Examples from various AAS groups, based on differences in their structural composition, are presented in **Figure 1**.

This study aims to summarise the current knowledge of the mechanisms of AAS action and their impact on the cardiovascular system, as well as the prevalence of adverse cardiac events.

Materials and methods

This narrative review synthesised data from PubMed and Google Scholar using keywords to identify studies investigating the cardiovascular effects of AAS. The keywords covered "anabolic-androgenic steroids", "testosterone", "athletes", "AAS abuse", "cardiovascular risk", "myocardial hypertrophy", "fibrosis", "hypertension", "myocardial infarction", "thrombosis", "heart failure", and "sudden cardiac death".

Methodology

The selection process for research studies was based on the author's qualitative assessment of clinical relevance. From a total of 320 identified records, 70 sources were selected for detailed analysis, primarily original research, clinical trials, meta-analyses, and significant case reports. Inclusion criteria focused on studies documenting molecular mechanisms of action, structural heart changes and clinical cardiovascular outcomes.

Similar reviews have been conducted in the past. Still, a unique feature of this review is its integration of detailed descriptions of the pathomechanisms underlying AAS effects alongside well-documented clinical studies and outcomes.

Mechanism of action

AAS exert their effects via both direct and indirect cellular pathways. The direct genomic pathway (the primary mechanism) involves binding to intracellular androgen receptors (AR). This binding induces a conformational change, leading to dimerisation and translocation into the cell nucleus, where the complex modulates the transcription of androgen-dependent genes [11]. The direct non-genomic pathway, a recently discovered mechanism, involves the activation of specific G protein-coupled receptors. This action causes rapid changes in intracellular ion levels, such as calcium ions (Ca^{2+}). This effect was initially observed in macrophages; however, it may also occur in other cells that express these membrane receptors [12]. The indirect (estrogenic) pathway involves AAS being converted to estradiol, specifically 17β-estradiol, via the enzyme aromatase (an oxidoreductase). The resulting estradiol binds to its receptor, which then translocates to the nucleus to bind to the estradiol response region on DNA, thereby triggering the expression of downstream genes [11].

Effects of AAS on the human body

The primary desired outcome of AAS is an anabolic effect achieved through increased nitrogen retention and subsequent stimulation of protein synthesis, notably within skeletal muscle structural proteins such as myosin and actin [13]. This results in skeletal muscle hypertrophy and, to a lesser extent, stimulates myogenesis by muscle satellite cells [14]. The secondary outcome is the development of male sexual characteristics (androgenic effect). In clinical medicine, these properties are utilised for the treatment of conditions such as hypogonadism and delayed puberty. Current research is exploring the therapeutic use of T, particularly in men with heart failure with preserved ejection fraction and malnutrition, where T deficiency (the cut-off point in this study was 2.3 ng/mL, although 3.5 ng/ml is generally considered the lower limit of normal range) was, along with age and B-type natriuretic peptide (BNP) serum concentration, an independent predictor of clinical heart failure progression [15]. However, emerging data indicate that T may reduce the risk of all-cause mortality and acute heart failure in this selected group. A tendency toward a higher risk of venous thromboembolism, transient ischemic attack and stroke has also been observed [16].

The use of supraphysiological doses of AAS is linked to numerous adverse effects. The aromatisation of T to estradiol can lead to gynecomastia, adipose tissue accumulation, erectile dysfunction, and decreased libido [11,17,18]. Conversely, the conversion of T to DHT by 5α-reductase promotes male pattern baldness, benign prostate hypertrophy, acne, and various cardiovascular complications [18]. Importantly, most of the toxic effects of AAS result from doses that significantly exceed therapeutic levels. Users commonly take doses five to fifteen times higher than therapeutic recommendations, dramatically increasing health risks [19]. The systemic burden and risk of serious side effects are further exacerbated by the practice of using multiple agents simultaneously in "steroid cycles", often following a "pyramidal" scheme for up to 48 weeks.

Recent literature increasingly highlights the use of AAS among athletes as one of the causes of premature death that should be addressed. The problem has prompted debate about the need to implement an even broader range of preventive and educational measures in this area [20–22].

