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Ruptured Tendons in Anabolic-Androgenic Steroid Users: A Cross-Sectional Cohort Study

Gen Kanayama, M.D., Ph.D.[*], **James DeLuca, B.A.**[†], **William P. Meehan III, M.D.**[‡], **James I. Hudson, M.D., Sc.D.**[*], **Stephanie Isaacs, B.S.**[†], **Aaron Baggish, M.D.**[†], **Rory Weiner, M.D.**[†], **Lyle Micheli, M.D.**[‡], and **Harrison G. Pope Jr., M.D.**[*]

[*]Biological Psychiatry Laboratory, McLean Hospital, Belmont, Massachusetts, and the Department of Psychiatry, Harvard Medical School, Boston, MA, USA

[†]Division of Cardiology, Massachusetts General Hospital, Boston, MA and Department of Medicine, Harvard Medical School, Boston, MA, USA

[‡]Division of Sports Medicine, Boston Children's Hospital Hospital, Boston, MA and Harvard Medical School, Boston, MA, USA

Abstract

Background—Accumulating case reports have described tendon rupture in men using anabolic-androgenic steroids (AAS). However no controlled study, to our knowledge, has assessed history of tendon rupture in a large cohort of AAS users and comparison nonusers.

Hypothesis—We hypothesized that men reporting long-term AAS abuse would report an elevated lifetime incidence of tendon rupture as compared to non-AAS-using bodybuilders.

Study Design—Cross-sectional cohort study.

Methods—We obtained medical histories from 142 experienced male bodybuilders age 35–55, recruited in the course of two studies. Of these men, 88 reported at least two years of cumulative lifetime AAS use and 54 reported no history of AAS use. In men reporting a history of tendon rupture, we recorded circumstances of the injury, prodromal symptoms, concomitant drug or alcohol use, and details of current and lifetime AAS use if applicable. We also obtained surgical records for most participants.

Results—Nineteen (22%) of the AAS users, but only 3 (6%) of the nonusers reported at least one lifetime tendon rupture. The hazard ratio (95% confidence interval) for a first ruptured tendon in AAS users versus nonusers was 9.0 (2.5, 32.3); $P < .001$. Several men reported two or more independent lifetime tendon ruptures. Interestingly, *upper body* tendon ruptures occurred exclusively in the AAS group (15 [17%] of the AAS users versus 0 non-users; risk difference 0.17 (0.09, 0.25); $P < 0.001$ [hazard ratio not estimable]), whereas we found no significant difference between users and nonusers in risk for *lower body* ruptures (6 [7%] AAS users, 3 [6%] nonusers; hazard ratio 3.1 (0.7, 13.8), $P = 0.13$). Of 31 individual tendon ruptures that we assessed, only 6 (19%) occurred while weightlifting, with the majority occurring during other sports activities.

Address correspondence to Harrison G. Pope Jr., M.D., McLean Hospital, Belmont, Massachusetts, 02478 USA. hpope@mclean.harvard.edu.

Eight (26%) ruptures followed prodromal symptoms of nonspecific pain in the region. Virtually all ruptures were treated surgically with complete or near-complete ultimate restoration of function.

Conclusions—AAS abusers, as compared to otherwise similar bodybuilders, showed a markedly increased risk of tendon ruptures, particularly upper body tendon rupture.

Clinical relevance—Tendon rupture represents a major adverse consequence of AAS abuse and a substantial public health problem.

Keywords

Anabolic-androgenic steroids; testosterone; weightlifting; tendon; orthopedics; bodybuilding; tendon rupture; men

Abuse of anabolic-androgenic steroids (AAS), among both competitive athletes and recreational bodybuilders, has grown into a major substance-abuse problem in the United States and most other Western countries.[45] Prior to about 1980, AAS use was largely confined to elite athletes, but over the last three decades the use of these drugs has become widespread in the general population.[18] One recent paper has calculated that between 2.9 and 4.0 million American men have used AAS at some time in their lives, and that perhaps 1 million of these men have developed AAS dependence, with chronic AAS use often persisting despite adverse medical and psychiatric effects.[44]

