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Hepatotoxicity Associated with Anabolic Androgenic Steroids and Selective Androgen Receptor Modulators: A Literature Review

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ABSTRACT

Nowadays, the misuse of anabolic androgenic steroids (AAS) and selective androgen receptor modulators (SARMs) is becoming a growing concern. People are using AASs and SARMs to enhance physical performance and muscle mass. Testosterone derivatives are particularly prevalent among bodybuilders and young male athletes. These substances are easy to buy, and some have been even found in over-the-counter dietary supplements. The wide availability has led to off-label use of AASs and SARMs. The purpose of this review is to provide an overview of the most recent studies on the hepatotoxic effects of AASs and SARMs including direct hepatocellular damage, acute cholestatic syndrome, peliosis hepatis, and development of benign and malignant liver tumors, predominantly hepatic adenomas and HCC.

Keywords: Selective Androgen Receptor Modulators, Androgenic-Anabolic Steroids, Hepatotoxicity, Drug-Induced Liver Injury, Hepatocellular carcinoma

1. INTRODUCTION

In recent years, there has been an emerging concern about the search for drugs designed to enhance physical performance and muscle tone. Derivatives of testosterone have been detected in certain over-the-counter nutritional supplements and online products, often promoted as solutions for improving well-being, building muscle mass, or aiding bodybuilding. Particularly, it has been reported that 33% of adults use dietary supplements daily, with 12.5% of these samples being contaminated with anabolic steroids (Hildebrandt et al., 2011). The misuse of these substances remains particularly common among bodybuilders and young male athletes (El Sherrif et al., 2013a).

Testosterone, the key male sex hormone, is predominantly produced in the testes in men, with a smaller amount secreted by the adrenal glands in both men

and women. Since testosterone is only effective when delivered via intramuscular injections, sublingual administration, or transdermal patches, modifications were made to enhance its properties. These advancements led to the development of anabolic-androgenic steroids (AAS) and selective androgen receptor modulators (SARM), which offer improved bioavailability and longer-lasting effects (Androgenic Steroids, 2012).

AASs are derivatives of testosterone. They are commonly used recreationally by amateur athletes and physique-focused individuals. Testosterone increases muscle protein synthesis that results in muscle mass gain, ultimately improving strength (Kicman, 2008).

Some of the most commonly used oral AAS include methyltestosterone, methenolone acetate, mibolerone, oxandrolone, stanozolol, and oxymetholone. Injectable forms frequently used include boldenone undecylenate, clostebol, drostanolone propionate, nandrolone decanoate, trenbolone, testosterone cypionate, and stanozolol (Harvey et al., 2019).

SARMs represent another group of testosterone derivatives that selectively bind to androgen receptors. They have emerged as promising agents with potential applications in diverse medical conditions, including sarcopenia for improving muscle strength, as well as the treatment of osteoporosis and breast cancer, while minimizing the risk of excessive androgenic side effects. However, none of these compounds have been approved for human use (Mohideen et al., 2023; Böker et al., 2023). They were initially believed to provide anabolic effects similar to those of AASs while offering a better safety profile and reducing androgenic side effects such as virilization, androgenic alopecia, and prostatic hyperplasia (Mohideen et al., 2023). SARMs, comparatively to AASs, function as agonists of the androgen receptor in target tissues such as skeletal muscle and bone. However, SARMs differ from AAS by demonstrating reduced activity in non-targeted tissues, including the heart, prostate, and liver (Narayanan et al., 2018). Most AASs are ineffective when administered orally due to rapid hepatic clearance while SARMs are characterized by their better oral bioavailability and therefore presumed lower risk of hepatotoxicity (Mohideen et al., 2023). A significant problem is the vast online availability of dietary supplements labeled as containing SARMs. They often do not contain any SARMs or provide false information about their composition.

2. METHODOLOGY

The authors searched the PubMed, Scopus, and Google Scholar databases using phrases such as “Selective Androgen Receptor Modulators,” “Androgenic-Anabolic Steroids,” “Hepatotoxicity,” “Drug-Induced Liver Injury” and “Hepatocellular carcinoma”. Boolean operators such as “AND” and “OR” were applied to refine and optimize the search results. Articles published in English between January 2014 and December 2024 were considered. Furthermore, additional articles were identified by screening the bibliographies of studies retrieved through the database searches.

3. RESULTS AND DISCUSSION

AAS addiction

AAS addiction is driven by three primary factors. First, the anabolic effects of AAS contribute to psychological dependence, as users develop a fear of losing muscle mass upon discontinuation. Second, the androgenic effects play a critical role, as cessation can lead to severe withdrawal symptoms, including depression and prolonged dysregulation of the gonadotropic axis, resulting in sexual dysfunction and persistent fatigue. Lastly, the hedonic effect is linked to increased dopaminergic activity in the nucleus accumbens, which may conduce to the compulsive use of AAS (Kanayama et al., 2010).