Testosterone – the most commonly used performance-enhancing substance

Testosterone, the main androgen hormone, plays a key role in the development of male sexual characteristics, in maintaining reproductive function, and in influencing many aspects of the body's functioning (see **Figure 1**). In men, it is produced primarily by Leydig cells in the testicles under the control of luteinizing hormone (LH) and, to a much lesser degree, by the adrenal glands [23]. The substrate for this reaction is cholesterol, converted to pregnenolone in the mitochondria. Subsequently, through a series of reactions, androstenedione is formed, which is ultimately reduced to T [23,24]. The hormone circulates in both its unbound (bioavailable) form and conjugated to proteins, primarily sex hormone-binding globulin (SHBG) and albumin.

From a chemical standpoint, T is a steroid composed of four fused carbon rings, belonging to the 17β-hydroxysteroid group. It has a characteristic structure based on two important functional groups, i.e., the ketone and hydroxyl groups [23]. The 20th century proved to be a breakthrough moment in the field of andrology, when three independent laboratories synthesised T. Today, T is widely used for non-medical purposes, also among physically active individuals, often in high doses. The remainder of this paper presents the consequences of such use of T and other AAS with respect to various aspects of the structure and function of the cardiovascular system, as graphically summarised in **Figure 2**.

AAS and blood pressure

As a result of physiological adaptations to physical activity, athletes across various disciplines often exhibit naturally elevated T levels. It is believed that regular physical exercise increases the pulsatile secretion of LH and T, raises AR sensitivity, and reduces SHBG levels, thereby increasing the amount of active, free T in serum [25]. Although such an increase is not harmful under

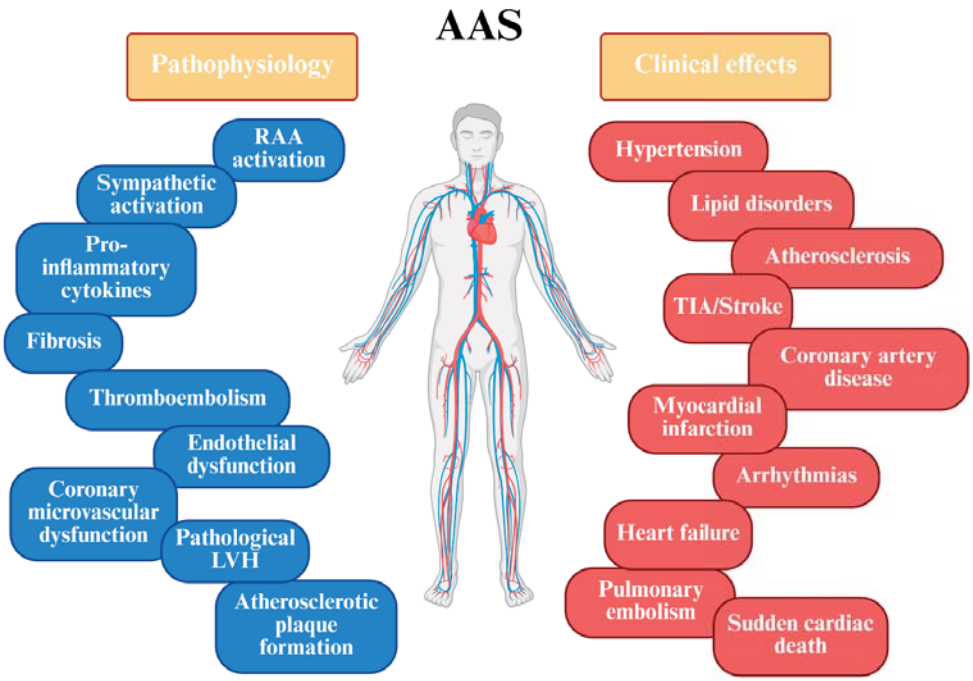

Figure 2. Summary – pathophysiology and clinical effects of AAS on cardiovascular system. Abbreviations: AAS – anabolic-androgenic steroids; RAA – renin-angiotensin-aldosterone system; LVH – left ventricular hypertrophy; TIA – transient ischemic attack.

physiological conditions, chronic overproduction or exogenous T supplementation can dysregulate the systems that control blood pressure.