Among the various adverse effects of long-term AAS use is an apparently elevated risk of tendon rupture, as suggested by accumulating case reports over the last 30 years. In the world literature to date, we have found such 15 reports describing tendon rupture in acknowledged AAS users,[1, 2, 7–9, 11, 12, 15, 24, 27, 28, 33, 49, 51, 54] and three additional reports describing individuals where AAS use was not acknowledged, but appears likely.[3, 4, 48] As shown in Table 1 of the Supplemental Appendix (see below), all patients were male, ranging in age from their 20s to their 40s, and typically reporting long-term use of AAS for bodybuilding or other athletic activities. Although some men ruptured a tendon while weightlifting, many of the cases occurred as a result of a fall, sports injury, or from simply lifting an object. Many of the men ruptured two or more tendons, with some cases of bilateral simultaneous ruptures and other cases where different tendons were ruptured at different times over the course of months or years of AAS use. Collectively, these observations would seem to suggest that AAS use increases the risk for tendon rupture, and this impression has been widely shared in various reviews examining the adverse effects of AAS use.[26, 29, 40, 45]

However, we are not aware of any quantitative studies showing that tendon rupture is indeed more common in AAS users than in otherwise similar men with a comparable history of regular weightlifting. Also of note, we have been unable to find any reports of tendon rupture in AAS users prior to 1980. Thus one might argue that the association of AAS use and tendon rupture is coincidental, and that with the massively increased numbers of AAS users after 1980, one might see an overlap of AAS use and tendon rupture simply by chance. Perhaps in favor of this possibility, it should be noted that some of the putative cases of AAS-associated tendon rupture occurred in men who had also been treated with corticosteroids,[49, 51] and these drugs, rather than AAS, may have been responsible for

tendon pathology.[25] Arguing against this possibility, however, is the observation that many of the reported AAS-associated cases involved rupture of the triceps brachii and biceps brachii tendons. These are relatively rare injuries, and thus the accumulation of AAS-associated cases is hard to ascribe to coincidence.

To provide more quantitative data on the association of AAS use with tendon rupture, we assessed the risk of tendon rupture in 88 male long-term AAS users and 54 comparison men reporting a similar degree of weightlifting experience but no history of AAS use, all recruited in the course of two ongoing studies.

METHODS

Design of the parent studies

Participants for the present study were drawn from two current studies whose primary aims were to evaluate cardiac and other medical pathology in AAS users. In both studies, we recruited men age 35–55 who were experienced bodybuilders and who reported either a) at least two years of cumulative lifetime AAS use or b) no lifetime AAS use. Although neither of the two studies was focused exclusively on orthopedic injuries, we obtained a medical history with attention to musculoskeletal injuries in all participants, since such injuries are common among weightlifters. Our studies were focused on men because AAS use is rare in women,[19] and long-term AAS use is extremely rare in women,[44] despite some misconceptions to the contrary.[17, 45] We chose a two-year threshold for cumulative lifetime AAS use, based on findings of prior studies,[20, 42] in order to secure men with substantial AAS exposure and consequently greater risk for potential long-term AAS effects.

We recruited study participants by advertising in gymnasiums in the Boston, Massachusetts area, using recruitment techniques that we have described in detail previously.[20, 42] Briefly, our advertisement requested men who “could bench-press 275 pounds for at least one repetition, currently or in the past, for a psychiatric and medical evaluation.” As we have explained in our previous studies,[20, 42] the 275-pound threshold was simply a method to secure an unselected group of experienced weightlifters. Notably, we screened advertisement respondents by telephone and invited them for participation in the studies without asking about their history of AAS use or revealing our specific interest in AAS. By this method, we sought to minimize selection bias that might arise if participants were informed in advance about the exposure variable of interest.

Individuals qualifying on telephone screen for the studies were then scheduled for a screening interview where we administered 1) demographic questions; 2) questions about lifetime weightlifting and exercise history, including information about interruptions of regular exercise attribu to injury; 3) determination of height, weight, and body fat – the latter based on six skin caliper measurements, using the equations of Jackson and Pollock;[16] 4) medical history, including overall health, prior hospitalizations, and current prescription medications; 5) psychiatric and substance abuse history, using the Structured Clinical Interview for DSM-IV (SCID);[10] and 6) history of use of AAS and other performance-enhancing drugs, if any. In men acknowledging use of AAS, we elicited detailed information about the particular drugs used, together with an estimate of the individual’s total lifetime

