Stiffness and Thickness of Fascia Do Not Explain Chronic Exertional Compartment Syndrome

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Abstract

Background Chronic exertional compartment syndrome is diagnosed based on symptoms and elevated intramuscular pressure and often is treated with fasciotomy. However, what contributes to the increased intramuscular pressure remains unknown.

Questions/purposes We investigated whether the stiffness or thickness of the muscle fascia could help explain the raised intramuscular pressure and thus the associated chronic compartment syndrome symptoms.

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Each author certifies that his institution approved the human protocol for this investigation, that all investigations were conducted in conformity with ethical principles of research, and that informed consent for participation in the study was obtained.

This work was performed at Umeå University Hospital and Institute of Sports Medicine, Bispebjerg Hospital.

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Patients and Methods We performed plain radiography, bone scan, and intramuscular pressure measurement to diagnose chronic compartment syndrome and to exclude other disorders. Anterior tibialis muscle fascial biopsy specimens from six healthy individuals, 11 patients with chronic compartment syndrome, and 10 patients with diabetes mellitus and chronic compartment syndrome were obtained. Weight-normalized fascial stiffness was assessed mechanically in a microtensile machine, and fascial thickness was analyzed microscopically.

Results Mean fascial stiffness did not differ between healthy individuals (0.120 N/mg/mm; SD, 0.77 N/mg/mm), patients with chronic compartment syndrome (0.070 N/mg/mm; SD, 0.052 N/mg/mm), and patients with chronic compartment syndrome and diabetes (0.097 N/mg/mm; SD, 0.073 N/mg/mm). Similarly, no differences in fascial thickness were present. There was a negative correlation between fascial stiffness and intramuscular pressure in the patients with chronic compartment syndrome and diabetes.

Conclusions The lack of difference in fascial thickness and stiffness in patients with chronic compartment syndrome and patients with chronic compartment syndrome and diabetes compared with healthy individuals suggests structural and mechanical properties are unlikely to explain chronic compartment syndrome. To prevent chronic exertional compartment syndrome, it is necessary to address aspects other than the muscle fascia.

Level of Evidence Level II, prognostic study. See the guidelines online for a complete description of level of evidence.

Introduction

Although chronic exertional compartment syndrome (CECS) is common, the current literature does not provide

a complete explanation of the pathophysiology of the disease. To prevent and optimally treat the condition, it is crucial to know its etiology and pathogenesis.

CECS presents as an aching or burning muscular sensation during exercise that is relieved by rest. Neurologic pressure symptoms may occur along with the pain symptoms [5, 7]. Most CECSs involve the anterior or deep posterior compartment of the leg [5], and the diagnosis is confirmed by measuring intramuscular pressure before and after exercise. An anterior intracompartmental pressure (ICP) greater than 15 mm Hg at rest and/or greater than 30 mm Hg 1 to 2 minutes after cessation of exercise and/or greater than 20 mm Hg 5 minutes after exercise is diagnostic [18]. Raised ICP is believed to play an important role in the pathophysiology of CECS, and fasciotomy reportedly yields a good clinical outcome and normalized ICP [11, 13, 14, 25]. Ischemia secondary to a raised ICP is believed to cause the pain symptoms [1, 8, 17, 19].

Little attention has been paid to the fascia that separates muscles in compartments, transmits force, and serves as a muscle attachment [6]. This fascia can consist of two to three layers of waved parallel collagen fiber bundles with different fiber orientation between adjacent layers [20, 21]. One study showed that patients with anterior lower leg CECS have thicker muscle fascia compared with fascia from unaffected muscles of the same leg [16]. The same study also suggested fascial stiffness may be related to CECS, although this association was not examined. Fascial thickness was measured by image analysis and stiffness was calculated from coherent measurements of force and deformation. It is well accepted that diabetic individuals have generalized stiffening of connective tissue [2–4, 22]. Recently it was shown that CECS is a common cause of lower leg pain in diabetic individuals [11, 12]. It is possible that diabetic individuals are exposed to CECS because of stiffer fascia.

