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# ADVERSE EFFECTS OF TRENBOLONE: A STRUCTURED REVIEW OF CASE REPORTS IN HUMANS

**Lucas Caseri Câmara*[1], Diogo Pinto da Costa Viana[2], Lucio de Sousa Monte Alto[3]**

[1]Department of Specialization in Clinical Anabolism College of Governance, Engineering, and Education of São Paulo - FGE-SP, Brazil.

[2]Chairman of the Brazilian Society of Endocrinology and Metabolism in Sports and Exercise.

[3]President of the Brazilian Society of Endocrinology and Metabolism in Sports and Exercise.

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***Corresponding Author: Lucas Caseri Câmara**
Department of Specialization in Clinical Anabolism College of Governance, Engineering, and Education of São Paulo - FGE-SP, Brazil.
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**ABSTRACT**

**Introduction:** Trenbolone is a potent anabolic steroid widely used in bodybuilding, despite its lack of approval for human use. Reports of adverse effects have increased, but systematic data on its health risks remain limited. This study aims to systematically review documented adverse effects of trenbolone in humans through published case reports. **Methods:** A structured review of case reports was conducted using PubMed, EMBASE, and CENTRAL databases, including studies up to 2024. The search strategy utilized Medical Subject Headings (MeSH) terms related to trenbolone. Inclusion criteria encompassed reports of adverse effects in humans with documented trenbolone use, while experimental studies, animal models, and reviews without original case data were excluded. Extracted data included patient demographics, trenbolone dosage, administration patterns, clinical presentation, toxicological findings, and affected organ systems. **Results:** A total of 23 case reports met the inclusion criteria. The most frequently reported complications involved the cardiovascular system (heart failure, myocardial infarction, ischemic stroke), hepatic system (cholestatic liver injury, hepatitis), renal system (nephropathy, acute kidney injury), and neuropsychiatric effects (psychosis, aggression, delirium). Most cases involved young male users, with trenbolone frequently sourced from unregulated markets. Toxicological confirmation was reported in only a minority of cases, complicating causality assessment. **Conclusion:** Trenbolone use is associated with severe health risks, particularly affecting the cardiovascular, hepatic, renal, and neuropsychiatric systems. The absence of clinical studies on its safety, along with widespread unregulated use, hinders risk assessment and patient management. Healthcare professionals should recognize potential adverse effects, promoting early detection and harm reduction. Further research is needed to clarify trenbolone's long-term impact on human health.

**KEYWORDS:** Trenbolone, Trenbolone Acetate, Anabolic Steroids, Testosterone, Androgens, Bodybuilding.

## INTRODUCTION

Trenbolone is a synthetic anabolic-androgenic steroid (AAS) belonging to the 19-nortestosterone class, alongside nandrolone and gestrinone.[1] Developed in the 1960s, its initial application was exclusively veterinary, aimed at improving feed efficiency and promoting muscle mass and weight gain in cattle. Due to its potent anabolic effects observed in these animals, trenbolone quickly gained interest within the bodybuilding community, where it became widely used despite lacking regulatory approval for human use.[1,2]

Trenbolone is distinguished by its exceptionally high anabolic potency. Preclinical studies suggest that its anabolic activity is approximately five times greater than that of testosterone, with measurable effects observed even at relatively low doses (3–7 mg/day in animal models).[3] However, these same studies also indicate a concerning toxicity profile, with potential severe adverse effects on multiple physiological systems. These safety concerns prevented trenbolone from advancing to clinical trials, thereby precluding a rigorous evaluation of its safety and therapeutic efficacy in humans.[1-3]

Despite the absence of formal clinical studies, trenbolone remains widely used illicitly, particularly among bodybuilders.[4,5] However, data on its clinical safety in humans are scarce, with most knowledge regarding its risks derived from case reports documenting adverse effects. This gap in the literature poses a significant challenge for healthcare professionals treating AAS users, particularly those managing complications associated with trenbolone abuse.[4,5]

Given this scenario, a comprehensive literature review using a systematic methodology is essential to consolidate current knowledge on trenbolone's adverse effects in humans. Thus, the primary objective of this manuscript is to systematically review published case reports, identify patterns of adverse effects, and provide clinically relevant information to aid in patient management and inform future guidelines.

