

Prevalence of Achilles and patellar tendinopathy and their association to intratendinous changes in adolescent athletes

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Achilles (AT) and patellar tendons (PT) are commonly affected by tendinopathy in adult athletes but prevalence of symptoms and morphological changes in adolescents is unclear. The study aimed to determine prevalence of tendinopathy and intratendinous changes in ATs and PTs of adolescent athletes. A total of 760 adolescent athletes (13.0 ± 1.9 years; 160 ± 13 cm; 50 ± 14 kg) were examined. History, local clinical examination, and longitudinal Doppler ultrasound analysis for both ATs and PTs were performed including identification of intratendinous echoic changes and vascularization. Diagnosis of tendinopathy was complied clinically in case of positive history of tendon pain and tendon pain on palpation. Achilles tendinopathy was diagnosed in 1.8% and

patellar tendinopathy in 5.8%. Vascularizations were visible in 3.0% of ATs and 11.4% of PTs, hypoechogenicities in 0.7% and 3.2% as well as hyperechogenicities in 0% and 0.3%, respectively. Vascularizations and hypoechogenicities were statistically significantly more often in males than in females ($P \leq 0.02$). Subjects with patellar tendinopathy had higher prevalence of structural intratendinous changes than those without PT symptoms ($P \leq 0.001$). In adolescent athletes, patellar tendinopathy is three times more frequent compared with Achilles tendinopathy. Longitudinal studies are necessary to investigate physiological or pathological origin of vascularizations and its predictive value in development of tendinopathy.

Alterations of the tendon structure, such as hypo- or hyperechoic regions and vascularizations, are often visible using (Doppler) ultrasound techniques in patients suffering from tendinopathy (Ohberg et al., 2001; Scott et al., 2013). Tendinopathy is defined as a clinical presentation of pain leading to a decreased functional ability and may be accompanied by the presence of symptoms like swelling or thickening (Cook & Purdam, 2003; Maffulli et al., 2003, 2004; Mayer et al., 2007). Tendinopathic pathology characteristically arises in athletes exposed to high tendon loading (Maffulli et al., 2003). In most of the cases, Achilles (AT) and patellar tendons (PT) are affected, typically seen in long-distance runners and athletes of repetitive jumping disciplines. In adult athletes, a point prevalence of up to 36% has been reported for Achilles tendinopathy in runners and up to 45% for patella tendinopathy in volleyball players (Lian et al., 2005; Hirschmuller et al., 2010).

The prevalence of Achilles and patellar tendinopathy in adolescent athletes is not well examined. In 268 patellar tendons of 14- to 18-year-old volleyball players, a prevalence of 7.3% concerning patellar tendinopathy has

been reported (Cook et al., 2000). In 891 nonelite young athletes with an average age of 24 years out of seven different high-impact sport disciplines, prevalence varied between 2.5% in soccer players and 14.4% in volleyball players (Zwerver et al., 2011). Data regarding Achilles tendinopathy in adolescents is sparse. By use of questionnaires, in high school runners age 13–18 years, lifetime prevalence in the development of Achilles tendinopathy with approximately 7.5% has been reported (Tenforde et al., 2011).

Regarding intratendinous changes in adult athletes, the prevalence of hypoechoic changes has been described to be as high as 70% in PT of male basketball players and 35% in AT of male gymnasts (Cook et al., 2000; Emerson et al., 2010). In adolescent athletes, hypoechogenicities have been reported to be present in approximately 30% of basketball players (Cook et al., 2000). High rates of structural tendon changes and vascularization in PT were also seen in young volleyball players suffering from patella tendinopathy (Gisslen et al., 2005; Gisslen et al., 2007). To our knowledge, further data on prevalence of intratendinous changes in

adolescent athletes are not available, neither for AT nor for PT. Moreover, the origin of intratendinous vessels in tendons is not completely understood. It is still unclear whether its presence is of pathological value representing degenerated tissue caused by vasculo-neural ingrowth responsible for pain in chronic tendinopathy (Ohberg et al., 2001; Alfredson et al., 2003; Rees et al., 2013). In contrast, it could also be interpreted as a physiological adaptation to external load as at least low-grade vascularization has been shown to be influenced by loading in adults (Boesen et al., 2006b, 2012). However, vascularization status in healthy AT and PT of adolescent athletes has not been examined yet.

