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Risk and Clinical Patterns of Tendon Injury and Rupture Associated with Anabolic-Androgenic Steroids and Exogenous Testosterone: A Systematic Review

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ABSTRACT

Anabolic-androgenic steroids (AAS) and exogenous testosterone may increase muscle strength more rapidly than tendon adaptation, potentially increasing the risk of tendon injury and rupture. This systematic review evaluated the association between AAS or exogenous testosterone exposure and tendon injury or rupture, while describing injury patterns, potential mechanisms, structural findings, management approaches, and clinical outcomes. PubMed, the Cochrane Library, and ScienceDirect databases were searched through July 12, 2026, following the PRISMA guidelines. Human observational studies, case series, and case reports involving adult participants were included. The risk of bias was assessed using the Newcastle–Ottawa Scale and Joanna Briggs Institute critical appraisal tools, and the findings were synthesized narratively. Eight studies involving 1,150,337 participants were included. Long-term AAS users demonstrated a ninefold higher hazard of first tendon rupture compared with nonusers. Prescribed testosterone use was associated with increased odds of Achilles tendon injury (adjusted odds ratio = 1.24) and quadriceps tendon injury (odds ratio = 5.4), as well as subsequent surgical intervention. Among 35 case-based patients, 32 experienced pectoralis major tendon ruptures, predominantly during bench-pressing activities. Imaging and histopathological findings indicated tendinopathy, degenerative changes, and neovascularization, while most patients who underwent surgical treatment achieved favorable recovery outcomes. AAS and exogenous testosterone exposure are associated with tendon injuries across various clinical settings; however, heterogeneity in study designs and exposure definitions limits causal interpretation and quantitative pooling.

INTRODUCTION

Anabolic-androgenic steroids (AAS) are synthetic derivatives of testosterone developed to enhance anabolic effects, including protein synthesis, muscle growth, and

strength development. Although these agents have legitimate therapeutic applications, supraphysiological doses are frequently used without medical supervision to improve athletic performance and physical appearance. AAS use, which was once concentrated among elite athletes, has expanded to recreational bodybuilding and the general population, particularly among young and middle-aged adults (Kanayama et al., 2015). Recent evidence also indicates that AAS exposure is not limited to men, with substantially higher prevalence among female bodybuilders and recreational athletes compared with women in the general population (Albright et al., 2023). This broader pattern of exposure has raised concerns regarding adverse effects extending beyond the endocrine, cardiovascular, hepatic, reproductive, and psychiatric systems (Quinn et al., 2024; Rebello et al., 2023; Scarth et al., 2025; Seynnes et al., 2013).

Tendons transmit muscular forces to bone and rely on a highly organized extracellular matrix dominated by type I collagen. Their adaptation to progressive mechanical loading occurs more slowly than skeletal muscle hypertrophy, creating a potential imbalance when muscular strength increases rapidly. AAS may exacerbate this mismatch by producing substantial increases in muscle mass and force without proportional improvement in tendon capacity. Proposed mechanisms include altered collagen synthesis and turnover, collagen fibril abnormalities, reduced tendon elasticity, increased stiffness, and impaired remodeling under repetitive mechanical loading (Brinkman et al., 2024; Cleri et al., 2024). These changes may reduce the ability of tendons to absorb energy and withstand eccentric forces, allowing accumulated microdamage to progress to partial or complete rupture. However, experimental findings remain inconsistent across steroid compounds, doses, exercise protocols, and tendon models, and their direct applicability to human exposure remains uncertain (Stefanou et al., 2023; Testa et al., 2023; William & Angellee, 2026; Yarnell et al., 2021).

Clinical evidence supports a potential association between AAS exposure and tendon rupture. In a controlled cohort of experienced male bodybuilders, long-term AAS users demonstrated a markedly higher hazard of first tendon rupture than comparable nonusers, with upper-body ruptures occurring exclusively among AAS users (De Castro Pochini et al., 2024). More recent administrative database studies have extended this concern to medically prescribed exogenous testosterone. Prescription testosterone exposure has been associated with increased odds of distal biceps tendon injury and subsequent surgical repair (Fenelon et al., 2016), Achilles tendon injury and surgery (Guzzoni et al., 2018), and quadriceps tendon or muscle injury requiring operative management (Jones et al., 2018). These findings suggest that tendon-related risks may not be limited to athletes using extreme supraphysiological regimens. However, claims-based studies generally identify broad diagnostic categories of tendon injury rather than clinically verified complete ruptures and often lack information regarding testosterone indication, serum concentration, adherence, physical activity, and injury mechanisms.