## METHODS

To conduct a structured search, three major scientific databases were initially used, employing a systematic search strategy[6] based on Medical Subject Headings (MeSH) terms: **PubMed** (www.pubmed.gov), **EMBASE** (www.embase.com), and **CENTRAL (Cochrane Collaboration)** (www.cochranelibrary.com). The methodology for the structured search is outlined separately for each database in the following table (**Table 1**).

**Table 1: Scientific Databases (PubMed, EMBASE, CENTRAL) and Search Strategy.**

| Database | Search Strategy |
|---|---|
| PubMed | "Trenbolone Acetate"[Mesh] OR "Acetate, Trenbolone" OR "Trenbolone Acetate, (17beta)-isomer" OR "Trienbolone Acetate" OR "Acetate, Trienbolone" OR "17 beta-acetoxyestra-4,9,11-trien-3-one" OR "Progro T-S" OR "Progro T S" OR "Progro TS" OR Trenbolone OR Trienbolone |
| EMBASE | #1 'trenbolone'/exp<br>#2 #1 AND 'human'/de |
| CENTRAL | MeSH descriptor: [Trenbolone Acetate] explode all trees |

For study inclusion, we selected articles published in indexed journals that reported adverse effects in humans and documented trenbolone use. Exclusion criteria included experimental studies or animal models, narrative reviews without case reports, and studies that failed to adequately describe the clinical presentation associated with trenbolone use.

Additionally, data extraction focused on obtaining relevant information from published cases, including patient gender, age, history of trenbolone use (dose, source of acquisition, and whether the substance was used in isolation), medical history, history of other drug use/abuse, affected organ system, laboratory findings, toxicological screening results, and imaging studies performed.

## RESULTS

According to the structured search conducted across the three different scientific databases and applying the predefined inclusion and exclusion criteria, a total of 907 references were identified in PubMed (27 when filtered for case reports), 186 references in EMBASE (12 when filtered for case reports), and 6 references in CENTRAL (none of which were case reports, as all were animal model studies).

Among the 39 initially identified case reports related to trenbolone use and adverse effects in humans, 23 studies were ultimately selected for further analysis and presentation. The remaining 16 studies were excluded for being duplicates in more than one database, for failing to meet the inclusion and exclusion criteria, or for not being actual case reports on the intended subject (as they appeared in the search results due to potential imprecision in the search methodology).

For better readability and comprehension of the findings, the following three tables (**Tables 2–4**) have been divided, and together they analyze the 23 case studies related to Trenbolone abuse[7-29], providing an overview of clinical outcomes, dosages, sources, usage patterns, and the toxicological and personal characteristics of the affected individuals.

**Table 2** focuses on demographic characteristics and clinical outcomes. All patients are male, with ages ranging from 20 to 60 years. The majority fall within the 20 to 40-year range, highlighting young adults as a potential highest-risk group. The reported outcomes include a wide range of complications, such as: 1) Cardiovascular: Advanced ischemic heart disease (posthumous), heart failure (posthumous), heart failure (LVH – left ventricular hypertrophy, pulmonary edema, ACD – arterial coronaries disease stenosis), dilated cardiomyopathy, myocardial infarction, LVH, pulmonary edema (posthumous), ischemic stroke; 2) Hepatic: Cholestatic liver injury, acute cholestatic syndrome, severe cholestasis, cholestatic hepatitis; 3) Psychiatric / Psychological / Neurological: Mallory-Weiss syndrome; Wernicke's encephalopathy, delirium, extreme violence (homicide), acute psychosis; and 4) Other: Fulminant acne, recurrent renal infarction, associated severe cholestasis and nephropathy, rhabdomyolysis and acute kidney injury, recurrent ophthalmic herpes zoster, abrupt-onset diabetes, gluteal necrosis, acute pancreatitis.