Clinical diagnosis of jumper's knee in junior elite volleyball, soccer, and basketball players have been shown to be probably associated to vascularizations and echoic tendon changes (Fredberg & Bolvig, 2002; Gisslen & Alfredson, 2005; Gisslen et al., 2005). Furthermore, vascularization alone or vascularization accompanied by structural tendon changes may indicate a risk for developing jumper's knee or Achilles tendinopathy in asymptomatic volleyball and soccer players as well as long-distance runners (Fredberg & Bolvig, 2002; Gisslen & Alfredson, 2005; Gisslen et al., 2005; Hirschmuller et al., 2012). Nevertheless, clinical relevance and predictive value in the development of tendinopathy of these structural alterations is still a matter of debate. As described above, several studies showed an association between intratendinous alterations and the development tendinopathy (Gisslen & Alfredson, 2005; Gisslen et al., 2005; Shaikh et al., 2012; Comin et al., 2013; Kulig et al., 2013). In contrast, ultrasound investigations on badminton players presented no correlations of vascularization with clinical symptoms (26). Additionally, all of the examined ATs presented intratendinous flow after training (Boesen et al., 2006a). Furthermore, intratendinous vessels at the beginning of the season did not predict for symptoms in the follow-up (Boesen et al., 2012). Thus, it is still unclear, whether the presence of intratendinous vessels can be considered a physiological adaptation to loading or has to be interpreted pathologically (van Snellenberg et al., 2007; Boesen et al., 2012). However, prevalence data of vascularizations in AT and PT of adolescent athletes are currently not available.

The aim of the study was to evaluate the prevalence of clinical symptoms and morphological changes in ATs and PTs of adolescent athletes. Furthermore, the association of vascularizations and echoic altered tendons to clinical symptoms should be analyzed.

Methods

Subjects

The study examined adolescent athletes within a preparticipation examination or an annual health evaluation before and after entrance to an elite sport school. In total, 760 adolescent athletes (442 males, 318 females; 13.0 ± 1.9 years; 160 ± 13 cm;

50 ± 14 kg) out of 16 different sport disciplines took part in the study. Anthropometry and training history data are shown in Table 1. A list of the different sport disciplines is given in Table 2. Exclusion criteria were diagnosed rheumatic disease, a lipid metabolic disorder or a recent injury of knee or ankle. Parents of all adolescent athletes signed written informed consent before inclusion. The ethics committee of the local university approved the study.

Measurement procedure

All preparticipation and annual health examinations took place at the local university outpatient clinic, responsible for athletes' medical care (Mayer et al., 2012). Within each examination, a short history of pain, injuries, and complaints of both ATs and PTs and a local tendon examination including the adjacent joints were performed. Anamnesis included anthropometric (age, weight, and height) and training data (sport type and discipline, training hours a week and total training years). Diagnosis of tendinopathy was complied clinically in case of positive anamnesis of tendon pain and tendon pain on palpation according to its highest accuracy in diagnosing tendinopathy (Hutchison et al., 2013). Maturity diseases of bony structure (e.g., Osgood Schlatter disease, apophysitis calcanei) were verified by radiologic imaging techniques if indicated.

The ultrasound analysis was done using two different high-resolution ultrasonographs (Xario SSA-660A and Viamo SSA-640A; Toshiba, Tokyo, Japan) with a 7.5 MHz (PLT-704SY at 11 MHz; Viamo) and a 12 MHz (PLT-1204AT at 14 MHz; Xario) multi-frequency linear transducer. For all measurements, standardized transducer settings (Viamo: gain = 93, DR = 50, penetration depth = 3 cm, focus at 0.5 cm; Xario: gain 80, DR = 65 penetration depth = 3 cm, focus at 0.5 cm) were used and the transducer was placed strictly parallel and orthogonal to the fiber direction. The examinations of the Achilles tendons were investigated in a prone position with the feet hanging over the edge of the examination table with ankles passively flexed at 90° while the examination of patellar tendons were carried out in a supine position with 30° knee flexion. The thickness of both Achilles and Patella tendons was measured by use of longitudinal scans at a reference point of 2 cm proximal to the calcaneal insertion and 2 cm distal of the inferior patella pole (Cassel et al., 2012). Furthermore, the thickest part of the Achilles tendons "midportion area" was examined in a second longitudinal scan. Additionally, structural irregularities including hypo- and hyperechoic regions were recorded and verified in transversal scans. Finally, all tendons were investigated for intratendinous microvessels using power Doppler ultrasonography (advanced dynamic flow, ADF; Fig. 1) with standardized power Doppler settings (Xario: ADF frequency = 10 MHz, pulse repetition frequency = 12.2 kHz, color velocity = 1.9 cm/s; Viamo: ADF frequency = 4.5 MHz, pulse repetition frequency = 0.5 kHz, color velocity = 4.39 cm/s, color intensity just below the artifact threshold, size of the color box (region of interest, ROI) = 3 cm^2 (Ohberg & Alfredson, 2002; Mayer et al., 2007). The pressure on the probe was reduced to a minimum in order to avoid compression of small vessels (11). Vascularization within the ROI was graded according to a modification of the "Ohberg score" from 0 to 5 (0 = no vessels, 1 = 1–2 vessels, 2 = 3–5 vessels, 3 = vessels in up to 30%, 4 = vessels in 30–50% and 5 = vessels in > 50% within the ROI; Ohberg & Alfredson, 2002; Hirschmuller et al., 2010; Boesen et al., 2012). For all Doppler ultrasound measurements, subjects were requested to relax muscles as presented by Boesen et al. (2012) leading to an individual plantarflexion angle of approximately 70° . The position of the knee angle stayed at 30° flexion. Because of a reported increased intensity of intratendinous flow after exercise examinations were performed at least 2 h after training activities (Malliaras et al., 2008; Hirschmuller et al., 2010). Four trained examiners