T stimulates the release of neuropeptide Y and intensifies the action of endothelin-1, thereby causing vasoconstriction and increasing vascular resistance [26]. Animal studies have shown increased expression of the thromboxane A2 receptor and decreased production of 20-hydroxyeicosatetraenoic acid (20-HETE), which is responsible for sensitising smooth muscle cells to vasoconstrictive factors. However, these effects depend largely on the initial state of vascular endothelial cells, whose functions may already be impaired, e.g., by atherosclerotic processes [26]. T affects the activity of the autonomic nervous system, specifically its sympathetic part. It increases the activity of tyrosine hydroxylase, which promotes norepinephrine synthesis, leading to subsequent tachycardia and vasoconstriction and thus contributing to an increase in blood pressure [26]. It also modulates the stress response by influencing the hypothalamic-pituitary-adrenal axis. It inhibits cortisol secretion while enhancing the sensitivity of adrenergic receptors. In such a case, there is a slower vascular response to changes in blood pressure, which, in turn, promotes further increases in blood pressure [27,28]. In particular, it affects the renin-angiotensin-aldosterone axis by increasing the synthesis of angiotensin II, a potent vasoconstrictor, and increasing the secretion of aldosterone, which promotes sodium and water retention. This may be a risk factor for the development of volume-dependent hypertension in predisposed individuals, e.g., those with renal hypertension. In athletes, this can exert additional stress on the kidneys, especially when combined with a high-protein diet [29].

Grace et al. [30] demonstrated that high-dose AAS use leads to a temporary but significant increase in diastolic blood pressure and the rate-pressure product (systolic blood pressure × heart rate). These levels returned to normal within a few weeks after the end of the AAS cycle; however, the long-term effects are unknown. In another study [31], the blood pressure response to physical activity was compared among individuals taking AAS, bodybuilders not using AAS, and subjects leading a sedentary lifestyle. AAS users had higher blood pressure levels, both at rest and during exercise, which may also constitute a factor that increases cardiovascular risk. Furthermore, it was found that resting systolic

blood pressure was higher in active AAS users (mean 140 ± 10 mm Hg) compared to former users (130 ± 5 mm Hg; $p < 0.05$) and athletes who did not use AAS (125 ± 10 mm Hg; $p < 0.001$) [23]. Other researchers observed that in individuals who had stopped using AAS around five months earlier, elevated systolic blood pressure persisted. They found a small but significant increase in systolic blood pressure of 3 mm Hg and an acceleration of heart rate by seven beats per minute in individuals after a several-week steroid cycle [32]. As in other forms of hypertension, a further consequence of elevated blood pressure induced by T use is an increase in overall cardiovascular risk, the development of pathological myocardial hypertrophy, and an increased risk of arrhythmia, coronary artery disease, or heart failure.

AAS and structural changes in the heart and blood vessels

Pathological myocardial hypertrophy and fibrosis are structural changes that occur in response to chronic overload, ischaemia, hormonal stress (mainly via vasoactive hormones), and inflammatory stress, as well as other factors, such as exogenous substances (e.g., AAS). Both hypertrophy and fibrosis of the heart muscle may precipitate the development of heart failure. Excessive AR stimulation by T can result in pathological myocardial hypertrophy [31,33]. The hormone activates signalling pathways, such as mTOR/S6K1, responsible for protein synthesis and cardiomyocyte growth [34]. This effect is exacerbated by oxidative stress and changes in calcium metabolism that promote cell growth and, consequently, cardiac remodelling [35]. Myocardial fibrosis, conversely, results from excessive fibroblast activation and collagen deposition in the extracellular matrix. T modulates the expression of factors such as TGF-β and angiotensin II, which are essential for fibrosis development [36]. Studies have shown that T has a permissive effect, leading to activation of the TGF-β/Smad and Gas6/Axl pathways, resulting in increased extracellular matrix synthesis and impaired cardiac diastolic function [37]. Another study found that supraphysiological doses of T administered to rats caused myocardial hypertrophy via an additional signalling pathway, PI3K/Akt/mTOR [38]. For many years, studies including strength athletes have shown that active or former AAS users have greater left ventricular muscle mass (adjusted for lean body mass) and left ventricular wall thickness [39, 40–41].