**Table 2: Sex, Age, and Outcomes of Trenbolone Abuse.**

| # | Author/Year | Sex | Age | Outcome |
|---|---|---|---|---|
| 1 | Christopoulos P. et al. (2012)[7] | M | 32 | Mallory-Weiss Syndrome; Wernicke's Encephalopathy |
| 2 | Kang G. et al. (2019)[8] | M | 23 | Cholestatic liver injury |
| 3 | Kraus SL. et al. (2012)[9] | M | 22 | Fulminant acne |
| 4 | Coulburn S. et al. (2017)[10] | M | 43 | Recurrent renal infarction |
| 5 | Gheddar L. et al. (2021)[11] | M | 60 | Advanced ischemic heart disease (posthumous) |
| 6 | Flores E. et al. (2019)[12] | M | 27 | Acute Cholestatic Syndrome |
| 7 | El Khoury C. et al. (2017)[13] | M | 35 | Severe cholestasis and nephropathy |
| 8 | Gnanapandithan K. et al. (2019)[14] | M | 33 | Rhabdomyolysis and Acute Kidney Injury |
| 9 | LoBue S. et al. (2020)[15] | M | 34 | Recurrent Ophthalmic Herpes Zoster |
| 10 | Geraci M. et al. (2011)[16] | M | 33 | Abrupt-Onset Diabetes |
| 11 | Kintz P. et al. (2021)[17] | M | 59 | Heart failure (posthumous) |
| 12 | Lehmann S. et al. (2019)[18] | M | 34 | Heart failure (LVH, Pulmonary Edema, ACD stenosis) |

| | | | | |
|---|---|---|---|---|
| 13 | Anand J. et al. (2006)[19] | M | 21 | Severe cholestasis |
| 14 | Friedman O. et al. (2016)[20] | M | 35 | Gluteal necrosis |
| 15 | Khoodoruth M. et al. (2020)[21] | M | 33 | Delirium |
| 16 | Aknouche F. et al. (2021)[22] | M | 32 | Extreme Violence (Homicide) |
| 17 | Bispo M. et al. (2009)[23] | M | 40 | Dilated Cardiomyopathy |
| 18 | Kumar V. et al. (2019)[24] | M | 20 | Acute Pancreatitis |
| 19 | Boks M. et al. (2017)[25] | M | 24 | Cholestatic Hepatitis |
| 20 | Shahsavari Nia K. et al. (2014)[26] | M | 23 | Myocardial Infarction |
| 21 | Bey C. et al. (2023)[27] | M | 21 | Acute Psychosis |
| 22 | Gheddar L. et al. (2023)[28] | M | 29 | LVH, Pulmonary Edema (posthumous) |
| 23 | Choulerton J. et al. (2021)[29] | M | 34 | Ischemic Stroke |

**Table 3** addresses the doses, sources of acquisition, and usage patterns of Trenbolone. Many doses are not reported (NR) in the studies, but when documented, they vary widely, ranging from 25 mg/day to 1000 mg/week (usage patterns include prolonged cycles and continuous use).

The predominant sources are the black market and veterinary stores and only four cases (17.4%) reported Trenbolone as the sole AAS used (6 studies did not reported, 17 studies trenbolone was not used as isolated AAS).