dose of AAS in milligrams, calculated as milligrams of testosterone equivalent as described previously,[20, 42] and the total cumulative duration of time that the individual was actually using steroids during the course of his life. We also collected urine and hair samples from all participants. We tested urine samples for AAS, the performance-enhancing drug clenbuterol, and for opiates, amphetamines, cannabis, cocaine, and phencyclidine (Anti-Doping Research, Los Angeles, California), using methods previously described.[5, 6] We tested hair samples for opiates, cannabis, phencyclidine, amphetamines, and cocaine from the last 90 days (Psychemedics, Culver City, California). We excluded participants who displayed urine or hair results inconsistent with their self-reports of AAS or other substance use. We also calculated participants' fat-free mass index (FFMI), a measure of muscularity that we have previously presented,[23] based on our measurements of height, weight, and body fat. We excluded participants who denied having used AAS, but who showed an FFMI greater than 25.5 kg/m^2 while showing body fat of less than 10% – because we have previously shown that an FFMI beyond this threshold in a lean individual likely reflects surreptitious AAS use.[23]

Following the screening evaluation, participants were referred for a medical evaluation with a particular focus on cardiovascular function. In the course of these latter evaluations, participants received a second review of medical history from a study physician. We have reported preliminary findings from the first study;[46, 55] findings from the second study are still being analyzed.

The above procedures in both studies were approved by the institutional review boards of our institutions, and all participants provided written informed consent before study procedures were undertaken.

Participants and methods for the present study

We evaluated a total of 88 long-term AAS users and 54 non-AAS-using weightlifters in the course of the two parent studies between February, 2011 and November, 2014. The larger size of the AAS group was by design, since we wanted greater statistical power to examine subgroups within the AAS users. Over this interval, we noted a remarkable number of participants who reported a history of one or more tendon ruptures. Accordingly, we reviewed each participant's medical history, using both the medical history information obtained at screen and information from the second medical history obtained at the subsequent evaluation in each study. Among the 142 men, we found 23 who had reported at least one definite or possible tendon rupture. We recorded each participant's age and type of activity at the time of the injury, together with his total cumulative lifetime use of AAS (if any) as of that date. If applicable, we also recorded whether participants were currently using AAS at the moment of injury, and the drugs and doses that they were taking. We also recorded any other drugs, including prescription medications, over-the-counter medications, illicit drugs, and alcohol that the participant reported taking at the time. Finally, we recorded whether participants had experienced any prodromal symptoms, such as pain in the region of the tendon rupture, prior to the injury itself. We also successfully recontacted 22 of the 23 identified participants to request that they sign a release granting us permission to obtain copies of the surgical notes or other medical records describing the tendon repair.

Statistical analysis

For comparisons between AAS users and non-users on demographic variables, we used linear regression for continuous variables, and Fisher's exact test for categorical variables. To examine the association between AAS use and tendon rupture, we used a "cross-sectional cohort design." In a previous methodological paper, we are formally presented the properties of this design, including the conditions required for its validity.[13] Briefly, the cross-sectional cohort design samples a source population cross-sectionally, and then measures the association between exposures (in this case AAS use) and outcomes (in this case tendon rupture) assessed retrospectively.

As the primary measure of risk for tendon rupture, we estimated the hazard ratio for first tendon rupture, adjusted for self-defined race (modeled as White versus Non-White) in AAS users compared with non-users by a Cox proportional hazards model, with AAS as a time-varying exposure (i.e., unexposed until onset of AAS use, and exposed after onset of AAS use). Thus, the hazard ratio represents an estimate of the probability that a first tendon rupture will occur at any given age among individuals who have been exposed to AAS, divided by the probability that a first tendon rupture will occur at that same age among individuals who have not been exposed to AAS. We adjusted for race on the basis of evidence for racial differences in vulnerability to tendon rupture.[41] As a secondary measure of risk for tendon rupture, we estimated the mean total number of tendon ruptures in AAS users vs. nonusers, using linear regression, adjusted for race.

Alpha was set at 0.05, two-tailed. The analyses were performed using Stata 12.0 software (Stata Corp., College Station, TX).