Signs of concentric cardiac hypertrophy in these individuals persisted for many years after discontinuing AAS, and adverse changes were observed in echocardiographic parameters of left ventricular diastolic function compared with athletes who had never used AAS [39]. Other authors assessed how chronic use of AAS (lasting at least 2 years, with participants in the "on-cycle" phase) affected heart structure and function and the presence of focal fibrosis. The study, involving a group of 21 strength athletes, showed that long-term use of supraphysiological doses of steroids led not only to left ventricular wall hypertrophy, but also to impaired left and right ventricular function parameters. An interesting finding that warrants further research is the absence of focal fibrosis on LGE-CMR (late gadolinium enhancement) imaging, despite significant myocardial dysfunction [40]. It has not yet been clearly specified which AAS dose or duration of use may cause fibrosis foci to be observable on CMR imaging. One of the case reports in the literature describes a man with symptoms of increasing shortness of breath and general fatigue who abused AAS for over 20 years. T1-weighted CMR images revealed focal contrast enhancement corresponding to fibrosis. Importantly, these changes occurred despite normal coronary artery anatomy, which excluded ischemia as a potential cause. This indicates a direct, potentially toxic effect of AAS on myocardial structure, ultimately resulting in fibrosis [42].

Additionally, Gregersen et al. demonstrated that active AAS users had significantly elevated levels of interleukin-8 (IL-8) and matrix metalloproteinase-9 (MMP-9) compared with both former users and individuals who had never used these substances [43]. Furthermore, MMP-9 correlated with sex hormone levels, suggesting that its increase may be partially mediated by endocrine disturbances induced by AAS. Higher concentrations of IL-8 and MMP-9 were associated with parameters indicating myocardial damage, and MMP-9 was additionally linked to increased cardiac muscle mass. These associations were not found among former users, indicating that

inflammatory changes and myocardial remodelling processes are enhanced during active AAS use and may be partially reversed after discontinuation. As regards changes in vessel walls, in men using high doses of AAS, increased arterial stiffness and elevated systolic and diastolic blood pressure were observed, which are further indications of adverse remodelling of the cardiovascular system [32].

AAS and lipid disorders

Supraphysiological doses of T lead to significant lipid profile disorders, which are one of the key risk factors for the development of atherosclerosis and, consequently, coronary artery disease. High concentrations of T increase the activity of hepatic lipoprotein lipase, an enzyme responsible for the catabolism of high-density lipoprotein (HDL) molecules, resulting in a significant reduction in their levels [44]. Additionally, androgens increase the expression of the low-density lipoprotein (LDL) receptor in the liver, leading to higher concentrations of LDL and very-low-density lipoproteins (VLDL) in plasma [45]. Paradoxically, AAS may reduce the concentration of lipoprotein (a). However, this effect is largely clinically neutralised by the profound changes in HDL and LDL levels. Finally, the overall effect of AAS remains highly atherogenic [45].

These changes also have an inflammatory component: supra-physiological T levels increase pro-inflammatory cytokines, i.e., tumour necrosis factor-alpha (TNF-α) and IL-6. Moreover, it exacerbates oxidative stress, which further damages the endothelium and contributes to vascular dysfunction [46]. Clinical studies show that after just a few weeks of AAS use, HDL levels can decrease by more than 50% and LDL levels can increase by up to 40% compared to baseline [47]. This effect may persist for a long time, depending on the cumulative dose of AAS taken. This process is presented in a study that analysed several metabolic and cardiological parameters in the discipline of weightlifting among men aged 35–54 who were active or former AAS users (cumulative use of AAS for at least two years) compared to athletes not taking AAS [48]. Those using AAS were significantly more likely to have lipid disorders (defined as LDL cholesterol concentration > 160 mg/dl): 23% vs. 13%. Considering the criteria adopted in relation to those currently in force, a significantly higher incidence of these disorders should be expected. Furthermore, in another study conducted on a small group of Australian bodybuilders using AAS, significantly lower HDL cholesterol concentrations were observed compared to the control group not using AAS (0.6 ± 0.1 vs. 1.4 ± 0.1 mmol/L; $p < 0.001$) [38]. The available data indicate that changes in the lipid profile may be partially irreversible, depending on the dose and duration of AAS exposure. In some cases, HDL and LDL levels normalize within 6 to 12 months following discontinuation of T use, however, the consequences of these disorders may persist longer [47–49]. Based on AAS-induced adverse changes in cardiac and vascular remodelling, lipid profile disorders, and increased blood pressure, further complications associated with myocardial ischemia may develop, including myocardial infarction, cerebral vascular insufficiency, arrhythmias, heart failure, and sudden cardiac death.