The clinical presentation of suspected AAS-associated tendon rupture is heterogeneous. Published reports have described injuries involving the pectoralis major, biceps, triceps, quadriceps, patellar, Achilles, and hamstring tendons. Pectoralis major rupture has been particularly reported among bodybuilders and weightlifters during the eccentric phase of

bench-press exercises. A recent case series demonstrated complete pectoralis major ruptures in young male bodybuilders, including patients actively using combinations of AAS at the time of injury (Kanayama & Pope, 2018). Imaging and histopathological findings from another cohort of AAS-using weightlifters identified contralateral tendinopathy, myxoid degeneration, neovascularization, and tendon fissures, suggesting that acute rupture may occur on a background of pre-existing structural degeneration (Meghani et al., 2024). Nevertheless, these observations are primarily derived from small case reports and case series, which cannot establish causality or accurately estimate the magnitude of risk.

A previous review published in 2018 summarized the mechanical, structural, and biological effects of AAS on tendons but preceded several large comparative database studies and recent clinical and histopathological reports. Available evidence also combines two clinically distinct exposures: nonmedical supraphysiological AAS use among athletes and prescribed testosterone therapy intended to restore physiological hormone levels. Furthermore, studies differ in participant characteristics, tendon locations, exposure definitions, outcome verification, and study designs. A contemporary synthesis is therefore required to distinguish these exposure types and integrate comparative risk estimates with clinical characteristics reported in individual rupture cases. This systematic review aimed to evaluate the association between AAS or exogenous testosterone exposure and tendon rupture while describing affected tendon sites, injury mechanisms, exposure patterns, structural findings, management strategies, and clinical outcomes.

METHOD

Search Strategy

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. A comprehensive literature search was performed in PubMed, the Cochrane Library, and ScienceDirect, with the final search conducted on July 12, 2026. No publication year restriction was applied to capture all relevant studies. The PubMed search strategy was: (“androgenic-anabolic steroid” OR “anabolic-androgenic steroid” OR “anabolic steroid” OR “testosterone therapy” OR “testosterone replacement therapy” OR “exogenous testosterone”) AND (“tendon rupture” OR “tendon injury” OR “tendon tear” OR tendinopathy OR “quadriceps tendon” OR “Achilles tendon” OR “pectoralis major tendon” OR “triceps tendon”). The Cochrane Library search strategy was: (“androgenic-anabolic steroid” OR “anabolic-androgenic steroid” OR “anabolic steroid” OR testosterone OR “testosterone replacement therapy”) AND (“tendon rupture” OR “tendon injury” OR “tendon tear” OR tendinopathy). The ScienceDirect search strategy was: (“anabolic steroid” OR “anabolic-androgenic steroid” OR testosterone) AND (“tendon rupture” OR “tendon injury” OR tendinopathy). The reference lists of eligible studies were also screened to identify additional relevant publications.

Eligibility Criteria

Studies were selected according to the Population, Exposure, Comparator, and Outcome framework. The population included adults aged 18 years or older from the general population, athletic populations, bodybuilders, and weightlifters. The exposure of interest was the use of anabolic-androgenic steroids, supraphysiological anabolic steroids, exogenous testosterone, or medically prescribed testosterone replacement therapy. The comparator consisted of individuals without AAS or exogenous testosterone exposure when a comparison group was available. The primary outcome was tendon rupture, while secondary outcomes included clinically coded tendon injury, tendon tear, tendinopathy, surgical tendon repair, tendon location, injury mechanism, imaging or histopathological abnormalities, treatment, functional recovery, and return to activity.

Observational cohort studies, cross sectional studies, retrospective comparative studies, case series, and case reports were eligible because of the limited and heterogeneous clinical evidence available. Only full text studies published in English and involving human participants were included. Animal studies, in vitro studies, narrative reviews, systematic reviews, editorials, letters without individual clinical data, conference abstracts, and studies that did not report tendon related outcomes among AAS or testosterone exposed individuals were excluded. Studies assessing corticosteroids without exposure to anabolic-androgenic steroids or exogenous testosterone were also excluded.

