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Do Anabolic Androgenic Steroids Really Cause Harm? A Comprehensive Examination of Adverse Effects

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Abstract:
Anabolic steroids, synthetic derivatives of testosterone, are frequently employed in clinical settings to address hormonal deficiencies and, controversially, in athletic contexts to augment physical performance. These compounds exert their physiological effects by activating androgen receptors, thereby stimulating protein synthesis, promoting muscle hypertrophy, and accelerating tissue regeneration. These processes are mediated through the modulation of insulin-like growth factor-1 (IGF-1) and the inhibition of glucocorticoid-induced catabolism. However, the unsupervised and excessive use of anabolic steroids, often motivated by the pursuit of rapid aesthetic or performance enhancement, is associated with a myriad of adverse health outcomes. Such complications encompass damage to the reproductive system, renal dysfunction, hepatotoxicity, cardiovascular pathology, and neuropsychiatric disturbances. Given their substantial potential for misuse, anabolic steroids are stringently regulated, necessitating that healthcare professionals remain astutely aware of their adverse effects to ensure timely recognition and intervention.

Objective:
This review endeavors to furnish healthcare practitioners with a comprehensive, evidence-based overview of the deleterious effects associated with anabolic steroids. It elucidates the underlying pathophysiological mechanisms and explores therapeutic and diagnostic modalities aimed at mitigating harm.
Materials and Methods:
The analysis synthesizes data from peer-reviewed articles retrieved from PubMed and Google Scholar, encompassing research spanning a 13-year timeframe. Selection criteria prioritized studies of high relevance and robust scientific rigor, focusing on the clinical ramifications of anabolic steroid usage and its associated complications.
Conclusion:
Anabolic-androgenic steroids (AAS) significantly impact cardiovascular, hepatic, renal, reproductive, and neuropsychiatric health. They disrupt lipid metabolism, accelerate atherosclerosis, and cause irreversible vascular damage, increasing the risk of cardiovascular events. Hepatotoxic effects include oxidative stress, chronic inflammation, and heightened liver tumor risk, while renal damage involves glomerular dysfunction and progression to chronic kidney disease. AAS misuse impairs reproductive function by disrupting the hypothalamic-pituitary-gonadal axis and altering testicular cell epigenetics. Neuropsychiatric effects encompass structural and functional CNS changes, leading to mood instability, cognitive deficits, and aggression. Although some effects may be reversible upon cessation, long-term damage, including fibrosis, neuronal injury, and addiction, remains challenging to address. Enhanced clinical awareness, diagnostic strategies, and targeted interventions are essential to mitigate AAS-related harm, alongside further research to elucidate underlying mechanisms and develop effective therapies.

Keywords: anabolic steroids, adverse effects, cardiovascular pathology, hepatotoxicity, renal dysfunction, reproductive system, neuropsychiatric disorders.

Introduction

Anabolic-androgenic steroids (AAS) are synthetic derivatives of testosterone commonly utilized in the management of conditions related to testosterone deficiency. While their therapeutic applications are well-established, these substances are also widely misused in athletic and bodybuilding contexts to enhance physical performance [1]. Estimates suggest that between 1% and 5% of the global population has engaged in AAS use, with a higher prevalence observed among men [6,9]. Anabolic steroids can be broadly categorized into two classes: 17-alpha-alkyl derivatives (e.g., oxandrolone, oxymetholone) and 17-beta-ester derivatives (e.g., testosterone cypionate, testosterone enanthate) [1]. Frequently used compounds include nandrolone, stanozolol (STZ), oxandrolone, methandrostenolone, and trenbolone, which are administered either orally or via intramuscular injection [25].

All anabolic steroids are classified as Schedule III substances under the Controlled Substances Act (DEA) due to their potential for misuse and associated risks. These compounds exert their effects primarily through androgen receptor activation, leading to increased receptor density and enhanced nitrogen retention in muscle tissues.

These mechanisms drive elevated protein synthesis, resulting in significant gains in muscle mass and strength. Additionally, AAS influence the insulin-like growth factor-1 (IGF-1) axis, promoting muscle growth and tissue regeneration. Beyond their physical effects, anabolic steroids impact the central nervous system, modulating mood and aggression, and suppress the catabolic effects of glucocorticoids. Nandrolone derivatives (e.g., nandrolone decanoate) are notable for their high anabolic-to-androgenic activity ratio, offering advantages in muscle mass enhancement and joint pain alleviation [1].