**Table 3: Dose, Source, and Usage Pattern of Trenbolone.**

| # | Author/Year | Tren Dose | Source | Used as Isolated AAS? |
|---|---|---|---|---|
| 1 | Christopoulos P. et al. (2012)[7] | NR | NR | No |
| 2 | Kang G. et al. (2019)[8] | 1000 mg/week (8 weeks) | NR | NR |
| 3 | Kraus SL. et al. (2012)[9] | NR | NR | No |
| 4 | Coulburn S. et al. (2017)[10] | NR, 5 years | NR | No |
| 5 | Gheddar L. et al. (2021)[11] | NR | NR | NR |
| 6 | Flores E. et al. (2019)[12] | NR | NR | No |
| 7 | El Khoury C. et al. (2017)[13] | 300 mg/week | NR | No |
| 8 | Gnanapandithan K. et al. (2019)[14] | NR, two applications | Online (black market) | NR |
| 9 | LoBue S. et al. (2020)[15] | 200 mg/week, 6 weeks | NR | No |
| 10 | Geraci M. et al. (2011)[16] | NR | Veterinary store | No |
| 11 | Kintz P. et al. (2021)[17] | NR | Eastern European vial (exact origin unknown) | No |
| 12 | Lehmann S. et al. (2019)[18] | NR | NR | No |
| 13 | Anand J. et al. (2006)[19] | 25 mg/day, 3 weeks, 2 cycles | Thailand vial (exact origin unknown) | NR |
| 14 | Friedman O. et al. (2016)[20] | NR, 3 weeks | Black market | No |
| 15 | Khoodoruth M. et al. (2020)[21] | Up to 200 mg/day | NR | No |
| 16 | Aknouche F. et al. (2021)[22] | NR | NR | No |
| 17 | Bispo M. et al. (2009)[23] | 500-700 mg/week, 6-10 weeks | NR | No |
| 18 | Kumar V. et al. (2019)[24] | Gradual increase up to 400 mg/week | Black market (online) | No |
| 19 | Boks M. et al. (2017)[25] | 50 mg/day, 8 weeks | NR | No |
| 20 | Shahsavari Nia K. et al. (2014)[26] | Daily > 1 year | NR | NR |
| 21 | Bey C. et al. (2023)[27] | NR | Black market | NR |
| 22 | Gheddar L. et al. (2023)[28] | NR | NR | No |
| 23 | Choulerton J. et al. (2021)[29] | 50 mg/day, 12 weeks | NR | No |

**Table 4** explores the toxicological findings, personal health history, and drug use background. Toxicological analyses confirmed the presence of Trenbolone in 7 cases (30.4%), primarily detected in biological samples such as blood, hair, and nails. In contrast, 18 cases did not undergo toxicological testing to verify Trenbolone presence.

Additionally, the most prevalent adverse health history reported in **Table 3** was chronic AAS use, present in 6 cases (26.0%), followed by one case of dyslipidemia, one case of heart failure, and one case of bipolar disorder. Two studies did not report any health history (NR), and 17 cases (73.9%) had no noteworthy health conditions relevant to the observed outcomes (NDN).

Finally, regarding drug use history, alcohol consumption was the most frequently reported factor (n=3; 13.0%), with one case also reporting a history of former smoking. Eight studies (34.8%) did not report any drug use (NR), while 12 studies (52.2%) assessed drug use and found negative results (No) (**Table 4**).