Study Selection

All records identified through the database searches were compiled in a single reference list. Titles and abstracts were screened independently by two reviewers against the predefined eligibility criteria. Duplicate records identified among the potentially eligible articles were removed before full text assessment. The full texts of the remaining records were then evaluated independently by three reviewers. Disagreements regarding study eligibility were resolved through discussion and consensus. The reasons for excluding studies during full text assessment were documented, and the overall selection process was presented using a PRISMA flow diagram.

Data Extraction

Data were extracted independently using a standardized data extraction form. The extracted information included author and publication year, country, study design, population characteristics, sample size, age, sex, type and duration of AAS or testosterone exposure, comparator characteristics, tendon involved, injury mechanism, outcome assessed, follow up duration, and principal findings. For comparative studies, the number of tendon injuries or ruptures in each group, incidence rates, odds ratios, adjusted odds ratios, hazard ratios, risk differences, 95% confidence intervals, and p values were recorded when available. For case reports and case series, details regarding the AAS regimen, tendon location, laterality, imaging findings, histopathological findings, surgical treatment, postoperative recovery, complications, and return to sport or activity were extracted. Any discrepancies in the extracted data were resolved by rechecking the original article and reaching consensus.

Risk of Bias Assessment

The methodological quality of the comparative observational studies was evaluated using the Newcastle Ottawa Scale. The assessment covered participant selection, comparability between exposed and unexposed groups, exposure ascertainment, outcome assessment, and adequacy of follow up. Case series were evaluated using the Joanna Briggs Institute Critical Appraisal Checklist for Case Series, while individual case reports were assessed using the Joanna Briggs Institute Critical Appraisal Checklist for Case Reports. Each study was independently appraised by three reviewers, and disagreements were resolved through discussion. Risk of bias findings were considered when interpreting the overall strength and consistency of the evidence.

Data Synthesis

A narrative synthesis was performed because of substantial heterogeneity in study design, participant characteristics, exposure definitions, tendon sites, outcome definitions, and reported effect measures. Comparative observational studies were synthesized separately from case reports and case series. Effect estimates were reported as presented by the original studies, including odds ratios, adjusted odds ratios, hazard ratios, risk differences, incidence rates, and corresponding 95% confidence intervals. Case based evidence was summarized descriptively using frequencies, percentages, and ranges. The findings were further grouped according to tendon location, injury mechanism, type of AAS or testosterone exposure, structural tendon abnormalities, treatment, and clinical outcome. A quantitative meta analysis was not performed because the available studies did not provide sufficiently homogeneous populations, exposures, comparators, and outcome definitions.