RESULTS

The 88 long-term AAS users and 54 non-using weightlifters were closely matched in age (mean [SD] age 42.9 [5.2] years vs 43.2 [6.1] years; $P = 0.73$), as would be expected since they were chosen to be age 35–55, as described above. All of the men lifted weights for the purpose of bodybuilding, although only a minority of them had participated in actual bodybuilding competitions. None of the men had competed in powerlifting or in Olympic weightlifting. The two groups of men were similar in their reported lifetime years of weightlifting (mean [SD] for AAS users: 20.8 [6.6] years; non-users: 20.0 [9.5] years; $P = 0.61$), but they differed somewhat in racial distribution (AAS users: 82 White, 6 African-American; non-users: 41 White, 12 African-American, one Asian; $P = 0.005$ for White versus Nonwhite). Of the 23 men with definite or possible tendon rupture, one was excluded after further review because his surgical records showed an intact biceps tendon; his injury was a torn glenoid labrum. (Specifically, the surgical note stated, "the biceps tendon appeared completely within normal limits. The attachment of the biceps itself was carefully probed and noted to be normal; however at the 1:00 position, just anterior to the biceps insertion, there was detachment which appeared to be traumatic in nature of the labrum… This was repaired….") Of the remaining 22 men with definite tendon ruptures (Table 1), 19 were AAS users, representing 22% of the total AAS-using group of 88, and only 3 were non-using weightlifters, representing 6% of the 54 men in that group. None of the AAS users had ruptured a tendon prior to first using AAS.

The time-to-event distributions (survival curves) for first tendon rupture in the AAS users and non-users are presented in Figure 1. The hazard ratio (95% confidence interval) in the AAS users versus the non-users was 9.0 (2.5, 32.3); $P < .001$. The percentage of men with *lower body* tendon ruptures was 7% (6 cases) in the AAS group versus 6% (3 cases) in the non-user group, with a non-significant hazard ratio for first lower body tendon rupture of 3.1 (0.7, 13.8); $P = 0.13$. By contrast, *upper body* tendon ruptures were reported by 17% of the AAS users (15 individuals) versus 0% of the non-users. Since there were zero cases among the non-users, we could not estimate the hazard ratio for this comparison. Therefore, we instead estimated the risk difference, using an approach suggested by Spiegelman and Herzmark that employed a generalized linear regression model using the binomial distribution and the identity link function.[50] This approach yielded a risk difference of 0.17 (0.09, 0.25), $P < 0.001$.

As shown in Table 1, several men had ruptured two tendons and one had ruptured four tendons over the course of a lifetime. In all cases, these ruptures occurred on separate occasions, rather than simultaneously. The total number of tendon ruptures was 27 (19 upper body and 8 lower body) in the AAS group and 5 (all lower body) in the non-user group. The estimated adjusted mean difference between groups in number of lifetime tendon ruptures per person was 0.27 (0.06, .48), $P = 0.011$; for upper body ruptures alone, the difference was 0.21 (0.07, 0.35), $P = 0.004$; and for lower body ruptures alone it was 0.06 (−0.07, 0.18), $P = 0.38$.

Among the 32 separate tendon ruptures that occurred in the two groups collectively, only 6 occurred while weightlifting (representing 19% of the 31 cases for which history was obtained). Interestingly, several of these men recalled that they were lifting a light weight, just *after* a workout with much heavier weights, at the moment that the rupture occurred. A majority of the ruptures occurred during sports activities, with the remainder of cases attributed to falls or to moving or carrying heavy objects. Of the 14 biceps ruptures; 8 occurred at the distal end of the biceps (at the elbow), and 6 involved the long head of the biceps (at the shoulder). Of the 2 triceps ruptures, both occurred distally. Of the 31 ruptures for which history was obtained, 8 (26%) were preceded by pain in the vicinity of the site of the rupture, whereas the remainder occurred without warning. At the time of the rupture, two of the men were intoxicated with alcohol and two were using oxycodone (illicit in one case and medically prescribed in the other). One man had recently received corticosteroid injections for back pain, raising the possibility that in his case, corticosteroid exposure – a known risk factor for tendon rupture[22, 25, 53] – had contributed to his vulnerability to tendon rupture.

Almost all of the AAS users were taking a course of AAS at the time of the injury, usually at markedly supraphysiologic doses. As shown in Table 1, the users reported typical weekly doses between 210 mg and 2500 mg of total AAS per week – representing 3.5–40 times the normal male endogenous production of about 60 milligrams of testosterone per week. At the time of their tendon ruptures, many users had accumulated well over a decade of cumulative lifetime steroid use (i.e., total weeks during which they were actually using AAS), for a total lifetime dose of hundreds of thousands of milligrams.

Virtually all cases of tendon rupture were treated surgically, and all subjects reported complete or nearly complete return of function after completing rehabilitation from the surgical repair.