**Table 4: Toxicological Examination, Personal History of Drug Use, and Health Background.**

| # | Author/Year | Toxicological? | Drug History | Health Background |
|---|---|---|---|---|
| 1 | Christopoulos P. et al. (2012)[7] | No | Alcohol (non-dependent) | NDN, diet poor in thiamine |
| 2 | Kang G. et al. (2019)[8] | No | NR | NDN |
| 3 | Kraus SL. et al. (2012)[9] | No | NR | NR |
| 4 | Coulburn S. et al. (2017)[10] | No | NR | NDN |
| 5 | Gheddar L. et al. (2021)[11] | Yes, autopsy (Tren detected in femoral blood, cardiac tissue, bile, hair) | NR | NDN, chronic AAS use |
| 6 | Flores E. et al. (2019)[12] | No | NR | NDN |
| 7 | El Khoury C. et al. (2017)[13] | No | Excessive alcohol use on weekends | NDN, controlled dyslipidemia |
| 8 | Gnanapandithan K. et al. (2019)[14] | No | No | NDN |
| 9 | LoBue S. et al. (2020)[15] | No | No | NDN |
| 10 | Geraci M. et al. (2011)[16] | No | No | NDN |
| 11 | Kintz P. et al. (2021)[17] | Yes, autopsy (Tren detected in hair) | No | Heart failure |
| 12 | Lehmann S. et al. (2019)[18] | Yes, autopsy (traces in blood) | No | NDN, chronic use of ergogenic substances |
| 13 | Anand J. et al. (2006)[19] | No | NR | NDN |
| 14 | Friedman O. et al. (2016)[20] | No | NR | NDN, chronic AAS use |
| 15 | Khoodoruth M. et al. (2020)[21] | No | No | One episode of mania (bipolar disorder) |
| 16 | Aknouche F. et al. (2021)[22] | Yes, forensic (Tren in blood) | NR | NDN |
| 17 | Bispo M. et al. (2009)[23] | No | No | Chronic AAS use (10 years) |
| 18 | Kumar V. et al. (2019)[24] | No | No | Chronic AAS use |
| 19 | Boks M. et al. (2017)[25] | No | No | NDN, previous AAS use |
| 20 | Shahsavari Nia K. et al. (2014)[26] | No | No | NDN |
| 21 | Bey C. et al. (2023)[27] | No | No | NDN |
| 22 | Gheddar L. et al. (2023)[28] | Yes, autopsy (Tren in blood, hair, and nails) | No | NR |
| 23 | Choulerton J. et al. (2021)[29] | No | Former smoker, moderate alcohol consumption | NDN |

## DISCUSSION

The primary finding of this review, which included an analysis of 23 published case reports, was that abusive use of trenbolone was associated with a range of adverse health outcomes, including cardiovascular complications (advanced ischemic heart disease, heart failure, left ventricular hypertrophy, pulmonary edema, coronary artery disease stenosis,

dilated cardiomyopathy, myocardial infarction, and ischemic stroke), hepatic complications (cholestatic liver injury, hepatitis), psychiatric/ psychological/ neurological effects (Mallory-Weiss syndrome, Wernicke's encephalopathy, delirium, extreme violence and homicide, acute psychosis), and other effects such as fulminant acne, nephropathy and renal infarction, rhabdomyolysis, ophthalmic herpes zoster, abrupt-onset diabetes, gluteal necrosis, and acute pancreatitis.[7-29]

The aggregated data from the twenty-three case reports identified and presented in this review align with previously proposed potential adverse effects of trenbolone use. In a recent review published by Borecki R. et al. (2024)[4], various health impairments have been highlighted, affecting the nervous system, muscle and adipose tissues, immune, reproductive, and cardiovascular systems, among others. These findings are summarized in the following table (**Table 5**).

**Table 5: Adverse effects of Trenbolone in selected organ / system.[4]**

| Organ/System | Effects of Trenbolone |
|---|---|
| **Nervous System** | - Cerebral cortical atrophy.<br>- Beta-amyloid accumulation (Ab42).<br>- Cognitive and memory deficits.<br>- Aggressiveness and impulsivity. |
| **Reproductive System** | - Functional hypogonadism.<br>- Testicular atrophy.<br>- Reduced spermatogenesis. |
| **Immune System** | - Suppression of innate and adaptive immunity.<br>- Reduction of T lymphocytes and complement system activity. |
| **Cardiovascular System** | - Increased LDL and reduced HDL.<br>- High blood pressure.<br>- Arrhythmias and elevated cardiovascular risk. |
| **Injection Site** | - Inflammation and fibrosis.<br>- Tissue necrosis (Nicolau Syndrome). |
| **Others** | - Gynecomastia due to progesterone receptor activation and elevated prolactin levels. |

Regarding the cardiovascular system, we previously published a letter to the editor providing a more detailed description of three post-mortem cases, which presented cardiovascular alterations such as cardiac hypertrophy, heart failure, coronary artery disease, and pulmonary edema.[30] Forensic analysis detected high concentrations of trenbolone in biological matrices such as blood, hair, and nails, suggesting a potential contribution to these observed outcomes. This is particularly relevant when considering the chronic effects of dyslipidemia, increased atherogenesis (elevated LDL, reduced HDL), and hypertension associated with trenbolone use.[4,30]