However, the misuse of anabolic steroids is associated with significant adverse health outcomes. These range from cardiovascular and hepatic complications to reproductive dysfunction and neuropsychiatric disorders. Compounding the issue, many patients fail to disclose their use of these substances, complicating diagnosis and management. This underscores the importance of equipping healthcare professionals with the knowledge and skills necessary to identify and address the potential consequences of AAS misuse [2].

Impact of Anabolic Androgenic Steroids on Cardiovascular Health

The use of anabolic-androgenic steroids (AAS) significantly disrupts lipid metabolism, accelerating the progression of atherosclerotic plaques and arterial calcification via androgen receptor-mediated mechanisms that impair vascular elasticity. AAS decrease high-density lipoprotein (HDL) cholesterol levels while increasing low-density lipoprotein (LDL) cholesterol levels, contributing to an atherogenic lipid profile. Furthermore, elevated homocysteine levels, commonly observed with AAS misuse, exacerbate the risk of coronary artery disease [3].

The method of AAS administration plays a critical role in determining toxicity levels. Oral AAS, such as oxymetholone, are associated with a higher risk of hepatotoxicity and secondary cardiovascular complications due to their extensive hepatic metabolism. This metabolic process amplifies adverse lipid alterations, resulting in significant reductions in HDL and marked elevations in LDL levels [15,13]. In contrast, injectable AAS (e.g., testosterone cypionate or enanthate) exert comparatively milder effects on lipid profiles [15,13].

In younger adults (aged 18–30 years), the cardiovascular side effects of AAS may initially be masked by robust cardiac reserves. However, prolonged use in this demographic precipitates early endothelial dysfunction, heightening the risk of hypertension and arrhythmias [18,13]. Conversely, individuals over 40 years of age, especially those with pre-existing cardiovascular conditions, are at considerably greater risk of complications such as left ventricular hypertrophy, myocardial fibrosis, arrhythmias, and heart failure. Age-related vascular stiffness compounds the adverse effects of AAS on hemodynamics and circulation, significantly increasing the likelihood of severe health outcomes [13,3,2].

AAS use also contributes to hypertension through mechanisms such as sodium retention in the kidneys and vascular structural remodeling, although studies on these pathways have yielded inconsistent results [3,2,4]. Moreover, AAS-induced prothrombotic states, characterized by enhanced platelet aggregation, increase the risk of venous thromboembolism and pulmonary embolism [2,4]. Chronic vascular damage, including fibrosis and calcification, is often irreversible, even after the cessation of AAS use [4,2].

Impact of Anabolic Androgenic Steroids on the Liver

Anabolic-androgenic steroids (AAS) are chemically engineered to enhance anabolic activity, extend their duration of action, and reduce hepatic metabolism. However, these modifications significantly increase the risk of liver damage, particularly with oral AAS formulations, which demonstrate higher hepatotoxicity compared to their injectable counterparts [8,9].

The hepatotoxic effects of AAS are closely linked to their hepatic metabolism, where the accumulation of toxic metabolites damages liver cells. AAS induces excessive production of reactive oxygen species (ROS), leading to mitochondrial dysfunction and disruption of hepatocyte membranes. This cascade of events results in cell necrosis and impaired hepatic function [9]. Furthermore, activation of Kupffer cells within the liver and subsequent release of pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin-1 beta (IL-1β), promote fibrosis and chronic hepatic damage. Long-term exposure to AAS also stimulates hepatocyte proliferation, significantly increasing the risk of developing hepatic tumors [10,7,9].

The misuse of AAS has been associated with the formation of both benign hepatic adenomas and malignant tumors, including hepatocellular carcinoma [8,9]. Additionally, AAS disrupts normal bile flow within the liver, leading to cholestasis - a condition characterized by jaundice, pruritus, and elevated serum bilirubin levels. While cholestasis may resolve upon discontinuation of AAS use, severe cases often require medical intervention to prevent further complications [10,7,9].

Chronic use of AAS can lead to the development of peliosis hepatis, a rare but potentially fatal condition characterized by blood-filled cysts in the liver. This condition poses a significant risk of internal hemorrhage. Elevations in hepatic enzyme levels, including aspartate aminotransferase (AST), alanine aminotransferase (ALT), and lactate dehydrogenase (LDH), are frequently observed in AAS users. However, these increases may not always indicate liver damage, particularly in athletes engaged in intense physical training where muscle injury may contribute to enzyme elevation [8,9].