Table 1. Anthropometric and training data and tendon diameters (mean of both sides) of males (M) and females (F) and the four age groups

Groups	<i>n</i>	Age (years)*	Height (cm)*	Weight (kg)*	Training (h/week)*	Training years*	AT 2 cm (mm)*	AT max (mm)*	PT 2 cm (mm)*
Total	760	13.0 ± 1.9	160 ± 13	50 ± 14	8.1 ± 5.5	4.1 ± 2.1	5.0 ± 0.6	5.4 ± 0.7	3.5 ± 0.5
M	442	13.0 ± 1.9	161 ± 14	51 ± 15	8.0 ± 5.2	4.3 ± 2.0 [†]	5.1 ± 0.6 [†]	5.6 ± 0.7 [†]	3.7 ± 0.5 [†]
F	318	12.9 ± 1.8	160 ± 10	49 ± 12	8.1 ± 5.8	3.9 ± 2.1	4.8 ± 0.6	5.2 ± 0.7	3.4 ± 0.5
< 11	34	9.9 ± 0.9	142 ± 13	34 ± 10	7.0 ± 4.6	2.9 ± 1.9	4.7 ± 0.7	5.0 ± 0.7	3.2 ± 0.6
M	22	9.7 ± 0.9	138 ± 10	32 ± 9	7.1 ± 3.6	2.8 ± 1.7	4.6 ± 0.7	4.9 ± 0.6	3.1 ± 0.5
F	12	10.1 ± 1.0	149 ± 15 [†]	39 ± 12	7.0 ± 6.3	2.9 ± 2.3	4.9 ± 0.6	5.1 ± 0.8	3.4 ± 0.6
11–12	466	11.9 ± 0.4	155 ± 9	44 ± 9	6.0 ± 3.9	3.8 ± 2.1	5.1 ± 0.6	5.5 ± 0.7	3.5 ± 0.5
M	264	11.9 ± 0.5	155 ± 9	44 ± 9	5.8 ± 3.5	4.0 ± 2.1 [†]	5.2 ± 0.6 [†]	5.6 ± 0.7 [†]	3.6 ± 0.5 [†]
F	202	11.9 ± 0.4	156 ± 8 [†]	45 ± 9	6.2 ± 4.4	3.5 ± 2.0	4.9 ± 0.6	5.3 ± 0.7	3.4 ± 0.4
13–14	102	14.1 ± 0.6 [†]	167 ± 10	57 ± 11	9.1 ± 5.6	4.4 ± 2.0	5.1 ± 0.7	5.5 ± 0.8	3.7 ± 0.6
M	61	14.1 ± 0.7 [†]	168 ± 10	58 ± 12	9.1 ± 5.3	4.4 ± 1.9	5.3 ± 0.7 [†]	5.8 ± 0.7 [†]	3.9 ± 0.5 [†]
F	41	14.0 ± 0.6	165 ± 8	56 ± 11	9.1 ± 6.1	4.4 ± 2.2	4.7 ± 0.6	5.1 ± 0.6	3.4 ± 0.5
15–17	158	16.0 ± 0.8	174 ± 9	67 ± 11	13.8 ± 5.3	5.2 ± 1.6	4.9 ± 0.5	5.2 ± 0.6	3.7 ± 0.5
M	95	16.0 ± 0.8	178 ± 8 [†]	70 ± 12 [†]	13.7 ± 5.0	5.3 ± 1.5	5.0 ± 0.6 [†]	5.5 ± 0.7 [†]	3.8 ± 0.5 [†]
F	63	16.0 ± 0.8	169 ± 8	61 ± 8	14.1 ± 5.7	4.9 ± 1.7	4.7 ± 0.5	4.9 ± 0.5	3.5 ± 0.5

*Represents statistically significant differences of parameters between the age groups. Anthropometric data: $P \leq 0.001$ for age, height, and weight for each comparison. Training hours/week: $P < 0.001$ for group 15–17 years against all other age groups and for group 11–12 years to 13–14 years. Training years: $P \leq 0.001$ for group 15–17 years against < 11 years and 11–12 years and group 13–14 years against < 11 years; $P = 0.04$ for group 15–17 years against 13–14 years and group 13–14 years to 11–12 years. AT 2 cm: $P \leq 0.034$ for groups 11–12 years and 13–14 years thicker than < 11; $P = 0.012$ for group 15–17 years thicker than 11–12 years. AT max: $P \leq 0.002$ for groups 11–12 years and 13–14 years thicker than < 11 years and group 15–17 years against 11–12 years ($P < 0.001$) and 13–14 years ($P = 0.01$). PT 2 cm: $P < 0.001$ for groups 15–17 years and 13–14 years thicker than < 11; $P = 0.020$ for group 11–12 years thicker than < 11 years; group 15–17 years ($P = 0.013$) and 13–14 years ($P = 0.003$) thicker than 11–12 years.