AAS and ischemic heart disease

Over the past 30 years, the literature has documented an increasing number of reports regarding myocardial infarction of various aetiolo-

Table 1. Testosterone stimulates erythropoiesis through several interrelated mechanisms.

Mechanism of testosterone's action	Description of biological effect
Increased production of erythropoietin	Stimulates the glomerular endothelial cells to increase the synthesis of erythropoietin, the main hormone regulating erythropoiesis [56,57]
Effect on progenitor cells	Directly stimulates the proliferation and differentiation of erythroid cells in the bone marrow [56,57]
Inhibition of hepcidin	Decreases the level of hepcidin, a peptide regulating iron metabolism – it results in increased bioavailability of iron required for haemoglobin synthesis [56,57]
Increased iron absorption from the gastrointestinal tract	Increased iron absorption from the gastrointestinal tract – secondary effect to the reduction of hepcidin and the increased iron demand by the bone marrow [56,57]

gies in individuals using AAS. One of the earliest documented cases involved a previously healthy 37-year-old bodybuilder who suffered an acute myocardial infarction, probably because of taking T for about two years. Coronary angiography performed in the athlete revealed signs of coronary artery thrombosis without the presence of classic atherosclerotic plaques [50]. This finding suggests a direct effect of supra-physiological T doses on the activation of prothrombotic mechanisms and destabilisation of the vascular endothelium. Another case concerned a 27-year-old bodybuilder, with no other risk factors, who had used AAS for ten years and eventually suffered occlusion of the proximal segment of the left anterior descending artery and extensive myocardial infarction [51]. Consequently, researchers have emphasised the necessity of considering AAS when investigating the aetiology of coronary incidents in young athletes, listing several potential pathomechanisms of these substances, including platelet hyperreactivity, dyslipidemia, and vascular reactivity. Subsequent reports have reiterated that extensive thromboembolic changes in the coronary arteries are the cause of heart attacks in athletes who had used AAS for many years [52,53].

The use of exogenous T, both for therapeutic and non-medical purposes, causes numerous changes in the haematopoietic system, most notably secondary polycythaemia. This condition is associated with increases in erythrocyte count, haemoglobin, and haematocrit, leading to increased blood viscosity. High haematocrit levels (above 52–54%) significantly increase the risk of TIA, stroke, myocardial ischaemia, and venous thrombosis [54,55]. The mechanisms of T's stimulating effect on erythropoiesis are presented in **Table 1**.

However, it was observed that ischaemic complications can also occur with relatively short-term AAS exposure. An example is a case of a 30-year-old male, with no medical history, taking AAS (stanozolol and T) for a period of two months, who was admitted to the hospital due to an acute myocardial infarction. Angiography revealed signs of thrombosis of the left anterior descending artery, without atherosclerotic changes in the coronary arteries. Due to his history of smoking, the authors suggested that the effects of AAS could be one of several potential causes of acute cardiac ischemia [53]. Another mechanism underlying myocardial infarction is coronary artery spasm. AAS weaken the vascular response to vasodilators (nitric oxide, NO) through cGMP inhibition and direct endothelial damage [53]. While coronary artery atherosclerosis remains a leading cause of myocardial ischemia in AAS users, it is more likely to be associated with long-term use of these substances and results from the cumulative vascular disorders they cause. In computed tomography angiography (CTA) studies of the coronary arteries conducted among men aged 34–54 who were previously amateur or professional weightlifters, a higher total AAS dose was associated with a larger volume of atherosclerotic plaques compared with the non-user group.