The prevalence and severity of AAS-induced hepatotoxicity are likely underestimated due to underreporting and insufficient surveillance, highlighting the need for heightened clinical awareness and robust diagnostic strategies [6,9].

Impact of Anabolic Androgenic Steroids on the Kidneys

Anabolic-androgenic steroids (AAS) negatively affect kidney health by triggering multiple mechanisms that contribute to renal damage. One primary process involves the induction of reactive oxygen species (ROS) production and the increased expression of inflammatory mediators such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6). These mechanisms result in kidney damage through oxidative stress and inflammation [12,11]. Additionally, AAS stimulate endothelin activity and the renin-angiotensin system (RAS), leading to glomerular hypertension and proteinuria, which accelerate the progression of chronic kidney disease (CKD) [12,11].

To mitigate AAS-induced kidney damage, new biomarkers, such as urinary monocyte chemoattractant protein-1 (MCP-1) and neutrophil gelatinase-associated lipocalin (NGAL), are under investigation. These markers may facilitate the early detection of renal abnormalities [11].

Chronic use of AAS can result in secondary focal segmental glomerulosclerosis (FSGS), particularly among athletes who combine AAS use with intensive training and high-protein diets [19]. Kidney diseases related to AAS misuse represent a growing public health concern, as the use of these substances continues to rise among athletes and bodybuilders [6,11].

A study by Herlitz et al. involving 10 bodybuilders identified severe proteinuria, hypertension, and kidney damage, including glomerulomegaly and podocyte loss. The mechanisms of damage included glomerular hyperfiltration and the direct nephrotoxic effects of steroids on kidney cells. Discontinuing AAS and reducing protein intake improved kidney function in most patients. However, reinitiating steroid use resulted in a recurrence of renal damage [19].

Similar findings were reported in a study by El-Reshaid et al., which examined 22 AAS users. The study documented cases of chronic interstitial nephritis and nephrocalcinosis, with the severity of these conditions correlating with the duration of AAS use [11]. These findings underscore the importance of monitoring renal health in AAS users and highlight the critical need for early cessation of AAS use to prevent irreversible kidney damage [6,11].

Impact of Anabolic Androgenic Steroids on the Reproductive System

Chronic use of anabolic-androgenic steroids (AAS) significantly impacts the male reproductive system, with profound clinical and molecular consequences. The primary mechanism of AAS-induced dysfunction is the suppression of the hypothalamic-pituitary-gonadal (HPG) axis, which leads to reduced secretion of gonadotropin-releasing hormone (GnRH). This suppression causes a downstream decrease in luteinizing hormone (LH) and follicle-stimulating hormone (FSH) levels, resulting in the inhibition of endogenous testosterone production and disruption of spermatogenesis [18,13].

Clinically, secondary hypogonadism associated with AAS misuse manifests as decreased libido, erectile dysfunction, and infertility [14,15]. AAS disrupts normal spermatogenesis, leading to a significant reduction in sperm count, motility, and morphological quality. Histopathological studies further confirm testicular atrophy and abnormalities in the structure of the seminiferous tubules [18,13].

At the molecular level, AAS interfere with steroidogenesis and cholesterol metabolism by inhibiting key enzymes such as CYP11A1 and CYP17A1, which are essential for testosterone synthesis [17,13]. Additionally, AAS induces oxidative stress, resulting in mitochondrial damage and apoptosis of testicular cells [17,18]. Emerging research highlights the potential for AAS to cause epigenetic modifications, including DNA methylation and histone structural changes, which may disrupt gene expression necessary for normal spermatogenesis [17,15].

Although some hormonal and spermatogenic recovery may occur after discontinuation of AAS, long-term damage, such as fibrosis and persistent hypogonadism, may remain [14,18]. Of particular concern is the use of AAS in combination with substances such as human chorionic gonadotropin (hCG) or aromatase inhibitors, which can exacerbate reproductive dysfunction and compound broader health risks [13].