DISCUSSION

Over the last two decades, various case reports and small case series have described tendon rupture in men using anabolic-androgenic steroids (AAS) – suggesting that use of these drugs may render individuals particularly prone to this type of injury. However no study, to our knowledge, has systematically assessed history of tendon rupture in a large sample of AAS users and comparison weightlifters without AAS exposure. In the present study, we used a cross-sectional cohort study design to examine the risk of tendon rupture in 88 weightlifters who used AAS versus 54 who did not. The risk for first tendon rupture, as assessed by the hazard ratio, was 9.0 times greater in the AAS users compared with the non-users. In addition, the mean total number of lifetime tendon ruptures was markedly higher among the AAS users. Interestingly, the elevation in risk for lower-extremity tendon ruptures was much less than that for upper-extremity ruptures.

These findings have several implications for public health and injury prevention. First, they suggest that the risk for tendon rupture, particularly upper body tendon rupture, is much greater among AAS users than among otherwise similar men with comparable years of exposure to weightlifting. Thus our findings strongly support the impression that AAS use is indeed a risk factor for tendon rupture. By identifying those at risk for AAS use and offering counselling regarding the risk of tendon rupture as well as other adverse effects of AAS use, clinicians might reduce the use of AAS and subsequent tendon rupture among high risk populations.[29, 45]

The causes of AAS-associated tendon rupture are still incompletely understood. Two alternative (and not mutually exclusive) hypotheses should be considered. One possibility is that AAS use has little or no deleterious effect on tendons themselves, but merely causes massive hypertrophy of muscles without causing any corresponding strengthening of the associated tendons. Thus, the muscle may simply become too strong for its tendon, increasing the possibility of rupture in response to a sudden stress. Alternatively, it is possible that high doses of AAS, perhaps in conjunction with intense muscular exercise, may damage the structure of the tendons themselves, making them more vulnerable to rupture even in the absence of excessive stress. Evidence favoring the latter hypothesis comes from various animal studies, which have typically found that AAS exposure, usually in conjunction with exercise, led to collagen dysplasia, causing tendons to become stiffer and less flexible, with an increased crimp angle and earlier liability to failure.[14, 21, 30–32, 34–39, 52] However, one human study using electron microscopy found no evidence of collagen fibril ultrastructural abnormalities in the ruptured tendons of two AAS users as compared to two non-AAS-using controls.[9] Another recent study found no significant difference in maximal strain and toe limit strain in the patellar tendons of 8 long-term AAS users as compared to 8 experienced weightlifters reporting no AAS use, also arguing against the hypothesis of changes in collagen crimp pattern associated with AAS

use.[47] Thus the evidence for a direct toxic effect of AAS on human tendons remains somewhat inconclusive.

Although our study provides no direct evidence on the above issues, it is notable that we found a markedly increased risk of upper body tendon ruptures in AAS users versus non-using weightlifters, whereas the risk of lower body tendon ruptures was only modestly and non-significantly increased among AAS users. If indeed AAS use causes damage to human tendon architecture, one might predict that our AAS users, with histories of very extensive exposure to very high doses of AAS, would show a uniformly elevated prevalence of all types of tendon rupture throughout the body, rather than primarily an elevated risk of upper-body tendon ruptures. Therefore, our findings might be taken to weigh to at least some degree against a hypothesis of direct tendon damage, and might weigh more in favor of the simple theory that hypertrophied muscles can more easily break their tendons. In favor of the latter possibility, it is notable that AAS users are particularly prone to increase upper body musculature, leading to massively hypertrophied pectoralis, biceps brachii, and triceps brachii muscles, while often showing less of an effect on lower body musculature.[43] Thus, it is conceivable that muscle hypertrophy alone might account for our findings without postulating an additional mechanism of AAS-induced tendon damage.

Our findings are subject to several methodological limitations. First, because we deliberately selected men age 35–55 reporting at least two years of cumulative AAS exposure for our parent studies, our sample of men is not representative of the entire population of American AAS users. In particular, younger users, and those reporting briefer exposure to AAS, might well be less likely to have experienced a tendon rupture.

Second, we cannot exclude the possibility of unmeasured confounding variables, in that some other aspect of AAS users' lifestyle, such as concomitant use of other non-AAS drugs, might have contributed increased vulnerability to tendon rupture.