Additionally, some of the subjects in the AAS-using group had a history of coronary events that occurred within a few to several years of using AAS [48]. Buhl et al. assessed the incidence of atherosclerotic changes in the arteries and myocardial function in recreational athletes, 80 active AAS users (including 24% women), 26 previous users (including 31% women), and 58 non-users [58]. The study group included active and former AAS users, with the mean age of 35 and 36 years, respectively, training strength or endurance with high intensity for an average of about ten hours per week, and non-AAS users, with the mean age of 40 years, who did similar training for five hours a week. No differences were observed between the groups in terms of the incidence of atherosclerotic plaques in the carotid and femoral arteries or calcium scores (CAC scores) in the subgroups. However, the non-users were older than the active AAS users. However, it was shown that active AAS users had significantly more non-calcified plaques in the coronary arteries than non-users (24% vs 10% of study participants). The risk of both non-calcified plaques in the coronary arteries and a positive CAC score increased with longer AAS use, with a duration exceeding 5 years being a critical factor.

AAS and heart failure

Heart failure may be precipitated by ischemia, pressure or volume overload, fibrosis resulting in diastolic dysfunction, as well as thromboembolic or inflammatory mechanisms. One of the earlier studies on heart damage in strength athletes included participants who had used AAS for more

than five years (on average, 31 weeks per year). The study revealed that apart from elevated systolic blood pressure and myocardial hypertrophy, the subjects had developed echocardiographic features of diastolic dysfunction and impaired left ventricular systolic function [59]. Subsequent studies confirmed these observations and the diagnostic utility of selected echocardiographic indicators, and the new techniques enabled the detection of left atrial muscle dysfunction in AAS users [58,60,61]. Additionally, the observed abnormalities were associated with reduced exercise capacity in these athletes, as assessed by peak oxygen consumption (peak VO_2) during cardiopulmonary exercise testing [59,61].

The primary parameter for assessing cardiac systolic function is the left ventricular ejection fraction (LVEF). The literature on LVEF values in AAS users varies in the findings reported. Some authors report no significant differences in this parameter between AAS users and non-users [60–63], whereas others report reduced LVEF values in AAS users [48,58,63–65]. For example, a study by Baggish et al. [48] showed significant differences in 140 experienced male weightlifters, aged 34 to 54 years. In this group, 86 athletes reported at least 2 years of combined AAS use. Compared with non-users, they exhibited lower LVEF values, i.e., 52 ± 11% vs 63 ± 8% ($p < 0.001$). Moreover, active AAS users demonstrated even lower LVEF values, with a mean of 49 ± 10%.

Additionally, active or former AAS users had worse parameters of left ventricular diastolic function and greater atherosclerotic plaque volume in CTA assessments. The type and extent of heart damage, as well as the reversibility of these changes after discontinuation of AAS, remain a subject of debate. Other authors studied men aged 18–50 who engaged in recreational resistance training. They were divided into subgroups: active AAS users, former AAS users (on average, 30 months after discontinuing AAS), and non-users [63]. Reduced global longitudinal strain (GLS) values were observed in former users compared to non-users, and in active users compared to non-users. In addition to abnormal GLS, lower LVEF values were observed. No differences were found between the groups in the presence of fibrosis features on CMR.