[†]Represents statistically significant differences between the genders. Within the whole cohort: males had more training years ($P = 0.012$) and larger tendon diameters (AT 2 cm, AT max, and PT 2 cm; $P < 0.001$). Within different age groups: within group < 11 years, females were taller ($P = 0.04$); in group 11–12 years, higher height (0.03), more training years ($P = 0.02$), and higher tendon diameters (AT 2 cm, AT max, and PT 2 cm) in males ($P < 0.001$); in group 13–14 years, higher tendon diameters (AT 2 cm, AT max, and PT 2 cm) in males ($P < 0.001$); in group 15–17 years, higher anthropometric (height and weight) and tendon data (AT 2 cm, AT max, and PT 2 cm) in males ($P \leq 0.002$).

AT, Achilles tendon; PT, patellar tendon.

Table 2. Number of athletes and their distribution in 16 different sport disciplines

Sport discipline	<i>n</i>	%
Boxing	25	3.1
Canoeing	51	6.4
Cycling	55	6.8
Gymnastics	37	4.6
Handball	88	11.0
Horse riding	21	2.6
Judo	62	7.7
Modern pentathlon	17	2.1
Recreational	42	5.2
Rowing	43	5.4
Shooting	23	2.9
Soccer	89	11.1
Swimming	38	4.7
Track and field	99	12.3
Volleyball	16	2.0
Weight bearing	23	2.9
Wrestling	74	9.2

with experience in Doppler ultrasound examinations performed all investigations.

Data categorization and analysis

All subjects were categorized into four age groups: group 1: < 11 years; group 2: 11–12 years; group 3: 13–14 years; group 4: 15–17 years (Table 1). Prevalence of diagnosis and sonographically detected morphological tendon changes (vascularizations,

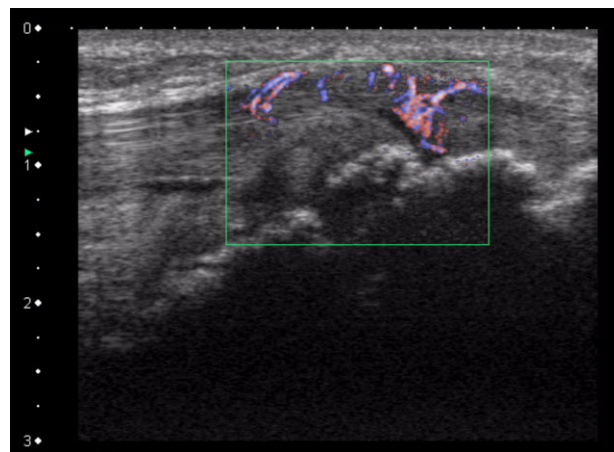


Fig. 1. Vascularization grade 3 in the right distal patellar tendon (close to tibial tuberosity) of a 13-year-old handball player with chronic patellar tendinopathy and Osgood–Schlatter disease.

hypo- and hyperechogenicities) was analyzed descriptively by gender, age groups, and tendinopathic state. Tendon thickness values were presented by building means of the both sides of each thickness parameter. Group differences for interval scaled data were analyzed by the use of unpaired *t*-test and one-way analysis of variance (ANOVA; post-hoc Tukey–Kramer test) or by use of Kruskal–Wallis ANOVA (post-hoc Wilcoxon test) after testing for normal distribution by Kolmogorov–Smirnov test. Nominal and ordinal scaled data were analyzed by Fisher's exact test. The overall level of significance was set $\alpha = 0.05$ [Jmp 9.0 (SAS

Institute, Cary, North Carolina, USA); SPSS (IBM Corporation, Armonk, New York, USA)].

Results

In total, 1516 ATs and 1512 PTs out of 760 adolescent athletes were investigated. Baseline data of subjects' anthropometric, training, and tendon data are given in Tables 1 and 2. Anthropometric and training data increased up to highest values in subjects of the oldest age group ($P < 0.001$). While there were no overall gender differences visible, females of the youngest ($P < 0.04$) and males of the oldest age group ($P < 0.001$) were significantly taller and heavier compared with their counterparts (males < 11 years and females 15–17 years; Table 1). Tendon thickness values overall were higher in males than in females, for both AT parameters and PT diameters ($P < 0.001$). Within the age groups 11–12 years, 13–14 years, and 15–17 years males presented higher tendon thickness for both AT and PT parameters ($P \leq 0.002$), while females of the youngest age group showed thicker PTs ($P = 0.04$).

In total, 7.8% of the investigated adolescents presented a diagnosis of pain in AT or PT midportion and/or their corresponding bony insertion zones (Table 3). Mostly, patellar tendinopathy was present (5.8%), in some cases accompanied by Osgood Schlatter disease. Achilles tendinopathy was diagnosed in 1.8% of subjects. Prevalence of diagnosis did not differ between genders ($P > 0.05$). Highest prevalence for Achilles tendinopathy was seen in group 13–14 years (4.9%), patellar tendinopathy was most often seen in group 15–17 years (11.4%; $P < 0.001$; Table 3).

