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ORIGINAL RESEARCH

Greater Resting Lumbar Extensor Myofascial Stiffness in Younger Ankylosing Spondylitis Patients Than Age-Comparable Healthy Volunteers Quantified by Myotonometry

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Abstract

Objective: To quantify resting lumbar erector myofascial stiffness in younger patients with ankylosing spondylitis (AS) and age-comparable healthy control subjects using a handheld mechanical impulse-based myotonometric device.

Design: A case-control study of 24 patients with AS and 24 age-comparable healthy control subjects.

Setting: University physical therapy department.

Participants: Patients with AS (men: n=19; women: n=5; total: N=24) and healthy volunteers (men: n=19; women: n=5; total: N=24) without low back pain (age range, 18–46y).

Interventions: Not applicable.

Main Outcome Measure: Lumbar myofascial stiffness.

Results: At the initial measurements, median stiffness (Nm) of the averaged right- and left-sided values was greater ($P=.021$) in 24 patients with AS than 24 control subjects (268.9 vs 238.9, respectively). Repeated measurements after a 10-minute prone resting period were also greater ($P=.007$) in patients with AS than control subjects (281.0 vs 241.4, respectively). The 48 averaged right- and left-sided values from baseline and 10-minute measurements were compared in each subject group. The patients with AS more frequently ($P=.012$) had stiffness values >250Nm (35 [72.9%] vs 22 [45.8%] in control subjects).

Conclusions: Lumbar myofascial stiffness was greater in 24 patients with AS than in the control subjects. A hypothesized biomechanical concept of increased resting lumbar myofascial stiffness in AS may be supported by this preliminary controlled study.

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Ankylosing spondylitis (AS) is a chronic, usually progressive, spinal disease with symptomatic onset typically developing in later teen and younger adult ages and predominating in men.[1] Definite diagnosis is often delayed ≥5 years.[2] Objective loss of function and radiographically evident spinal damage usually occur in later stages of disease.[3-5] Symptomatic stiffness of the low back is an early manifestation and has been reported as a significant quality of life concern in 90% of patients with AS.[6] Greater palpable low back muscle stiffness and tightness were reported clinically in early stage patients with AS,[7,8] but these physical properties have not been quantified. Such objective measurements could enhance clinical evaluation of patients with AS and may aid in both earlier diagnosis and management by physical medicine professionals.[9]

Inflammation and osteoproliferation are the characteristic pathologic lesions of the spine in AS, occurring at attachment (enthesopathic) sites of ligaments, fibrous tissues, or capsules to vertebral bodies or facet joints.[10,11] The underlying mechanisms for the onset and course of AS are not known.[1,11,12] Pathogenesis

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is currently believed to mainly involve immunologic and inflammatory system pathways, but biomechanical stress and microinjury may also be predisposing and perpetuating contributors to the inflammatory reactions.[1,9,11-13]

Intensive physical rehabilitation treatments were reported to have continued symptomatic benefits for patients with AS,[14] which also implies that biomechanical pathways may be operating in the course of AS.[1,9,13] Available pharmacologic therapies for AS, including nonsteroidal anti-inflammatory drugs and antitumor necrosis factor inhibitors, have been shown to improve spinal mobility,[15] but not to prevent osteoproliferation or ankyloses.[16] To our knowledge, no previous study has quantified the biomechanical properties of lumbar myofascial tissue in this disease.

Abnormalities in human resting muscle/myofascial tone[17] and correlated muscle firmness have been associated with clinical disorders, including muscle pain and tension-type headache.[18-20] Polymorphic variation in vertebral instability, as occurs in adolescent idiopathic scoliosis, was hypothesized to result from insufficient innate spinal myofascial tone or stiffness.[21] To the contrary, excess spinal myofascial tone or stiffness property was hypothesized to increase risk of developing AS.[21] Excess axial myofascial stiffness might contribute to increased stress and spinal microinjury at entheses (enthesopathy) and may lead to abnormal tissue repair responses.[9,10] To our knowledge, no quantitative research has been reported on the possible role of increased resting lumbar myofascial tone as an associated factor in AS.[1,9,21]