The aim of this study was to measure and compare the mechanical properties (peak strain, peak force, stiffness) and thickness of the anterior tibialis muscle fascia in healthy individuals, patients with CECS only, and patients with CECS and diabetes. We hypothesized CECS would have stiffer and thicker fascia. We also wished to determine if a correlation between ICP and fascial stiffness could be identified in patients with CECS.

Patients and Methods

All biopsy material was obtained from patients aged between 15 and 65 years admitted to Umeå University Hospital (Umeå, Sweden) during a 2-year period. The study population consisted of 10 patients with diabetes and CECS (the PDC group), 11 otherwise healthy patients with

CECS (the PC group), and for control six healthy individuals (the HI group) without signs of diabetes, CECS, or other diseases. Patients in the PDC group were older ($p < 0.05$) than patients in the HI and PC groups (Table 1). The PDC group consisted of eight patients with Type 1 diabetes and two with Type 2 diabetes, all of whom received insulin treatment. The two patients with Type 2 diabetes had received oral treatment before their insulin treatment. Eight of the 10 patients with diabetes had good diabetic control. Three patients with diabetes were diagnosed with diabetic complications, two of whom had neuropathy. Biopsy specimens from the fascia of the anterior tibial muscle were obtained from the 21 patients in the PC and PDC groups during treatment with fasciotomy, whereas biopsy specimens from patients in the HI group were obtained during surgery of the lower leg attributable to acute injuries. Three patients in the HI group sustained acute tibia condyle fractures and three patients had acute compartment syndrome. Each of the acute procedures performed allowed for biopsy specimens to be taken from the same anatomic location biopsied during fasciotomy. None of the patients in the HI group had a history of lower leg pain and did not show any signs of high compartment pressure after treatment. All 27 patients had normal palpable foot pulses and were without clinical signs and symptoms of distal circulatory impairment. Plain radiography and bone scan of the leg were performed on the patients in the PC and PDC groups to exclude bone or joint disorders causing pain. All patients in the PC group and most patients in the PDC group improved after fasciotomy. All patients who were asked chose to participate in the study. All patients gave informed consent for participation in the study, and the study was approved by the Ethics Committee of Umeå University.

Treadmill exercise was used to reproduce symptoms, with increasing velocity over 10 minutes, and the velocity and slope of the treadmill were adjusted so that previous leg pain was reproduced. Tibialis anterior muscle ICP

Table 1. Demographics and fascial mechanical properties and thickness

Variable	HI group	PC group	PDC group
Sex (female/male)	3/3	7/4	6/4
Age (years)	38.8 (19.7)	29.1 (13.7)*	47.5 (13.0)*
Peak strain (%)	34.5 (11.8)	33.8 (6.9)	40.1 (22.5)
Peak force (N/mg/mm)	2.2 (1.3)	1.5 (1.3)	2.4 (2.2)
Stiffness (N/mg/mm)	0.120 (0.077)	0.070 (0.052)	0.097 (0.073)
Thickness (μm)	453 (146)	454 (137)	478 (201)

Values are expressed as means, with SD in parentheses; * $p < 0.05$ between groups; HI = healthy individuals; PC = patients with CECS only; PDC = patients with diabetes mellitus and CECS; CECS = chronic exertional compartmental syndrome.

measurements were performed on the PDC and PC groups while at rest and after treadmill exercise. ICP was monitored during microcapillary infusion with isotonic saline at a rate of 1.5 mL/hour via a catheter (Myopress; Atos Medical, Höör, Sweden) connected to a pressure transducer (PMSET 2DT-XO 2TBG; Becton Dickinson, Singapore). During local skin anesthesia, an ordinary Teflon[®] cannula was inserted into the anterior tibial compartment of the leg approximately 10 to 15 cm below the fibular head at an angle of 30° to the skin in the distal direction. Using this cannula, the Myopress catheter was introduced to a depth of at least 15 to 20 mm beneath the fascia. The cannula then was retracted. Measurements were performed in both legs with the patient in the supine position at rest and 1, 5, 10, and 15 minutes after exercise. The diagnosis of CECS was based on the following criteria: (1) history of exercise-induced pain/symptoms with reproduction of symptoms and pain during the exercise test and negative plain radiography and bone scan, and (2) ICP at rest greater than 15 mm Hg and/or ICP greater than 30 mm Hg 1 to 2 minutes after the end of the exercise test and/or ICP greater than 20 mm Hg 5 minutes after the end of the exercise test concomitantly with the reproduced leg pain. All patients with CECS fulfilled each criterion.