Regarding intratendinous changes, vascularizations (classified as grade 1 or more) were visible in 7.2%, hypoechogenicities in 1.9% and hyperechogenicities in

0.2% of all tendons (Table 4). Altogether, ATs presented vascularizations in 3.0%, hypoechogenicities in 0.7% and no hyperechogenicities (0.0%). Within PTs, vascularizations were seen in 11.4%, hypoechogenicities in 3.2%, and hyperechogenicities in 0.3%. Vascularizations in ATs and PTs as well as hypoechogenicities in PTs were significantly more often in males than in females ($P < 0.05$). For vascularizations and hypoechogenicities in PTs, statistically significant differences between the age groups were present ($P = 0.02$; $P = 0.003$; Table 4).

Subjects having Achilles tendinopathy showed higher maximum tendon thickness (5.8 ± 0.9 mm) than those without AT symptoms (5.4 ± 0.8 mm; $P = 0.026$; Table 5). No statistically significant differences were seen regarding anthropometrical and training data as well as the other tendon parameters. In contrast, subjects with patellar tendinopathy were older, taller, and heavier ($P \leq 0.002$), and reported higher amount of training hours per week ($P = 0.02$) compared with athletes without patellar tendinopathy. Furthermore, higher PT thickness was present in patients (3.8 ± 0.7 mm vs 3.5 ± 0.5 mm; $P = 0.01$). Furthermore, a statistically significant higher prevalence of structural intratendinous changes (vascularizations, hypo- and hyperechogenicities) was detected in subjects with patellar tendinopathy compared with athletes without patellar tendinopathy ($P \leq 0.001$; Table 5).

Discussion

The present study aimed to determine the prevalence of clinical symptoms and morphological changes in ATs and PTs of adolescent athletes. Furthermore, the association of vascularizations and echoic changed tendons to clinical symptoms was evaluated.

Table 3. Distribution of athletes' diagnoses in the region of Achilles and patellar tendons

	Total		Total diagnoses*		Achilles tendinopathy		Patellar tendinopathy*		Apophysitis calcanei		Schlatter disease		Sinding–Larsen disease		St. p. partial Achilles tendon rupture	
	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%
Total	760		59	7.8	14 [†]	1.8	42 [‡]	5.6	1	0.1	14 [§]	1.8	1	0.1	1	0.1
Males	442 [¶]		39	8.8	9	2.0	27	6.1	1	0.2	10	2.3	1	0.2	1	0.2
Females	318 ^{**}		20	6.3	5	1.6	15	4.8	0	0	4	1.3	0	0	0	0
< 11	34		3	8.8	0	0	2	5.9	0	0	0	0	0	0	0	0
11–12	466 ^{††}		24	5.2	8	1.7	14	3.0	1	0.2	6	1.3	0	0	0	0
13–14	102 ^{‡‡}		13	12.7	5	4.9	8	8.0	0	0	4	3.9	1	1.0	1	1.0
15–17	158		19	12.0	1	0.6	18	11.4	0	0	4	2.5	0	0	0	0

*Represents statistically significant age group differences in prevalence total diagnoses ($P = 0.005$) and patella tendinopathy ($P = 0.001$).

[†]Achilles tendinopathy in 10 subjects on both sides.

[‡]Patellar tendinopathy in nine subjects on both sides.

[§]Schlatter disease in seven times accompanied by patellar tendinopathy and in eight patients on both sides.

[¶]In one male subject, Achilles tendon, and in one male subject, patellar tendon data are missing.

^{**}In one female subject, Achilles tendon data, and in three female subjects, patellar tendon data are missing.

^{††}In two subjects, Achilles tendon data, and in two subjects, patellar tendon data are missing.

^{‡‡}In two subjects, patellar tendon data are missing.

Table 4. Sonographic intratendinous changes in whole group, by gender and age groups

Groups	Tendons			Vascularizations						Hypoechoogenicities						Hyperechoogenicities						
	Total	ATs	PTs	Total		ATs*		PTs**		Total		ATs		PTs**		Total		ATs		PTs		
	<i>n</i>	<i>n</i>	<i>n</i>	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	
Total	760	3028	1516	1512	218	7.2	45	3.0	173	11.4	59	1.9	10	0.7	49	3.2	5	0.2	0	0	5	0.3
Males	442 [‡]	1764	882	882	149	8.4	33	3.7	116	13.2	46	2.6	8	0.9	38	4.3	2	0.1	0	0	2	0.2
Females	318 [§]	1264	634	630	69	5.4	12	1.9	57	9.0	13	1.0	2	0.3	11	1.7	3	0.2	0	0	3	0.5
< 11	34	136	68	68	9	6.6	3	4.4	6	8.8	5	3.7	1	1.5	4	5.9	0	0	0	0	0	0
11–12	466 [¶]	1856	928	928	130	7.0	27	2.9	103	11.1	23	1.2	5	0.5	18	1.9	2	0.1	0	0	2	0.2
13–14	102 ^{**}	404	204	200	46	11.4	10	4.9	36	18.0	12	3.0	2	1.0	10	5.0	1	0.2	0	0	1	0.5
15–17	158	632	316	316	33	5.2	5	1.6	28	8.9	19	3.0	2	0.6	17	5.4	2	0.3	0	0	2	0.6

*Represents statistically significant differences between the genders for vascularizations in ATs ($P = 0.045$) and PTs ($P = 0.01$) and hypoechoogenicities in PTs ($P = 0.005$).