In the current review, in addition to these three post-mortem cases[30], other vascular complications linked to trenbolone use were also observed, including myocardial infarction, dilated cardiomyopathy, heart failure, and ischemic stroke.[18,23,26,29]

Accordingly, as observed by Windfeld-Mathiasen J, et al. (2025)[31], over an average follow-up period of 11 years, individuals using anabolic-androgenic steroids (AAS) (n=1189) exhibited a significantly greater incidence of cardiovascular events compared to the control group (n=59,450). The use of AAS was linked to an elevated risk of acute myocardial infarction (adjusted hazard ratio [aHR] 3.00; 95% CI, 1.67–5.39), as well as percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG) (aHR 2.95; 95% CI, 1.68–5.18). Additionally, AAS users demonstrated a higher likelihood of developing venous thromboembolism (aHR 2.42; 95% CI, 1.54–3.80),

arrhythmias (aHR 2.26; 95% CI, 1.53–3.32), cardiomyopathy (aHR 8.90; 95% CI, 4.99–15.88), and heart failure (aHR 3.63; 95% CI, 2.01–6.55).[31]

Regarding the central nervous system (CNS), trenbolone is widely recognized for its neurotoxic effects. Due to its lipophilic nature, it readily crosses the blood-brain barrier, directly impacting cortical integrity, leading to neuronal atrophy, reduced NMDA receptor density, and the accumulation of neurotoxic proteins such as beta-amyloid.[1,2,4,32,33] These mechanisms are associated with cognitive and behavioral deficits, including memory loss, aggression, and impulsivity. Furthermore, trenbolone's modulation of GABA and NMDA receptors has been implicated as a key factor in the behavioral disturbances observed in users.[1,4,33]

Supporting the neurobehavioral effects, Aknouche F. et al. (2021)[22] reported a case of extreme violence (homicide) committed by a 32-year-old security officer, who fatally stabbed his girlfriend multiple times 90 days after using trenbolone in combination with methandienone. Forensic toxicology later confirmed high concentrations of both substances in biological samples, while also detecting no presence of alcohol or recreational drugs, effectively ruling out alternative causes and linking the event solely to AAS use.[22]

Additionally, Piatkowski T. et al. (2024)[34] investigated the relationship between trenbolone use and its psychosocial effects, such as psychological distress and aggression, using validated questionnaires in a sample of 282 participants. The results identified a significant association between higher trenbolone doses and increased levels of verbal aggression. However, other forms of aggression, such as physical aggression, anger, and hostility, did not show statistically significant associations.[34]

Moreover, in analyzing the most frequently observed adverse effects, this review documents multiple cases of hepatic and renal complications associated with trenbolone use (**Table 2**).[8,10,12-14,19,25] The hepatotoxic effects of AAS are well established in the literature, particularly in their capacity to induce drug-induced liver injury (DILI).[35] According to Robles-Diaz M, et al. (2015), the incidence of AAS-induced DILI has increased over time, accounting for 1% to 8% of all DILI cases.[35] A distinct phenotypic characteristic of AAS-induced liver damage is hyperbilirubinemia, regardless of whether the injury is hepatocellular (approximately 60% of cases) or cholestatic. One proposed mechanism is the inhibition of bile salt export pumps, leading to bile acid accumulation and subsequent hepatic dysfunction.[35] In fact, in the present review, three cases[10,13,14] presented trembolona use and kidney damages, as seen in **Table 2**.

Furthermore, increased bilirubin levels in cholestatic cases may be associated with a higher risk of acute kidney injury (AKI).[36] Bile acid nephropathy, a form of AKI triggered by severe hyperbilirubinemia, was described in a case report by Al Awadhi et al. (2021).[36] Their study detailed a 27-year-old patient who developed cholestatic liver injury and subsequent bile acid nephropathy due to AAS abuse, ultimately requiring hemodialysis.[36]