Fasciotomy of the anterior tibial and peroneal compartments was performed [14, 23]. By way of a 5-cm skin incision halfway between the fibular shaft and the tibial crest in the midportion of the leg, a subcutaneous dissection of the fascia was made as far as possible in the proximal and distal directions of the anterior and lateral compartments. Both compartments then were decompressed by a fasciotomy. During fasciotomy, a 1-cm-wide strip of fascia from the edge of the fasciotomy was removed. This strip was removed in all patients and at the same anatomic location of the fascia. Two biopsy specimens from the fascial strip were immediately frozen in liquid propane cooled in liquid nitrogen and then kept at -80°C. One biopsy sample was used for measurement of fascial thickness; the other underwent mechanical testing. Fascial samples, approximately 3 × 3 mm, were oriented and mounted for longitudinal and transverse sectioning in OCT compound (Tissue Tek[®]; Miles Laboratories, Naperville, IL, USA). Serial cross sections 6 to 7 µm thick were cut in a cryostat microtome at -20°C and mounted on glass slides. Hematoxylin and eosin and Van Gieson staining were used to observe collagen and general morphologic features. The fascial cross sections were scanned in a Nikon Eclipse E800 microscope (Nikon Inc, Melville, NY, USA) connected with a SPOT RT Color Camera (Diagnostic Instruments Inc, Sterling Heights, MI, USA). SPOT software v 4.0 (Diagnostic Instruments) was used for image analyses and measurements. The thickness of the fascia was measured in longitudinal and horizontal sections

of each sample. The measurements, performed on sections stained with hematoxylin and eosin, were repeated along the cross sections with a distance between each measurement of approximately 100 µm. One investigator (PS), who was blinded regarding the clinical data of the subjects, performed all morphologic analyses.

A microtensile mechanical rig (Fig. 1) with corresponding software (MICROTEST Materials Testing Module Version 5.2; Deben UK Ltd, Suffolk, UK) was used for mechanical testing of fascial specimens at the Institute of Sports Medicine, Bispebjerg Hospital (Copenhagen, Denmark).

The custom-made mechanical test rig consisted of a 1-kg strain gauge transducer mounted in a specimen chamber and a motorized spring-loaded linear variable differential transformer (LVDT; RDP Electronics, Wolverhampton, UK) that achieved and measured changes in grip displacement at a predetermined velocity. The strain gauge transducer displayed linearity from 0 to 1000 g, with a 0.16% accuracy, and the LVDT had a resolution of 0.1 mm. The rig was placed directly under a stereoscopic microscope (SMZ1000; Nikon, Tokyo, Japan) with a C-mount lens (×0.38). The microscope was equipped with a 15-Hz digital camera (DFWX700; Sony, Tokyo, Japan) with a 1024 × 768 output signal format that allowed acquisition of detailed image data of the sample.

Fascial specimens were prepared from fascial biopsy specimens and were cut to approximately 3 × 10 mm. The length axis of the fascial specimens corresponded to the axial lower leg direction. The middle specimen portion (5 mm) was wrapped in phosphate-buffered saline (PBS)-soaked (0.15 mol/L) gauze whereas the ends were allowed to air dry at room temperature. Each dry end was glued between two stainless steel plates that were glued (cyanoacrylate) to the aluminum mounting plates on the rig and

Fig. 1 The microtensile mechanical rig used for mechanical analyses is shown.