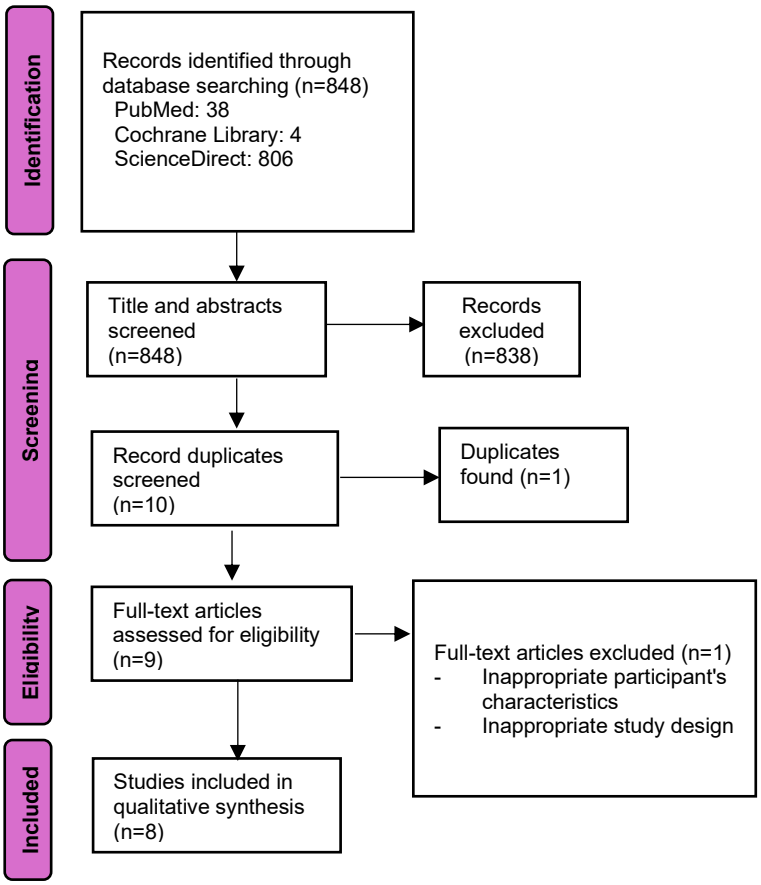

Figure 1. Diagram flow of literature search strategy for this systematic review

Table 1. Characteristics and results of the included studies

Study (Year, Country)	Design	Population	Tendon(s) Involved	Key Findings	Effect/Risk
Kanayama 2015 (USA)	Cross-sectional cohort	88 AAS users (≥2 yrs) vs 54 nonusers (bodybuilders)	Biceps, pectoralis major, triceps, Achilles, patellar, quadriceps, hamstring	At least one rupture in 19/88 AAS users vs 3/54 nonusers; upper-body ruptures occurred only in AAS users	First rupture HR 9.0 (95% CI 2.5–32.3), p<0.001
Fenelon 2016 (Ireland)	Case report	1 male, 29 yrs, current AAS use	Bilateral quadriceps + partial right ACL tear	Rupture during 280-kg squat attempt;	–

				surgically repaired, 5° extension lag at 5 months	
Dhillon 2020 (India)	Case report	1 male, 26 yrs, past AAS use early in career	Bilateral quadriceps (musculotendinous)	Rupture during clean-and-jerk; returned to national competition at 5 years, 80–85% performance recovery	–
Stefanou 2023 (Greece)	Retrospective case series	6 bodybuilders (3 AAS users, 3 nonusers)	Pectoralis major	AAS users lifted heavier loads (183.4 vs 147.6 kg); bench-press recovery 86.1% vs 81.3%; 1 AAS user died (myocardial infarction)	No formal effect estimate
Albright 2023 (USA)	Matched retrospective cohort	423,278 TRT users vs 423,278 controls	Achilles	Higher Achilles injury incidence in TRT users (377.8 vs 245.8 per 100,000 person-years)	aOR injury 1.24 (95% CI 1.15–1.33); aOR surgery 1.54 (95% CI 1.19–1.99)
Ntourantonis 2023 (Greece)	Case report	1 male, 32 yrs, cyclic AAS use >10 yrs	Right distal triceps	Near-complete rupture with no direct trauma;	–

				excellent functional outcome at 1 year post-surgery	
Meghani 2024 (USA)	Retrospective comparative database	151,797 testosterone users vs 151,797 controls	Quadriceps (muscle/fascia/tendon)	Substantially higher 1-year quadriceps injury rate in testosterone users, especially men	OR injury 5.4 (95% CI 3.4–9.2); OR surgery 4.7 (95% CI 2.0–13.8)
Pochini 2024 (Brazil)	Prospective case series + controls	26 AAS users with pectoralis major rupture vs 10 controls	Pectoralis major	Contralateral tendinopathy in most rupture cases (MRI); degenerative changes in 16/24 specimens	No rupture risk estimate reported

Figure 2. Risk of bias assessment results of the included studies

RESULTS AND DISCUSSION

Study Selection

The database search identified 848 records, comprising 38 records from PubMed, four from the Cochrane Library, and 806 from ScienceDirect. After title and abstract screening, 838 records were excluded, leaving 10 records for further evaluation. One duplicate was subsequently removed, and nine full text articles were assessed for eligibility. One article was excluded because of inappropriate participant characteristics and study design. Ultimately, eight studies were included in the qualitative synthesis, as presented in Figure 1.

Study Characteristics

The eight studies were published between 2015 and 2024 and were conducted in the United States, Greece, Ireland, India, and Brazil. The included evidence consisted of one cross sectional cohort study, two retrospective comparative database studies, two case series, and three case reports. Three comparative observational studies included 1,150,292 participants, whereas the five case reports and case series included 45 individuals, comprising 35 patients with tendon rupture and 10 uninjured controls. Across all studies, the total sample consisted of 1,150,337 participants.