Besides cardiovascular, hepatic, renal, and neuropsychiatric complications, some case reports have described less frequent adverse effects, such as sudden-onset diabetes[16], ocular herpes zoster[15], and fulminant acne.[9] While these findings are noteworthy, the lack of controlled studies in humans makes it difficult to determine a definitive causal relationship between trenbolone use and these manifestations, as well as to further clarify the potential underlying pathophysiology.[4]

This study clearly presents limitations inherent to case report-based methodology. Although the data collected, as we think, provide valuable insights into the potential adverse effects of trenbolone, the absence of controlled human studies prevents a definitive establishment of causality. Additionally, as observed in the results (**Table 3**), most users reported obtaining trenbolone from the black market, where adulterated drugs are frequently found due to the lack of regulatory oversight.[37] This uncontrolled distribution may influence the observed outcomes, a concern that we have previously reviewed and published.[37,38] Polypharmacy is another crucial factor, as it is common in the non-therapeutic use of AAS.[38] Trenbolone is rarely used in isolation, for example, 17.4% in the present review, making it difficult to differentiate its specific effects from those of other substances.[37] Finally, the lack of toxicological confirmation tests in most cases (almost 70.0% in the preset review) further limits the reliability of trenbolone exposure as the primary causal factor.

Finally, based on our observations, and in previous published studies[38-40], we propose some key considerations to assist healthcare professionals. The first concerns the importance clinical monitoring of AAS users, including trenbolone use, as this substance is not regulated for human use and the lack of controlled studies enable standardized adverse events treatment protocol. Thus, health professionals, especially physicians, should be vigilant for early signs of renal, hepatic, and cardiovascular toxicity. Second, there is a need to enhance education and awareness among healthcare professionals and users regarding potential risks, clinical monitoring, and early diagnosis, aiming to reduce the likelihood of severe health consequences.[38-40]

## CONCLUSION

This structured review, conducted across multiple scientific databases, analyzed 23 case reports of adverse effects associated with trenbolone use, with a particular focus on cardiovascular, hepatic, renal, and neuropsychiatric complications. The findings align with existing scientific literature, which suggests that AAS, including trenbolone, may pose significant health risks, such as heart failure, myocardial infarction, cholestatic liver injury, nephropathy, and psychiatric disorders like psychosis and aggression.

A major challenge in understanding trenbolone toxicity is the lack of clinical studies evaluating its safety and efficacy in humans. The predominant use of trenbolone from black-market sources (potentially adulterated), combined with polypharmacy (including other ergogenic substances and additional AAS) and the concomitant abuse of legal and illegal drugs, significantly hampers the ability to infer causality for the observed adverse effects. Furthermore, even among the case reports included in this review, toxicological analysis was not performed in all cases, meaning that trenbolone presence in the system was not always confirmed.

From a clinical perspective, healthcare professionals, particularly physicians, should be vigilant regarding the potential toxicity of trenbolone, especially among young male users seeking performance and aesthetic enhancement. Given the lack of standardized treatment guidelines, continuous monitoring and early detection of potential complications in the cardiovascular, hepatic, renal, and neuropsychiatric systems are of utmost importance.

Considering these findings, greater education for both users and healthcare professionals should be prioritized, with a focus on harm reduction strategies as part of public health campaigns. Future research aimed at better mapping the specific effects of trenbolone, including controlled clinical trials and longitudinal follow-up studies of users, would be highly valuable. Finally, until more robust evidence becomes available, healthcare professionals should remain vigilant

and adopt a harm reduction approach when engaging with and monitoring AAS users, ensuring a nonjudgmental and supportive clinical stance.

## COMPETING INTERESTS DISCLAIMER

Authors have declared that they have no known competing financial interests OR non-financial interests OR personal relationships that could have appeared to influence the work reported in this paper.

## DISCLAIMER (ARTIFICIAL INTELLIGENCE)

The authors declare that generative AI was used solely during the final stage of manuscript preparation (post-writing) and exclusively for linguistic refinement in the English language (Name: ChatGPT; Version: GPT-4; Model: OpenAI's Large Language Model; Source: OpenAI - https://openai.com). No original text was generated or substantively edited by the AI.

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