Impact of AAS on Neuropsychiatric Disorders

The neuropsychiatric effects of anabolic-androgenic steroids (AAS) are multifaceted, arising from several interconnected mechanisms. AAS exert their influence by binding to androgen receptors within the central nervous system (CNS), leading to structural and functional changes in brain regions responsible for regulating mood, cognition, and behavior. High-dose AAS exposure disrupts key neurotransmitter systems, particularly the serotonergic and dopaminergic pathways, which play critical roles in mood stabilization and reward processing [23,24]. Chronic use also induces oxidative stress and mitochondrial dysfunction, contributing to neuronal apoptosis and cognitive deficits [21,6]. Additionally, AAS inhibits the hypothalamic-pituitary-gonadal (HPG) axis, resulting in secondary hormonal dysregulation that exacerbates psychiatric symptoms, particularly during withdrawal periods [21,6].

One of the most recognized neuropsychiatric consequences of AAS misuse is heightened aggression, often referred to as "roid rage." Self-reported aggression increases significantly among AAS users, particularly with prolonged use and higher doses. A meta-analysis by Chegni et al. demonstrated a statistically significant increase in aggression among users, with similar patterns observed in animal studies [23,24]. These behavioral changes are attributed to AAS-induced alterations in the amygdala, which governs emotional responses, and the prefrontal cortex, responsible for impulse control [21].

AAS misuse is strongly associated with a range of mood disorders, including depression, anxiety, and mania. Chronic AAS use suppresses endogenous testosterone production through inhibition of the HPG axis, causing emotional instability. Depressive symptoms often worsen during withdrawal, with many individuals experiencing severe anhedonia and suicidal ideation [22,20]. Anxiety disorders are also prevalent, likely due to AAS-induced serotonin dysregulation [20]. High doses of AAS have been linked to manic episodes characterized by euphoria, hyperactivity, and impulsivity [21,20].

Neuroimaging studies highlight the long-term consequences of AAS misuse, including cortical thinning, amygdala enlargement, and reduced gray matter volume. These structural brain changes correlate with cognitive impairments such as memory deficits, diminished decision-making capacity, and weakened executive functions [21,22]. The prefrontal cortex, a key region for behavioral regulation and decision-making, appears particularly vulnerable to AAS-induced damage [21].

Acute psychotic episodes, characterized by paranoia, hallucinations, and delusions, have been reported in AAS users. Such episodes are more common in individuals with a predisposition to mental illness or those using other substances concurrently [20,6]. Severe psychotic symptoms often necessitate psychiatric intervention and can persist even after AAS discontinuation.

The potential for addiction is another significant concern associated with AAS misuse. Addiction is marked by compulsive use, cravings, and withdrawal symptoms, driven by neuroadaptive changes in dopaminergic pathways that reinforce reward-seeking behaviors [21,22]. Withdrawal symptoms, including fatigue, emotional instability, and sleep disturbances, further complicate cessation efforts [21,6].

The neuropsychiatric impact of AAS differs across age groups. Adolescents and young adults are particularly susceptible to aggression and emotional instability due to ongoing brain development [24,20]. In contrast, older individuals with a history of chronic AAS misuse are more likely to experience persistent depressive symptoms and cognitive decline, potentially linked to long-term neuronal damage [22].

Conclusion:

Anabolic-androgenic steroids (AAS) profoundly impact multiple organ systems, with significant implications for cardiovascular, hepatic, renal, reproductive, and neuropsychiatric health. AAS disrupt lipid metabolism, accelerate atherosclerosis, and contribute to irreversible vascular damage, heightening the risk of severe cardiovascular events. Hepatotoxic effects include oxidative stress, chronic inflammation, and increased risk of liver tumors, while renal damage is marked by glomerular dysfunction and chronic kidney disease progression. AAS misuse severely impairs reproductive function by disrupting the hypothalamic-pituitary-gonadal axis and inducing molecular and epigenetic changes in testicular cells. Furthermore, AAS use leads to structural and functional alterations in the central nervous system, resulting in mood instability, cognitive deficits, and increased aggression.

Despite potential reversibility in some cases upon cessation, many of the long-term effects of AAS misuse, such as fibrosis, neuronal damage, and addiction, remain challenging to address. These findings underscore the critical need for enhanced clinical awareness, robust diagnostic strategies, and targeted interventions to mitigate the adverse health effects associated with AAS misuse. Further research is essential to better understand the mechanisms underlying these outcomes and to develop effective preventive and therapeutic approaches.

Disclosure

Author's contribution:

Conceptualization: PS, AS, MŁ
Methodology: KP, WT
Software: WP, WM
Check: KP, AN
Formal analysis: JK, AN
Investigation: MŁ, PD
Resources: MG, AN
Data curation: WM, JK
Writing rough preparation: JK, MŁ
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