Third, our parent studies were not initially designed to elicit a detailed and exhaustive orthopedic history from study participants. Thus, it is possible that a study participant might have experienced a tendon rupture, but simply failed to mention this on his medical history, because he was not explicitly and systematically queried about orthopedic injuries. If such an omission occurred, however, it would have caused an underestimate of the risk of tendon rupture in this population, yielding overly conservative findings.

Fourth, our data were collected retrospectively, with some participants describing injuries that had occurred years earlier. Although we obtained surgical records to validate these injuries in most cases, our findings regarding details such as the circumstances of injury, concomitant drug use, and AAS use at the time of injury were limited by the accuracy of participants' retrospective reporting.

Fifth, there are two forms of selection bias that may have influenced our estimates of the hazard ratios for tendon rupture. One is that our selection of available AAS users and non-users might not have reflected the characteristics of long-term AAS users or of non-using weightlifters in the general population with respect to risk for tendon rupture. However, this form of selection bias seems unlikely to have greatly influenced the findings, since

participants were recruited without being informed of the specific exposure and outcome variables of interest (AAS use and tendon rupture), and it seems unlikely that participants would have chosen to enroll or not enroll in the study on the basis of these variables in any event. The other potential source of selection bias is the possibility of non-differential exiting from the underlying cohort (i.e., the theoretical cohort of individuals entering the source population during the period of time under retrospective observation), as explained in detail in our prior methodological publication presenting the cross-sectional cohort design.[13] However, this phenomenon seems unlikely in the present study, because even if the experience of a prior tendon rupture would influence a potential participant's availability to enroll in our study, there is little reason to believe that such an experience would have a *differential* influence in AAS users as compared to nonusers.

While acknowledging these limitations, our findings provide persuasive evidence that the risk of tendon rupture is strikingly higher among AAS users than among equally experienced weightlifters who have not used these drugs. Perhaps most notably, AAS users frequently reported a history of upper-body tendon rupture, but we found no cases of upper-body tendon rupture among 54 otherwise similar non-using weightlifters. These findings would suggest that if a muscular man presents with a ruptured tendon, perhaps especially an upper body tendon, the clinician should strongly suspect AAS use as a contributing factor. Given the high prevalence of AAS use and dependence in the United States and other Western countries, it appears that AAS-associated tendon rupture represents a significant public health problem, creating substantial costs in medical care, lost productivity, and reduced quality of life.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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What is known about the subject

A growing literature, consisting primarily of case reports, has suggested an association between AAS use and tendon rupture, but quantitative data on this phenomenon in human AAS users are as yet lacking.

What this study adds to existing knowledge

We present the first quantitative study, to our knowledge, examining the frequency and characteristics of tendon rupture in a large cohort of AAS users as compared to a group of age-matched experienced male bodybuilders who reported no AAS use.

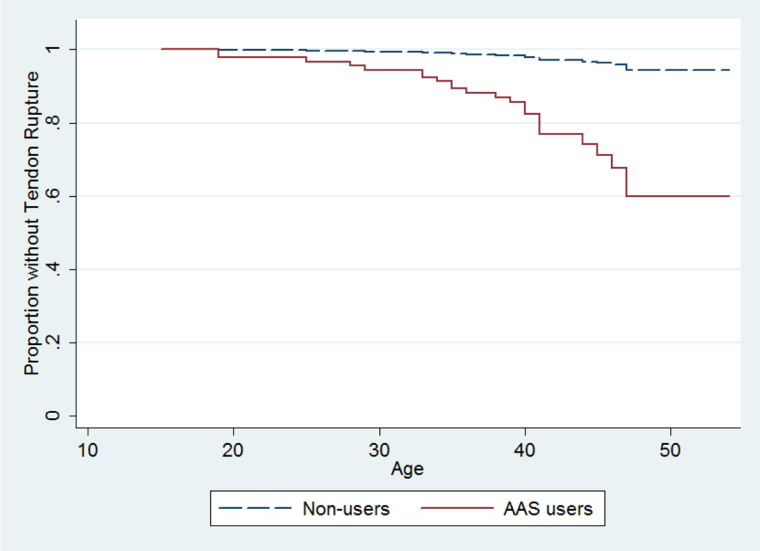

Figure 1.

Time to event distributions (survival curves) for first tendon rupture in AAS users and nonusers. The two survival curves indicate the percentage of men in the AAS and non-AAS groups, respectively (shown on the Y axis), who had reached a given age (shown on the X axis) without having yet experienced a tendon rupture.