The two administrative database studies included both men and women aged 35 to 75 years, whereas all athlete focused studies included only men. The ages of patients in the case reports and case series ranged from 21 to 40 years. Exposure definitions varied considerably across studies. The athlete focused studies evaluated nonmedical or supraphysiological AAS use, whereas the two database studies evaluated prescriptions for exogenous testosterone administered for at least three consecutive months. The characteristics and principal findings of the included studies are summarized in Table 1.

Risk of Bias Assessment

The risk-of-bias assessment showed that six of the eight included studies were judged to have an overall low risk of bias. Kanayama et al., Dhillon et al., Stefanou et al., Ntourantonis et al., Meghani et al., and Pochini et al. received low-risk judgments across the selection, performance, and detection or reporting domains.

Two studies had an overall unclear risk of bias. Fenelon et al. was judged to have an unclear risk in the performance domain, while the selection and detection or reporting domains were considered low risk. Albright et al. had an unclear risk in the selection domain but a low risk in the performance and detection or reporting domains. No study was classified as having a high overall risk of bias. The detailed domain-level assessment is presented in Figure 2.

Association Between AAS or Testosterone Exposure and Tendon Injury

Kanayama et al. evaluated 88 long term AAS users and 54 bodybuilders without a history of AAS use. At least one lifetime tendon rupture was reported by 19 of 88 AAS users, 21.6%, compared with three of 54 nonusers, 5.6%. AAS exposure was associated with a ninefold greater hazard of a first tendon rupture, HR 9.0, 95% CI 2.5 to 32.3, $p<0.001$. AAS users experienced 27 individual ruptures compared with five ruptures among nonusers. Upper body rupture occurred in 15 AAS users and no nonusers, corresponding to a risk difference of 0.17, 95% CI 0.09 to 0.25. In contrast, lower body rupture occurred in six AAS users and three

nonusers, with no statistically significant difference between groups, HR 3.1, 95% CI 0.7 to 13.8, p=0.13.

Albright et al. included 423,278 patients receiving testosterone replacement therapy and an equal number of matched controls. During two years of follow up, Achilles tendon injury was identified in 3,198 testosterone users and 2,081 controls. The incidence was 377.8, 95% CI 364.8 to 391.0, per 100,000 person years in the testosterone cohort and 245.8, 95% CI 235.4 to 256.6, per 100,000 person years in the control cohort. Testosterone therapy was associated with increased odds of Achilles tendon injury, aOR 1.24, 95% CI 1.15 to 1.33, p<0.001, with a number needed to harm of 371. Among patients with Achilles tendon injury, surgery was performed in 287 of 3,198 testosterone users, 9.0%, and 134 of 2,081 controls, 6.4%. Testosterone exposure was associated with increased odds of subsequent surgery, aOR 1.54, 95% CI 1.19 to 1.99, p<0.001.

Meghani et al. compared 151,797 testosterone users with 151,797 matched controls. Within one year, quadriceps injury occurred in 97 testosterone users, 0.06%, and 18 controls, approximately 0.01%. Testosterone exposure was associated with increased odds of quadriceps injury, OR 5.4, 95% CI 3.4 to 9.2, p<0.001. The association remained significant among men, OR 5.8, 95% CI 3.5 to 10.3, but was not demonstrated among women. Patients receiving testosterone were also more likely to undergo surgical quadriceps tendon repair following injury, OR 4.7, 95% CI 2.0 to 13.8.

Tendon Sites and Injury Mechanisms

Among the 35 patients described in the case reports and case series, 32, 91.4%, sustained pectoralis major tendon rupture, two sustained bilateral quadriceps injuries, and one sustained an almost complete distal triceps tendon rupture. All 32 pectoralis major injuries occurred during bench press exercises. The two quadriceps cases occurred during a 280 kg squat and the jerk phase of a clean and jerk lift. Overall, 34 of 35 patients, 97.1%, sustained their injuries during weightlifting. The remaining patient developed a triceps tendon rupture during a sudden upper limb movement while maneuvering a motorcycle, without a fall, collision, direct trauma, or recent exercise.

The broader cohort reported by Kanayama et al. demonstrated a more varied injury pattern. The affected tendons included the biceps, pectoralis major, triceps, Achilles, patellar, quadriceps, and hamstring tendons. Only six of 31 assessed rupture events occurred during weightlifting, whereas the remaining injuries occurred during other sports, falls, or the lifting and movement of objects.