†Represents statistically significant age group differences for vascularizations ($P = 0.017$) and hypoechoogenicities ($P = 0.003$) in PTs.

‡In one male subject, Achilles tendon, and in one male subject, patellar tendon data are missing.

§In one female subject, Achilles tendon data, and in three female subjects, patellar tendon data are missing.

¶In two subjects, Achilles tendon data, and in two subjects, patellar tendon data are missing.

**In two subjects, patellar tendon data are missing.

AT, Achilles tendon; PT, patellar tendon.

Table 5. Anthropometric and training data, tendon thickness, and morphology parameters in relation to Achilles and patellar tendinopathy or absence of symptoms

	Achilles tendinopathy*	No Achilles tendinopathy	Patellar tendinopathy*	No patellar tendinopathy
Subjects (<i>n</i>)	14	744 [‡]	42	714 [‡]
Age (years)	13.1 ± 1.4	13.0 ± 1.9	14.2 ± 2.1	12.9 ± 1.8
Height (cm)	159 ± 10	160 ± 13	166 ± 13	160 ± 13
Weight (kg)	47 ± 11	50 ± 14	59 ± 18	50 ± 14
Training (h/week)	9.7 ± 7.4	8.1 ± 5.4	10.2 ± 6.6	7.9 ± 5.4
Training years	4.7 ± 1.9	4.1 ± 2.1	4.4 ± 1.9	4.1 ± 2.1
Tendons (<i>n</i>)	23	1493	55	1457
Thickness parameters				
AT 2 cm point (mm)	5.1 ± 0.8	5.0 ± 0.6	5.1 ± 0.7	5.0 ± 0.7
AT max thickness (mm)	5.8 ± 0.9	5.4 ± 0.8	5.5 ± 0.8	5.4 ± 0.8
PT 2 cm point (mm)	3.5 ± 0.6	3.6 ± 0.6	3.8 ± 0.7	3.5 ± 0.5
Morphology parameters [†]				
No hypoechoogenicities	23	1483	39	1424
Hypoechoogenicities I [‡]	0	10	12	27
Hypoechoogenicities II [‡]	0	0	4	6
Hyperechoogenicities	0	0	3	2
No vessels	22	1449	39	1300
Vascularization I [‡]	1	41	11	142
Vascularization II [‡]	0	3	2	10
Vascularization III [‡]	0	0	2	5
Vascularization IV [‡]	0	0	1	0

*Represents statistically significant difference of parameters subjects with tendinopathy compared with the asymptomatic group. Tendons with Achilles tendinopathy had higher maximum AT thickness than corresponding asymptomatic tendons ($P = 0.026$). Subjects having patellar tendinopathy presented higher anthropometric data ($P \leq 0.003$ for age, weight, and height) compared with asymptomatic subjects; affected tendons had higher PT thickness compared with healthy ones ($P = 0.016$).

†Represents statistically significant difference in rate of morphology parameters. Tendons with patellar tendinopathy showed higher rate of hypoechoogenicities, hyperechoogenicities and vascularizations compared with asymptomatic athletes ($P < 0.001$).

‡In two subjects, Achilles tendon data, and in four subjects, patellar tendon data are missing.

AT, Achilles tendon; PT, patellar tendon.

Prevalence of tendinopathy

Results show that prevalence of patellar tendinopathy in young athletes at an average age of 13 years is 5.8%. Highest rates of patella tendinopathy were seen in the oldest age group (15–18 years of age), which is comparable to literature. Cook et al. (2000) found a prevalence

of patellar tendinopathy in 7% out of 134 basketball players aged 14–18. Investigations on patella tendons of 57 Swedish elite junior volleyball players (age 15–19) diagnosed jumper's knee in 11% (Gisslen & Alfredson, 2005; Gisslen et al., 2005). Within the study of Gisslen et al. (2005), data of the youngest age group (age 16

years) showed a tendinopathy prevalence of 5%. Other investigations in senior elite and nonelite athletes at age of 22–27 presented prevalence rates of up to 45% in volleyball players (Lian et al., 2005; Zwerver et al., 2011).