allowed to cure for 30 minutes. Pilot studies (data not published) showed the glue could withstand the highest tensile force when applied like this. Specimen and mounting plates were submerged in the PBS-filled (0.15 mol/L) Petri dish on the rig and incubated for 20 minutes before specimen displacement with a strain rate of 2.0 mm/second. The test included five preconditioning cycles to a strain of 2% followed by a failure test 1 minute later. The same methodology has been used for testing tendon tissue to subfailure strain levels with acceptable reproducibility [24]. The initial specimen testing length (mounting plate-to-mounting plate distance) was approximately 5 mm, and tissue displacement was recorded from the point of onset of force. All failure tests resulted in failure in the central region of the specimen. After tests, the fascial tissue was cut off as close to the mounting plates as possible and subsequently freeze dried in a ScanVac freeze dryer (NINOlabs, Glostrup, Denmark) to obtain dry weight.

Exact initial specimen lengths were measured from stereomicroscopic digital camera images of the mounted specimens in the PBS solution (0.15 mol/L) at the onset of force. Starting length, L_0 , was defined as specimen length at onset of tensile force. Peak point was defined as the point of highest force during failure. Deformation from L_0 to peak point, ΔL , was set in relation to starting length and expressed as a percentage to give peak strain ($\Delta L/L_0 \times 100\%$). Peak force was defined as the highest force normalized to specimen starting length and dry weight. Peak force was used instead of stress because it was not possible to determine the cross-sectional area of the specimen accurately. The measure has been used previously with wet weight [2]. We used dry weight to eliminate possible fluctuations in weight attributable to tissue hydration status. Stiffness of the specimens was obtained from the linear portion of the force-deformation curves and defined as force normalized to starting length and dry weight and divided by strain. Statistical analyses were performed by a nonblinded investigator (MD) using the Kruskal-Wallis test with subsequent Dunn's post hoc test when appropriate. Spearman correlation analysis was used to examine relationships between variables. An alpha level of 0.05 was considered significant. Results are reported mean $\pm$ SD.

Results

There were no differences among the three groups with respect to peak strain, peak force, stiffness, or thickness of the fascial biopsy specimens. (Table 1).

Regarding the relationship between fascial stiffness and ICP 5 minutes after exercise, there was no correlation for the PC group, but there was a negative correlation ($r = -0.66$; $p < 0.04$) between stiffness and ICP for the

Fig. 2 The graph shows fascia stiffness versus ICP 5 minutes after exercise. There was no correlation between stiffness and ICP for the PC group, but there was a negative correlation ($r = -0.66$; $p < 0.04$) between stiffness and ICP for the PDC group.

PDC group (Fig. 2). The two patients with diabetes with an ICP exceeding 100 mm Hg did not stand out in any way. One was 25 years old with insulin-treated Type 1 diabetes for 11 years. The other was 40 years old with insulin-treated Type 1 diabetes for 40 years. Neither showed diabetic complications or signs or symptoms different from the other patients with CECS. Despite their extraordinarily high ICP, they were not excluded from the analysis as that would be selection bias and there was no fair reason for excluding them. There was a positive correlation ($p < 0.01$) between age and fascial stiffness for the PDC group. No such correlation was present for the HI or PC group. Fascial stiffness was unrelated to diabetes duration.

Discussion

CECS is diagnosed based on symptoms and elevated intramuscular pressure and often is treated with fasciotomy. However, what contributes to the increased intramuscular pressure remains unknown. In this study, we compared the mechanical properties and thickness of the anterior tibialis muscle fascia in healthy individuals, patients with CECS, and patients with CECS and diabetes. There were no differences in mechanical properties or thickness of the fascia among the groups, and fascial stiffness appeared to be unrelated to ICP. In contrast to our hypothesis, these data imply structural and mechanical properties of the fascia are not major contributing factors to the etiology of CECS.

Our study has inherent limitations, including sample size and intersubject variation. To what extent the magnitude of the variation is related to biologic or measurement variation is difficult to ascertain. Also, the age difference between patients in the PDC and PC groups may have confounded the results. However, fascial stiffness in the PC group was unrelated to patient age. Fascial biopsies from diabetic patients without CECS might have provided more information on the effect of diabetes on fascial tissue and the role of diabetes in CECS. Arterial insufficiency as the cause of symptoms in the PDC group was ruled out by the absence of signs or symptoms of claudication, but measurements of ankle blood pressure and ankle/arm index would have provided additional information on this subject. Further, analyses of collagen content and crosslink composition would have provided a more coherent picture but were not possible to obtain in the current study.