Findings from Case Reports and Case Series

Stefanou et al. described six bodybuilders with complete pectoralis major rupture, three of whom were using AAS at the time of injury. Five ruptures occurred at the humeral insertion and one at the musculotendinous junction. All injuries occurred during flat bench press. The mean lifted load was 183.4 kg among AAS users and 147.6 kg among nonusers. All patients underwent surgical repair and achieved excellent postoperative outcomes. Full activity was resumed after a mean of 5.4 months, while preinjury bench press capacity recovered to 86.1%

among AAS users and 81.3% among nonusers. No postoperative complications were recorded, although one AAS user died from myocardial infarction within one year after surgery.

Pochini et al. evaluated 26 male weightlifters with acute total pectoralis major tendon rupture and 10 uninjured weightlifting controls. All rupture cases reported AAS use during or shortly before injury, and all injuries occurred during bench press. Contralateral MRI findings were reported as normal in two assessments, superior tendinopathy in 16, and total tendinopathy in 10. These categories totaled 28 observations despite a stated rupture cohort of 26 patients, indicating an internal reporting discrepancy. Among 24 evaluable tendon specimens, degenerative changes were present in 16, 66.7%, neovascularization in 16, 66.7%, and fissures in two, 8.3%. All control tendons were classified as normal.

Fenelon et al. reported a 29 year old male weightlifter with current AAS use who sustained complete bilateral quadriceps tendon rupture and a partial right anterior cruciate ligament tear while attempting a 280 kg squat. Both quadriceps tendons were surgically repaired. At five months, the patient had a bilateral extension lag of five degrees. The anterior cruciate ligament injury was managed conservatively, and symptoms of knee instability had resolved by eight months.

Dhillon et al. described a 26 year old elite weightlifter with a previous history of AAS use who sustained bilateral quadriceps musculotendinous tears during the jerk phase of a clean and jerk lift. Bilateral surgical repair was performed within 48 hours. The patient resumed training after nine months, returned to competitive sport after two years, and participated at the national level after five years. At six years, he had fully functional knees and had recovered approximately 80% to 85% of his preinjury performance.

Ntourantonis et al. reported an almost complete distal triceps tendon rupture in a 32 year old professional bodybuilder with more than 10 years of cyclic AAS exposure and nearly daily stanozolol use for 2.5 years. Surgical repair was performed using suture anchors and a modified double row technique. At six months and one year, the patient had no residual pain or limitation of motion, achieved perfect Mayo and Oxford elbow scores, and recovered triceps strength to the preinjury level.

This systematic review found a consistent association between exposure to anabolic-androgenic steroids or exogenous testosterone and an increased occurrence of tendon injury or rupture. The strongest direct evidence was provided by the controlled study of experienced bodybuilders, in which long-term AAS users had a ninefold higher hazard of experiencing a first tendon rupture than nonusers with comparable weightlifting experience.5 Large retrospective database studies also identified increased odds of Achilles and quadriceps tendon injuries among patients receiving prescribed testosterone.7,8 Meanwhile, the case reports and case series showed that clinically reported AAS-associated ruptures predominantly affected young male weightlifters, with the pectoralis major being the most frequently involved tendon. These findings suggest that the observed association extends across different tendon sites and exposure settings, although the magnitude and mechanisms of risk cannot be considered equivalent between supraphysiological AAS abuse and medically prescribed testosterone.

The findings of Kanayama et al. provide the most clinically relevant comparison for evaluating the effects of long-term nonmedical AAS exposure.5 The markedly greater hazard of tendon rupture persisted despite the use of non-AAS-using bodybuilders as the comparator, thereby partly controlling for the mechanical demands associated with prolonged resistance training. The concentration of upper-body ruptures among AAS users was particularly notable, as no upper-body rupture was observed among nonusers. This pattern may reflect the disproportionate loading of the pectoralis, biceps, and triceps tendons during high-intensity bodybuilding exercises, combined with rapid gains in upper-body muscle strength. However, lower-body rupture did not differ significantly between groups, and the wide confidence interval indicates limited statistical precision. The study was also based partly on retrospective self-reported exposure and lifetime injury histories, leaving the possibility of recall and exposure-classification bias.