In contrast, Achilles tendinopathy was seen less often in adolescent athletes. Tenforde et al. (2011) evaluated the lifetime prevalence of overuse injuries in 748 high school runners between 13 and 18 years of age by use of questionnaires. Prevalence of Achilles tendinopathy was reported to be as high as 6% in males and 9% in females (Tenforde et al., 2011). In a group of 16-year-old elite gymnasts, 12.5% of male and 17.5% of female athletes presented Achilles tendinopathy compared with none in the controls (Emerson et al., 2010). Compared with those data of the literature, it has to be considered as a relatively seldom diagnosis in the investigated adolescent athlete group at an average age of 13 years (Hirschmuller et al., 2010).

Prevalence of intratendinous changes

Vascularizations were seen in 3% of ATs and 11.4% of PTs, respectively. To our knowledge, this is the first study describing vascularization status of young adolescent athletes. Asymptomatic elite dancers (age 18–40 years) showed intratendinous vessels to be present in 8% of ATs and 6% of PTs (Comin et al., 2013). In contrast, data on adult long-distance runners suggest that vascularizations are present in 35% up to 78% of ATs, depending on the presence of symptoms (Hirschmuller et al., 2010). In tendinopathy patients with longer history of pain, prevalence and amount of vascularizations was shown to be at a higher rate (Hirschmuller et al., 2010). Similarly, in the study of Zanetti et al., vascularizations were present in 30 of 55 painful ATs and in one out of 25 asymptomatic ATs (Zanetti et al., 2003). Regarding PTs, Gisslen & Alfredson (2005) reported in their study on 60 elite junior volleyball players' vascularizations to be present in painful and pain-free, structurally altered and normal tendons, respectively. They found intratendinous flow in 26% of all volleyball players, 83% of those in chronic symptomatic tendons and 16% in asymptomatic tendons (Gisslen & Alfredson, 2005). Nevertheless, lower prevalence of vascularizations in the presented cohort of young adolescents is not negligible. Prevalence of hypoechogenicities was shown to be as low as 0.7% in ATs compared with 3.1% in PTs. Morphologic changes within PTs were significantly more often in males than in females. This is in line with results of structural tendon changes in PTs of young basketball players that were also more often in males (Cook et al., 2000). Contrasting this, in young asymptomatic volleyball players, intratendinous changes of the PT were seen significantly more often in females (Gisslen & Alfredson, 2005; Gisslen et al., 2005). Highest rates of intratendinous changes with 5.4% were present in the oldest age group.

Similarly, data of the already cited investigations on basketball and volleyball players showed higher amounts of structural changes of up to 29% in the older groups of young athletes (Cook et al., 2000; Gisslen & Alfredson, 2005; Gisslen et al., 2005). This might be explainable because of their investigated higher impact sport disciplines, the older age, and the years exposed to training. The high impact and training duration model of tendon alterations is supported by the study of Emerson et al. (2010). They reported high rates of sonographically detected changes up 35% in elite gymnasts at an average of 16 years of age. With 4% to 6% control group subjects were shown to have statistically significant fewer changes (Emerson et al., 2010). The lower prevalence of hypoechogenicities within ATs and PTs in the presented cohort might be explained by their relatively young age, different sport disciplines and relatively low amount of systematic training.

Association of intratendinous changes to tendinopathy

As mentioned before, the occurrence of structural tendon changes in asymptomatic tendons is well known, but the clinical importance is still unclear (Zanetti et al., 2003; Gisslen & Alfredson, 2005; Gisslen et al., 2005; Boesen et al., 2006b; van Snellenberg et al., 2007; Hirschmuller et al., 2010). Fredberg & Bolvig (2002) reported that in 17% of asymptomatic senior soccer players with structural PT alterations and in 11% of ATs, players developed tendon symptoms within the next season. Likewise, among junior basketball players, tendons with hypoechoic areas were shown to be of higher risk to develop jumper's knee than unchanged tendons (Gisslen et al., 2007). In ballet dancers, only the presence of moderate to severe hypoechoic defects predicted future AT and PT disability (Comin et al., 2013). In the present study, subjects with patellar tendinopathy showed a statistically significant higher amount of hyper- and hypoechogenicities within the PTs compared with the asymptomatic group. This supports the hypothesis that these changes are already part of the pain development process in adolescent athletes. Also subjects with Achilles tendinopathy presented a higher maximum tendon thickness compared with those without AT symptoms. However, prevalence of structural changes did not differ between groups. Thus, the higher tendon thickness in patients might implicate an initial structural change even in young adolescents, as already described by Malliaras & Cook (2011). Longitudinal investigations including ultrasound techniques for extensive tissue characterization are useful to learn more about the onset of structural tendon changes and its occurrence in the development of tendinopathy (van Schie et al., 2010; Kulig et al., 2013).