The authors of the study emphasised the fact that abnormalities in left ventricular systolic function persisted for many months after discontinuation of AAS. In this context, other researchers assessed left and right ventricular function at rest and during exercise (exercise stress echocardiography) [62]. Among the 115 bodybuilders (athletes at the highest level of sport, with no history of cardiovascular disease or diabetes) included in the study, 65 participants had taken AAS for at least 5 years. The subjects' training volume averaged 15–20 hours per week for at least five years. The study participants took AAS such as methenolone, nandrolone, and T esters (intramuscularly), and fluoxymesterone, mesterolone, methenolone, methandienone, oxandrolone, and oxymetholone (orally). In the vast majority of cases, combinations of oral and intramuscular forms were used. Division into the groups of AAS users and non-users was additionally determined based on tests of serum FSH and LH levels. The mean number of weeks of AAS use per year was 33 ± 6.4, and the weekly dose was 528 ± 91 mg. As in previous reports, AAS users showed left ventricular wall hypertrophy, increased LV mass index, and worse systolic and diastolic LV function parameters, with no differences in LVEF. Moreover, this group showed increased right ventricular (RV) dimensions and impaired free wall systolic function. During exercise, AAS users demonstrated significantly lower LV GLS and RV lateral strain values compared to non-users and the control group. They also had poorer physical performance and showed signs of pulmonary congestion during the exercise test. The authors of the study emphasised that the duration of AAS use (calculated in weeks per year) was an independent factor in the occurrence of myocardial damage already observed at rest, which clearly translated into alarming changes during exercise. When presenting the analyses, it is worth noting that with short-term use of AAS, the LVEF parameter may be insufficient to detect abnormalities, in which case speckle-tracking echocardiography and GLS assessment should be performed [64]. Myocardial damage resulting from the use of AAS can lead to more advanced cardiac changes. In a large study of 101 weightlifters with a cumulative long duration of AAS use (11 ± 7 years), significantly reduced LVEF values were observed compared to non-users (49 ± 7 vs. 59 ± 5%; $p < 0.001$). Moreover, 11% of active and former AAS users had LVEF lower than or equal to 40%, which is consistent with a clinical diagnosis

of heart failure [65]. Among active and former AAS users, 36% had LVEF ranging between 41 and 49%, with normal values observed in only slightly more than half of the group. This percentage was identical regardless of whether they had used AAS at the time of the study or previously, indicating the development of permanent heart damage. AAS use was associated with RV systolic dysfunction and other multidirectional changes in the myocardium. In the regression analysis for the whole population, the strongest determinant of reduced LVEF was a history of AAS use by athletes. It was proven that the use of these substances can lead to severe heart failure in all its forms, i.e., with preserved, mildly reduced or reduced LVEF.

AAS and sudden cardiac death

The association between AAS and sudden cardiac death has been given strong clinical emphasis due to the escalating prevalence of AAS abuse [22,41,66–70]. Case reports consistently illustrate severe, often fatal, cardiac pathology in AAS users, even in young athletes. Post-mortem analyses frequently reveal extreme cardiac hypertrophy, substantially exceeding physiological norms, e.g., with an average heart weight of 580 g compared to a normal male range of 300–350 g, and severe left ventricular wall thickening (up to 16 mm vs normal ≤ 11 mm) [67]. Post-mortem examinations performed in AAS users also show systemic damage, including microinfarcts in organs such as the liver and kidneys. In some cases, especially in women, findings may be less clear but still indicate a link to AAS [69]. The specific substance, dosage, and duration of use determine the precise effects. Recently published research results by Vecchiato M et al., based on long-term observation of large groups of athletes, clearly indicate a significant problem of premature cardiac deaths associated with the use of AAS in both sexes, not only in men [22,70].

Summary

The nonmedical, uncontrolled use of AAS to improve muscle strength, physical performance, or body appearance is associated with various complications affecting multiple organs and systems. As regards the cardiovascular system, multidirectional changes occur, clinically manifested as the development of hypertension, dyslipidemia, myocardial infarction, arrhythmia, heart failure, or sudden cardiac death. While professional athletes are subject to regular anti-doping controls and risk disqualification if they are found to use AAS, the uncontrolled use of AAS has become an increasingly common problem among a much wider population of amateur and recreational sportspeople, for whom the primary motivation is aesthetic improvement rather than competitive achievement. Considering numerous health complications, including those most dangerous ones affecting the cardiovascular system, it is necessary to both educate and take action to counteract the spread of AAS use.

Declarations and statements

Author contributions
R.B. – conception, drafting the article, preparation of tables and figures, final approval; J.S.-H., A.J., T.Z., D.D., I.S. – conception, drafting the article, final approval.

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