Side effects of AAS

AASs are associated with several common adverse effects, including dyslipidemia, hypertension, hypogonadism, infertility, aggression, mood disorders, addiction, hepatotoxicity and nephrotoxicity (Petrovic et al., 2022). Research suggests that AASs suppress hepcidin, a key regulatory peptide in iron homeostasis. The downregulation of hepcidin increases the levels of iron absorbed from intestine and facilitates the incorporation of iron into red blood cells, thereby stimulating erythropoiesis. (Beggs et al., 2014)

These risks have caused stricter regulations on AAS availability, particularly within major sports organizations. Such organizations not only ban the use of anabolic steroids but also conduct testing to detect illegal substances to prevent unfair competition and mitigate the potential health hazards linked to their use (Petrovic et al., 2022). The misuse of AAS is linked not only to severe adverse effects but also to significant risk arising from unsafe and irresponsible practices. Study indicates that approximately 13% of individuals who misuse AAS have reported sharing and reusing needles for injectable forms of these substances (Parkinson and Evans, 2006).

Furthermore, the usage of AAS injectors is associated with increased prevalence to infection with blood-borne virus such as hepatitis B and hepatitis C viruses (Aitken et al., 2002).

Table 1. Summary of the impact of AAS on liver heath.

Study	Hepatotoxic effect of AAS
Androgenic Steroids, 2012	Elevation of serum enzymes activity, cholestasis, peliosis hepatis and hepatic adenoma and hepatocellular carcinoma.
Tillmann and Rockey, 2020	Bland cholestasis which is characterized by an elevated bilirubin level that is typically much higher than the increase in aminotransferase levels.
Niedfeldt, 2018	Elevation of serum enzymes activity, cholestasis, peliosis hepatis, hepatic adenoma and hepatocellular carcinoma, or toxicant-associated fatty liver disease.
Evert and Dombrowski, 2008	Hepatocellular carcinoma that consistently develops in the absence of liver cirrhosis.
Gorayski et al., 2008	Malignant transformation to hepatocellular carcinoma from a pre-existing hepatic adenoma.
Socas et al., 2005	Hepatic adenoma.
Johnson et al., 1972	Hepatocellular carcinoma.
Martin et al., 2008	Hepatic adenoma regrowth, complicated by life-threatening hemorrhage.
Schwingel et al., 2011	Toxicant-associated fatty liver disease.
Stangl et al., 2021	The incidence of liver injury in transgender population receiving hormone therapy is found to be very low.
El Sherrif et al., 2013b	Bland benign recurrent intrahepatic cholestasis.

Hepatotoxicity

Many AAS cannot be taken orally due to rapid hepatic clearance. This has prompted the development of 17α-alkylated AAS, which reduces hepatic metabolism but increases the risk of liver toxicity. However, liver tumors have also been associated with unmodified AAS. AAS have been linked to direct hepatotoxicity, acute cholestatic syndrome, peliosis hepatis, and the development of liver tumors (Androgenic Steroids, 2012). A summary of the impact of AAS on liver heath is presented in Table 1.

The R-value is a critical parameter used to evaluate the biochemical pattern of drug-induced liver injury (DILI). It is calculated as the ratio of ALT in ULN to ALP to ULN. The R-value is >5 indicates a hepatocellular pattern, R-value between 2 and 5 reflects a mixed pattern, where both hepatocellular and cholestatic processes are involved, R-value <2 suggests "cholestatic" pattern (Tillmann and Rockey, 2020).

The most common tumors associated with AAS use are hepatic adenomas and hepatocellular carcinoma (HCC), although cholangiocarcinomas and angiosarcomas have also been reported (Niedfeldt, 2018). The liver is rich in estrogen and androgen receptors, which may explain the frequent occurrence of rare tumors such as hepatic adenomas in young women taking oral contraceptives and men using AAS (Evert and Dombrowski, 2008; Gorayski et al., 2008; Socas et al., 2005). HCC associated with AAS use generally has a more favorable prognosis compared to HCC linked to cirrhosis or chronic viral hepatitis (Poon et al., 2001). A nonsurgical approach may be preferable to resection of a lesion in the liver, as benign adenomas might regress spontaneously upon discontinuation of AAS use, particularly if detected early (Nakao et al., 2000).