Regarding tendon vascularization, some authors interpreted its presence as a generally pathological sign of tendon degeneration, resulting in pain during tendon

loading activity because of the accompanied neural ingrowth (Ohberg et al., 2001; Alfredson et al., 2003; Gisslen & Alfredson, 2005; Gisslen et al., 2005; Rees et al., 2013). Thus, early identification of vascularization seems to be of high clinical interest leading to potential methods to prevent development of symptomatic tendinopathy. In contrast to its generally believed pathological origin other authors considered the presence of low-grade intratendinous vessels a physiological adaptive response (Koenig et al., 2007a; Malliaras et al., 2008; Boesen et al., 2012). Malliaras et al. (2008) found intratendinous flow in 21% of ATs in 61 badminton players, associated with years of badminton competition and hours of weekly competition. Boesen et al. (2012) found no association between intratendinous flow in the Achilles and anterior knee tendons and current pain at the start of the season compared with an 8-month follow-up in 95 badminton players. Long-term results presented a decrease in Doppler activity over the season. Both authors concluded that vascularity could therefore also be a response to mechanical loading (Malliaras et al., 2008; Boesen et al., 2012). Koenig et al. (2007a) saw intratendinous blood vessels in all five asymptomatic subjects examined with morphologically normal Achilles tendons after injection of an ultrasound contrast agent indicating a physiological vascularization status. However, all of the cited studies lack of sufficient subject numbers. In contrast, Hirschmuller et al. (2010) found intratendinous blood flow in more than 30% of ATs in a huge cohort of 634 asymptomatic long-distance runners. In the 1-year follow-up, an increased risk in the development of symptoms for subjects with previous vascularization, a positive history of complaints, and a spindle-shaped tendon thickening was identified (Hirschmuller et al., 2012). For PTs, Gisslen and Alfredson (2005) reported that 33% of asymptomatic volleyball players with vessels at baseline developed symptoms in the follow-up examination after 7-month period of training. Therefore, the presence of vascularization can also be interpreted as an early sign of pathology in the development of tendinopathy (Gisslen & Alfredson, 2005; Hirschmuller et al., 2012). Data of the present study detected an association between prevalence of vascularization and patellar tendinopathy. Moreover, higher grades of vascularization have been found in the patellar tendinopathy group. This supports the hypothesis of an association between vascularization status and the development of clinical symptoms. Interestingly, higher amounts of intratendinous flow could not be seen in adolescent athletes suffering from Achilles tendinopathy compared with the asymptomatic group. This might be explainable by generally lower prevalence of AT complaints and also lower amounts of both vascularization grade and echoic AT changes. Further longitudinal studies with larger cohorts and longer follow-up periods are necessary to clarify the predictive value of those intratendinous alterations.

Limitations

It has to be kept in mind that data of the present study were derived from a cross-sectional study of adolescent athletes presenting in sports medical center due to the preparticipation and annual health examination. Methodologically, two general limitations of the study should be discussed. Firstly, the ultrasound images have been performed by use of two different ultrasonographs, with different probes, MHz rates and settings, especially in the Doppler mode. This could have led to a systematic bias in Doppler grading by an underestimation of the real flow by using the machine with lower capacity. Especially, the detection of small vessels, either if they are physiological or pathological, might be biased. Secondly, four examiners performed the ultrasound measurements, which might also influence the data. However, investigations examining interobserver reliability of eight different radiologists analyzing modified Öhberg score presented moderate to high reliability with an intraclass correlation coefficient up to 0.85 for symptomatic tendons (Sengerij et al., 2009). Furthermore, PT vascularization was scanned with the knee flexed in a 30° position. According to Koenig et al. (2007b), this could lead to an underestimation of the intratendinous flow, as they detected a higher amount of flow by a fully extended knee position (Boesen et al., 2012). Moreover, Koenig et al. (2007a) found intratendinous blood vessels in all asymptomatic subjects with morphologically normal Achilles tendons after injection of an ultrasound contrast agent. Hence, this could also lead to a detection of higher amounts of physiological flow. It is questionable if the detection of single, small vessels is of value in the prediction of pathology. Therefore, it has to be discussed, to modify the recently used grading systems according to high-resolution Doppler technique into a grading that potentially allows differentiation between single, potentially physiological vessels and higher amounts of vessels, which are potentially pathological (Boesen et al., 2012).

Perspectives

In adolescent athletes with an average age of 13 years, prevalence of patellar tendinopathy is 5.8%, while Achilles tendinopathy is present in 1.8%. Prevalence of tendinopathy does not differ between genders and is present in adolescents of all age groups. Intratendinous changes and vascularizations are visible in both symptomatic and asymptomatic Achilles and patellar tendons in adolescent athletes. Patellar tendinopathy is associated with a statistically significantly higher prevalence of structural intratendinous alterations (vascularizations, hypo-, and hyperechogenicities) compared with tendons in asymptomatic athletes. Longitudinal studies are necessary to investigate the physiological or pathological origin of vascularization and its predictive value in the development of

tendinopathy. Furthermore, onset of development of echoic intratendinous changes has to be examined by promising ultrasound techniques in longitudinal designs.

Key words: Prevalence, tendinopathy, sonography, Doppler ultrasound, vascularization, hypoechogenicities, hyperechogenicities, adolescent athletes.

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