The two large administrative database studies broadened the potential clinical relevance of the findings by evaluating testosterone prescribed at therapeutic rather than bodybuilding doses. Albright et al. demonstrated a modest but statistically significant increase in Achilles tendon injury and subsequent surgery among testosterone recipients, whereas Meghani et al. identified a substantially greater increase in quadriceps injury during the first year following testosterone prescription.7,8 These findings suggest that tendon-related complications may not be restricted to extreme AAS regimens. Nevertheless, the database studies identified testosterone prescriptions rather than confirmed medication use, dosage, serum testosterone levels, or nonmedical supplementation. Their outcomes were based on billing codes that combined tendon rupture with less severe tendon or musculotendinous injuries. Therefore, these studies should not be interpreted as establishing that physiological testosterone replacement produces the same tendon effects as prolonged supraphysiological AAS abuse.

The predominance of pectoralis major rupture in the case-based literature is consistent with the mechanical conditions of bench pressing. During the lowering phase of the exercise, the pectoralis major undergoes forceful eccentric contraction while the shoulder is extended and externally rotated. The rupture cases frequently occurred while lifting very heavy loads, and the AAS-exposed patients in the Stefanou series lifted greater weights than their nonexposed counterparts.9 This finding supports a combined mechanism involving biological changes within the tendon and behavioral or performance-related exposure to progressively greater mechanical loads. The high frequency of pectoralis major rupture should not, however, be interpreted as proof that this tendon is uniquely susceptible to AAS, because unusual injuries among bodybuilders are more likely to be reported and published than more common tendon disorders.

The imaging and histopathological findings reported by Pochini et al. provide potential structural support for the association.10 Most evaluable specimens demonstrated myxoid degeneration or neovascularization, while contralateral MRI frequently identified tendinopathy despite the absence of rupture on that side. These observations suggest that acute failure may occur in a tendon already affected by bilateral or systemic degenerative changes. However, the study included only AAS-exposed rupture cases and uninjured nonexposed controls, making it

impossible to separate the effects of AAS from those of long-term high-load weightlifting. The inconsistency between the stated number of rupture patients and the reported contralateral MRI categories also reduces confidence in the precision of these findings.

Several biological mechanisms may explain the observed clinical association. In a human study, long-term resistance-trained AAS users had greater patellar tendon stiffness and elastic modulus than trained nonusers, while sustaining higher maximal tendon stress.11 These findings indicate that AAS exposure may modify normal tendon adaptation to chronic resistance training and reduce its capacity to deform under increasing loads. In experimental models, AAS administration combined with mechanical loading suppressed matrix metalloproteinase activity, promoted fibrosis, and interfered with Achilles tendon remodeling.12 Another animal study demonstrated that the combination of exercise and nandrolone produced the lowest tendon fracture stress and histological evidence of collagen dysplasia, effectively reversing the beneficial biomechanical adaptations associated with exercise alone.13 Together, these findings support a mechanism in which rapid muscle-strength gains and altered extracellular-matrix remodeling expose a relatively stiff or inadequately adapted tendon to forces that exceed its structural tolerance.

Nevertheless, the biological evidence is not entirely consistent. Pärssinen et al. found that supraphysiological AAS exposure increased markers of type III collagen synthesis and appeared to increase type I collagen synthesis while decreasing its degradation.14 These changes indicate altered collagen turnover but do not necessarily represent weaker tissue, as increased collagen production may reflect adaptation, fibrosis, or repair. Furthermore, electron microscopy of ruptured human tendons from two AAS users and two nonusers found no apparent differences in collagen-fibril ultrastructure.15 The small sample and assessment of already ruptured tissue limit that study, but its findings show that tendon rupture cannot yet be attributed to a single reproducible microscopic abnormality. Differences in steroid compounds, doses, exposure duration, training load, tendon type, and timing of tissue assessment may explain the conflicting results.

Most patients described in the case reports and case series achieved favorable functional recovery after operative treatment. Surgical repair restored near-normal strength, motion, and return to activity in patients with pectoralis major, quadriceps, and triceps rupture.9,10 However, these observations primarily represent young, highly motivated athletes who received surgical treatment and structured rehabilitation. They should not be interpreted as evidence that continued AAS use is safe during tendon healing. The absence of postoperative complications in small case series is insufficient to exclude impaired healing, rerupture risk, cardiovascular complications, or injury to another tendon. One death from myocardial infarction among the patients continuing AAS use further illustrates that tendon injury represents only one component of the broader health risks associated with these substances.9