Tumor enlargement and recurrence have been observed in cases where steroid consumption is resumed (Johnson et al., 1972; Martin et al., 2008). AAS users have a sixfold higher risk of developing toxicant-associated fatty liver disease (TAFLD), despite being younger and not exhibiting symptoms of insulin resistance (Schwingel et al., 2015, 2011). The development of these disorders may result from disruptions in antioxidative mechanisms, increased bile acid production, and the stimulation of hepatocyte hyperplasia. Most cases of toxicity are managed with supportive care, and liver function typically returns to normal upon stopping AAS use. Nonetheless, some long-term consequences are irreversible (Loscalzo et al., 2022). Current data suggest that AAS-related DILI generally has a favorable prognosis, as most cases resolve completely, with only a small fraction of cases resulting in hepatic failure or death (Abeles et al., 2020).

Notably, prolonged use of AAS can result in peliosis hepatis, nodular regenerative hyperplasia, hepatic adenomas, and hepatocellular carcinoma. Peliosis hepatis is a vascular disorder defined by the formation of multiple blood-filled sinusoids, causing the liver to become enlarged and fragile. The pathogenesis of peliosis hepatis may be associated with the activity of vascular endothelial

growth factor (VEGF). VEGF is a key angiogenic cytokine involved in the formation of blood vessels. Importantly, two independent animal studies have reported divergent findings concerning the role of VEGF in the pathogenesis of peliosis hepatis. In a murine model, a study demonstrated a correlation between factors secreted by melanoma cells—including VEGF—and the development of peliosis hepatis. The authors suggested that this phenomenon may be due to the pro-proliferative effects of VEGF on hepatic endothelial cells. Conversely, a separate investigation using a rat model revealed a protective role of VEGF against the formation of peliotic lesions, suggesting a context-dependent or species-specific effect of VEGF signaling in hepatic vessels (Edwards et al., 2002)

The mechanism underlying AAS-induced liver injury is complex. One hypothesis is that AASs may impair bile transport. AAS binds to canalicular membrane transporters, leading to the accumulation of toxic bile acids and, as a result, pump failure. Another possibility is that AAS reduces the permeability of the biliary epithelium to water and disrupts osmotic agents, such as glutathione, thereby decreasing bile flow in the canalicular ducts (Mohideen et al., 2023).

Agriesti et al., (2020) in their in vitro study found that disruptions in mitochondrial respiratory chain reactions are associated with liver injury induced by high doses of nandrolone. A multi-center prospective study examining liver injury incidence among 889 transgender female and 1044 transgender male patients receiving hormone therapy that includes AASs found no significant increases in ALT, AST, alkaline phosphatase (ALP), or GGT (Stangl et al., 2021).

AAS may inhibit the expression of the genes ATPB81 and ABCB11, which likely contributes to the cholestatic damage by impairing bile salt transport and reducing excretion of different hepatic ectoenzymes (El Sherrif et al., 2013b). As of now, there have been no documented cases of SARMs causing conditions like peliosis hepatis, hyperplasia, or liver tumors. There is potential risk for such liver injuries as mentioned above with prolonged and high-dose use of SARMs.

4. CONCLUSIONS

The main objective of this review is to accentuate the notable health concerns such as hepatotoxicity associated with the use of AASs and SARMs. These substances are widely used by athletes to enhance physical performance and muscle mass despite their detrimental effects on health. AASs have been linked to a range of hepatotoxic manifestations including direct hepatocellular damage, acute cholestatic syndrome, peliosis hepatis, and development of benign and malignant liver tumors, predominantly hepatic adenomas and HCC. The mechanisms underlying AAS-induced liver injury are intricate. Notwithstanding most cases of AAS-related DILI resolve following discontinuation, prolonged use may lead to irreversible hepatic damage. SARMs may be perceived as a safer alternative to AAS. Furthermore, the addictive potential of AAS complicates cessation efforts. We believe that future research should aim to deepen our understanding of the molecular mechanisms involved in AAS-induced hepatotoxicity and to establish standardized screening protocols for at-risk populations. Physicians should remain vigilant for signs of AAS and SARM abuse when dysfunction evaluating patients with idiopathic liver diseases, particularly young male athletes presenting with biochemical hepatocellular or cholestatic injury patterns. We believe that a comprehensive approach incorporating early detection, and evidence-based treatment protocol are essential in management of patients taking performance-enhancing substances.

Author's Contributions

Conceptualization, M.B and P.K.; Methodology, J.B.K., M.L.; Software, P.K.; Validation, M.L., A.Z. and M.B.; Formal Analysis, A.B, J.B.K, A.M., K.H.; Investigation, P.K., M.B.; Resources, K.H., M.B.; Data Curation, P.K., K.S., K.H.; Writing – Original Draft Preparation, A.B., M.L., A.M., K.S. and A.Z.; Writing – Review & Editing, M.K., M.W., P.K., K.H. and J.B.K.; Supervision, A.B., A.M.; Project Administration, A.B., J.B.K., A.Z., M.L., A.Z., K.S., M.K., A.M., K.H, M.B.

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Conflict of interest
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Data and materials availability
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