In our study there were considerable variations in the stiffness values. In fact, the SDs of the stiffness represented 64% to 75% of the group mean values, which corresponds well with that of others (49%–74%) [10, 16]. However, albeit not statistically significant, it is noteworthy the average stiffness value was 71% larger for the patients in the HI group than for the patients in the PC group, rather than the contrary, which further supports the notion that CECS and fascial stiffness are unrelated. However, the sample size was rather small and the between-subject variation was considerable.

To our knowledge, there are no comparable studies reporting the same outcome measures of mechanical properties of human fascial tissue as those measured in our study. In one study, a peak force for diabetic rat tail tendon of 7 to 10 N/mg wet weight/mm initial specimen length was reported [2], compared with the 2.4 N/mg dry weight/mm initial specimen length for our PDC group. Studies of human tendon have generally reported peak strains lower than those in our study [9, 26]. Strain calculations depend on the method of measurement since grip-grip measurements typically exceed midsubstance measurements [15]. Our strain values thus might overestimate midsubstance strain and underestimate midsubstance stiffness. One study reported thicker posterior-proximal crural fascia (924 μm) [21], and another study reported thinner anterior crural fascia (220 to 350 μm) [16], than the fascial thickness of 453 μm in our HI group. Collectively, the fascial tissue in our study seems comparable to other human fascial tissue and with less stiffness than human tendon tissue.

In our study, the fascial thickness in the three groups was on average remarkably equal. The lack of difference in fascial thickness among our groups is in contrast to the findings of Hurschler et al. [16], and this discrepancy is difficult to reconcile. It is possible the discrepancies may be attributable to method of measurement. The measure of fascial thickness does not represent only collagen fiber content but also loose

connective tissue and fat. Notwithstanding these considerations, our data on fascial thickness suggest it does not play a major role in the etiology of CECS.

Individuals who have diabetes are known to have generalized stiffening of connective tissue [2–4, 22]. One possible explanation for tissue stiffening is the accelerated formation of the collagen cross-links called advanced glycation end products. Studies have shown CECS is a common cause of lower leg pain in diabetic individuals [11, 12]. With this in mind, we included persons with diabetes and CECS (the PDC group) to ensure the detection of a ‘stiffer’ fascia, which would support the hypothesis of stiff fascia playing a part in the pathophysiology of CECS. Surprisingly, the fascia of the PDC group was not stiffer than that of the PC or HI group. Therefore, the mechanical properties of fascia of diabetic individuals do not seem to confirm the study hypothesis of stiff fascia contributing significantly to CECS. They do not affirm the hypothesis either, since this would require a comparison of fascial tissue of diabetic persons with and without CECS, which was beyond the scope of this study.

The negative correlation between fascial stiffness and ICP (after exercise) for the PDC group is interesting. Not only does it further rule out the possibility of stiff fascia in diabetic individuals as the cause of CECS in our study, it also gives rise to the speculation that high ICP might decrease stiffness of the fascial tissue in persons with diabetes. Perhaps the diabetic individuals with higher neuropathy-induced thresholds of pain need to produce higher ICPs to provoke the symptoms, and higher ICPs might decrease stiffness of fascial tissue. There was a positive correlation between age and fascial stiffness only for the PDC group. The obvious interpretation would be that it was a matter of years of diabetes rather than biologic age, but diabetes duration and fascial stiffness were not correlated. Clearly, more research is needed to address these speculations.

The structural and mechanical properties of anterior tibialis muscle fascia were similar in healthy individuals, patients with CECS, and diabetic patients with CECS. Fascial stiffness appeared to be unrelated to ICP, and thus it seems structural and mechanical properties of the fascia are not major contributing factors to the etiology of CECS.

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