The present findings have practical implications for clinicians treating athletes, bodybuilders, and patients receiving testosterone. A history of AAS or testosterone use should be considered during the assessment of acute pain, weakness, bruising, or deformity following resistance exercise, particularly when the pectoralis major, biceps, triceps, quadriceps, or

Achilles tendon is involved. Clinicians should also distinguish prescribed testosterone replacement from nonmedical multidrug AAS cycles, documenting the indication, dose, duration, concurrent compounds, and training pattern whenever possible. Patients using testosterone or AAS may benefit from counseling regarding gradual load progression, avoidance of abrupt increases in maximal lifting, recognition of prodromal tendon pain, and prompt evaluation of suspected rupture.

This review has several strengths. It integrated controlled comparative evidence with detailed clinical, imaging, and histopathological observations, allowing both risk and injury phenotype to be considered. The inclusion of case-based evidence was appropriate because several AAS-associated rupture patterns are rare and are unlikely to be adequately represented in conventional cohort studies. The review also distinguished nonmedical supraphysiological AAS use from prescribed testosterone exposure, which is essential for appropriate clinical interpretation.

Several limitations must also be acknowledged. Only eight studies were included, and most were case reports or small case series without an unexposed comparator. The two large database studies contributed nearly all participants but assessed prescribed testosterone and broadly coded tendon injuries rather than confirmed AAS-associated ruptures. Exposure was frequently based on self-report or prescription records, and detailed information regarding dose, cycle duration, adherence, serum concentration, concomitant substances, and cumulative exposure was generally unavailable. The athlete studies included exclusively male participants, limiting applicability to women. Publication bias was likely because severe or unusual ruptures are more likely to be reported than uncomplicated tendon disorders. Considerable heterogeneity in study design, exposure definition, tendon site, outcome assessment, and effect measures prevented quantitative pooling. Consequently, the available evidence supports an association but does not establish a precise causal effect or a dose-response relationship.

Future studies should use prospective designs with verified AAS exposure, clearly defined therapeutic and supraphysiological dose categories, and standardized confirmation of partial and complete tendon ruptures. Comparisons should account for resistance-training intensity, maximal lifting loads, age, metabolic comorbidities, corticosteroid exposure, and previous tendinopathy. Serial ultrasonography or MRI combined with biochemical markers and biomechanical testing may clarify whether structural abnormalities precede rupture and whether they improve after AAS discontinuation. Current evidence indicates that AAS exposure is associated with clinically important tendon injury, but the respective contributions of direct tendon toxicity, altered remodeling, rapid muscle hypertrophy, and extreme mechanical loading remain incompletely defined.

CONCLUSION

This systematic review indicated that exposure to anabolic-androgenic steroids (AAS) or exogenous testosterone was associated with an increased occurrence of tendon injuries and ruptures. Long-term AAS users demonstrated a ninefold higher hazard of first tendon rupture compared with nonusers, while prescribed testosterone was associated with increased odds of

Achilles and quadriceps tendon injuries and subsequent surgical repair. Case-based evidence showed that pectoralis major rupture was the predominant injury, particularly among young male weightlifters during bench-press exercises. Imaging and histopathological findings also revealed tendinopathy, degenerative changes, neovascularization, and tendon fissures, suggesting that acute rupture may occur on a background of structural deterioration. Although most surgically treated patients achieved favorable functional recovery, heterogeneity in study designs, exposure definitions, tendon locations, and outcome assessments limited causal interpretation and quantitative synthesis.

Future research should employ prospective, multicenter designs with larger and more diverse populations, including women and nonathletic testosterone users. Studies should clearly differentiate medically prescribed testosterone therapy from nonmedical supraphysiological AAS use and document dosage, duration, cumulative exposure, concurrent substance use, serum testosterone levels, and treatment adherence. Potential confounding factors, including training intensity, maximum lifting loads, age, metabolic disorders, corticosteroid exposure, and previous tendinopathy, should also be controlled. Serial ultrasonography or magnetic resonance imaging combined with biomechanical assessments and biochemical markers is recommended to determine whether tendon abnormalities precede rupture and whether they improve following treatment discontinuation. Standardized definitions of tendon injury and rupture are also needed to support future meta-analyses and clarify causal mechanisms, dose-response relationships, and evidence-based strategies for prevention, clinical monitoring, rehabilitation, and return to activity.

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