Table 1

Features of tendon rupture in AAS users and comparison weightlifters

	AAS users											
Participant	**Race**	**Age at time of evaluation**	**Tendon ruptured[a]**	**Age at time of rupture**	**Activity at time of rupture**	**Drugs other than AAS at time of rupture**	**Prodromal syx prior to rupture?**	**Age at first AAS use**	**Lifetime yrs of AAS use at time of rupture**	**Average weekly dose of AAS, mg**	**Lifetime AAS dose at time of rupture, mg**	**On AAS at time of rupture?**
1	W	35	Pectoralis	29	Wrestling	Alcohol	Yes	20	9.0	1600	750000	Yes
2	W	35	Biceps	34	Bowling	Ibuprofen	Yes	20	11.9	800	496000	Yes
3	W	35	Pectoralis	28	Unknown	Unknown	Unknown	19	3.8	800	160000	Unknown
4	W	36	Quadriceps	25	Weightlifting	No	No	17	3.8	1000	200000	Yes
			Biceps	29	Weightlifting	No	Yes		4.6	1000	240000	Yes
5	W	37	Biceps	35	Moving heavy object	No	No	18	10.4	1000	540000	Yes
6	AA	41	Biceps	35	Football	None	No	19	9.6	800	400000	Yes
7	W	41	Biceps	36	Moving heavy object	Ibuprofen	No	23	7.2	1000	375000	No
			Triceps	36	Fall	Ibuprofen	No		7.2	1000	375000	No
8	W	42	R Patellar	33	Fall	None	No	18	9.8	800	408000	Yes
			L Patellar	34	Martial arts fighting	None	No		10.5	800	436000	Yes
9	W	43	Biceps	41	Moving heavy object	Oxycodone	Yes	30	10.0	900	468000	Yes
10	W	43	R Biceps	38	Football	None	No	17	15.8	600	490000	Yes
			L Biceps	43	Batting in softball	None	No		19.2	600	600000	Yes
11	AA	44	Achilles	33	Basketball	None	No	26	3.0	900	138000	Yes
12	W	46	Biceps	40	Moving heavy object	None	No	21	9.5	2000	988000	Yes
			Pectoralis	44	Weightlifting	None	No		11.5	2000	1200000	No
13	W	46	Biceps	46	Martial arts sparring	Escitalopram	No	21	15.4	1500	1200000	No
14	W	47	Biceps	44	Moving heavy object	None	Yes	42	0.1	400	1600	Yes
15	W	48	Triceps	47	Weightlifting	Corticosteroids	No	28	9.2	1000	480000	Yes
16	W	49	Patellar	40	Jumping rope	Oxycodone	No	21	6.0	600	187200	Yes
17	W	49	Quadriceps	41	Fall	None	No	35	4.8	2500	625000	Yes
18	W	51	Biceps	45	Weightlifting	Methylphenidate	no	42	0.9	210	10000	No
19	W	51	L Biceps	41	Volleyball	Alcohol	Yes	21	10.0	600	312000	Yes
			R Biceps	48	Weightlifting	Alcohol	No		12.7	600	396000	Yes

Participant	Race	Age at time of evaluation	Tendon ruptured[a]	Age at time of rupture	Activity at time of rupture	Drugs other than AAS at time of rupture	Prodromal syx prior to rupture?	Age at first AAS use	Lifetime yrs of AAS use at time of rupture	Average weekly dose of AAS, mg	Lifetime AAS dose at time of rupture, mg	On AAS at time of rupture?
AAS users												
			R Achilles	48	Carrying girl on shoulders	Alcohol	No		13.1	600	410000	Yes
			L Achilles	51	Jumping off stair	None	no		14.4	600	450000	Yes
AAS non-users												
20	AA	49	Achilles	42	Basketball	None	No	.	.	.	.	.
	AA		Patellar	39	Basketball	None	No	.	.	.	.	.
21	W	50	Hamstring	47	Hockey	None	No	.	.	.	.	.
22	AA	50	Patellar	19	Jumping	None	Yes	.	.	.	.	.
	AA		Patellar	32	Jumping	None	Yes	.	.	.	.	.

[a] In all cases, “pectoralis” = pectoralis major, “biceps” = biceps brachii, and “triceps” = triceps brachii.