The MyotonPRO[a] is a handheld mechanical impulse—based myotonometer which permits prompt, quantitative, and noninvasive measurements of tissue stiffness, tension, and elasticity. It was used to quantify myofascial stiffness of resting lower lumbar myofascial tissue in this study. In previous investigations,[22-24] this instrument precisely measured active and passive states of peripheral myofascial tissues, but not at the lumbar site. Additionally, the MyotonPRO device has been used to measure properties of pathologic muscle states (eg, Parkinson disease,[25] subacute stroke[26]). In patients with Parkinson disease,[25] changes in skeletal muscle stiffness at rest have been observed after pharmacotherapy using the MyotonPRO. Such results suggest the value of myotonometry in evaluating disease where skeletal muscle stiffness abnormalities may play a role.[1,9,21]

The primary objective of this study was to quantify resting lumbar myofascial stiffness in subjects with AS compared with age-comparable healthy individuals using concurrent surface electromyography (sEMG) measurements to monitor nonactivation status.[27] The patients with AS were hypothesized to have greater resting lumbar stiffness compared with healthy control subjects.

Methods

Participants

Men and women with AS between ages 18 and 46 years, diagnosed by board-certified rheumatologists, were recruited to participate as cases in this study. They were recruited from local medical centers and rheumatology clinics from October 2012 to February 2014. The research protocol was approved by our institutional review board, and all participants provided informed consent. All patients with AS satisfied the Assessment of SpondyloArthritis international Society criteria for axial spondyloarthritis.[28] Exclusion criteria were previous spine surgery or body mass index (BMI) $\geq$35kg/m^2. Healthy men and women were control subjects of a comparable age range with the patients and were recruited from community institutions via advertising. Exclusion criteria for healthy subjects were current pain or stiffness, chronic neurologic or musculoskeletal condition, previous spine surgery, or BMI $\geq$35 kg/m^2. Sample size was based on a predicted estimate of 80% of AS cases having lumbar myofascial stiffness >250Nm in a mixed sex sample (4 men, 1 woman) compared with 40% in healthy subjects based on previous analyses.[29] The estimate yielded a sample size of 23 in each group, assuming a power of .80 and a type 1 error of .05. A total of 24 (19 men, 5 women) patients with AS meeting Assessment of SpondyloArthritis international Society criteria for axial spondyloarthritis[28] were recruited and compared with 24 healthy control subjects (19 men, 5 women) of similar age, BMI, degree of physical activity, and hand dominance (table 1).

List of abbreviations:

AS	**ankylosing spondylitis**
BMI	**body mass index**
CNS	**central nervous system**
sEMG	**surface electromyography**

Questionnaire

Each subject completed a questionnaire, including 14 demographic, health, and physical activity items prior to undergoing MyotonPRO measurements in the Physical Therapy Department at Bradley University, Peoria, Illinois. The subjects with AS responded to 4 additional items regarding disease onset, family history of AS, and Bath Ankylosing Spondylitis Disease Activity Index.[30]

Measurement

Stiffness (Nm) of the erector spinae myofascia at the lumbar (L3-4) level was quantified using a myotonometer. Surface electromyography was monitored (<5μV), consistent with resting status. Measurements were made in the prone position on the left and right sides, at baseline and the 10-minute resting interval.

The MyotonPRO is a noninvasive device which applies a mechanical impulse to the skin via a probe, which is transmitted to the underlying soft tissue and muscle.[22-24,29] The myofascial tissue responds as a damped natural oscillation to the imposed mechanical impulse (0.40N for 15ms). The oscillation is quantified by an accelerometer attached to the probe. The myofascial stiffness is calculated from the acceleration signal measurements and the double integral of the signal.[24,29]

Dynamic stiffness (S; Nm) of tissue is defined as follows: $S = m\ a_{max}/\Delta l$, where m is the 18g preload/mass of the myotonometer sensor (probe), a_{max} is the maximum amplitude of the acceleration signal, and Δl is the amplitude of the first nadir in the displacement signal, derived by double integration of the acceleration signal.

The MyotonPRO was reported to have high validity and reliability in quantifying physical properties of stiffness, tension, and elasticity of skeletal muscle.[22-24] sEMG was recorded concurrently with all MyotonPRO measurements using FlexComp Infiniti.[27,b]

Standard small-size, pregelled round, silver-silver chloride electrodes (Uni-Gel T3425[b]) were used. Placements were 2.5cm above and below the marked site at the L3-4 level, where the