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Treatment of patellar tendinopathy with extracorporeal shock wave therapy

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Subjects in this evaluation of a new treatment for activity-related anterior knee pain demonstrated significant improvement.

Objective: To determine the effectiveness of extracorporeal shock wave therapy (ESWT) on patellar tendinopathy.

Method: This was a single-centre, randomized controlled trial, with 20 male and female subjects ranging from age 23 to 52. Treatment subjects received three to five sessions of ESWT and the control subjects received three to five sessions with the use of an energy-absorbing pad. The effects of the shock wave therapy were measured using the Victorian Institute of Sport Assessment (VISA) test and a vertical jump test. Pretreatment ultrasound was used to determine which subjects had neovascularization in the area of the patellar tendon.

Results: ESWT resulted in a significant improvement in the VISA score for questions 1, 3, and 6; for the VISA total score; and for the vertical jump score. Additionally, anecdotal evidence suggested an overall decrease in pain and an increase in function.

Conclusions: Extracorporeal shock wave therapy appears to be a useful adjunct to the eccentric drop-squat strength protocol in the treatment of chronic patellar tendinopathy. Future studies need to include a larger subject pool, a long-term follow-up, and a reliable objective measure of the microscopic and macroscopic improvement of the patellar tendon.

Patellar tendinopathy refers to a clinical condition of activity-related anterior knee pain assessed with tenderness at the inferior pole of the patella at the patellar tendon attachment. It is an overuse injury caused by repeated mechanical stress on the patellar tendon. Repetitive movement of the extensor mechanism of the knee overloads and damages the tendon. This injury is especially prevalent in running and in sports involving a large amount of running and jumping, such as basketball, volleyball, and soccer.^[1]

Numerous conservative treatments (ice, rest, heat, massage, nonsteroidal anti-inflammatory medication, physical therapy modalities, corticosteroid injections, strengthening, stretching, bracing, taping, and orthotics) and surgical treatments have been recommended for patellar tendinopathy.^[2-4] The most effective of these treatments to date is a strengthening program with eccentric drop-squat exercises.^[5] While most cases can be treated conservatively, surgical treatment may be required when noninvasive therapy has not been effective or if there is a complete rupture of the patellar tendon.^[2,3]

Objective

Overall, the conservative and surgical treatments for patellar tendinopathy have not proven to be highly successful in relieving symptoms to such a degree that athletes can continue to participate in their sport at their full potential. Although many methods of treatment permit athletes to return to activity, a form of therapy that eliminates the pain, resolves the symptoms, treats the patellar tendon itself, and makes surgical intervention unnecessary is required. Extracorporeal shock wave therapy (ESWT) may be such an effective treatment and therefore needs to be evaluated.

Extracorporeal shock wave therapy, or lithotripsy, was initially used to treat kidney stones, and then to treat stones at other sites, such as the gallbladder, bile duct, and pancreas. By the mid-1980s, Valchanou and Michailov were using ESWT to stimulate healing with bone fractures.^[6] Since the early 1990s, shock wave therapy has been shown to be effective in treating chronic tendinopathies of the elbow, shoulder, and heel regions,^[7-10] and chronic calcific tendinitis of the shoulder.^[11-15]

A substantial amount of research has involved the use of shock wave therapy on calcific shoulder tendinitis,^[7,11-14,16] lateral epicondylitis,^[9,10,12,13,17] and calcaneal spurs, but little research has involved patellar tendinopathy.

The purpose of this study was to evaluate the effectiveness of ESWT on patellar tendinopathy, as the current forms of therapy for this condition are not adequate, especially for elite athletes. The hypothesis was that ESWT would reduce pain and improve function in athletes with chronic patellar tendinopathy

Method

This study was a single-centre, randomized controlled trial. Twenty individuals, who participated regularly in running and/or jumping sports and were diagnosed with patellar tendinopathy, were recruited as subjects (10 males and 10 females, ranging from age 23 to 52). One of the subjects was removed from the control group before the study ended and his data were not utilized in any of the results.

The criteria used to make the diagnosis of patellar tendinopathy were:

- Pain at the inferior pole of the knee with training.
 - Tenderness at the inferior pole of the patella with palpation.
 - Absence of other knee disorders, such as patellofemoral stress syndrome.
- Each potential study subject was screened to meet the following inclusion criteria:

- Diagnosis of patellar tendinopathy as described above.
 - History of patellar tendinopathy for at least 3 months.
 - Pain that was non-responsive to nonsteroidal anti-inflammatory medication.
- Each potential study subject was also screened to meet the following exclusion criteria:

- Younger than 18 years of age.
- Receiving physiotherapy (eccentric loading/stretching, etc.) within 4 weeks of randomization visit.
- Receiving NSAIDs or acetaminophen for any chronic condition within 1 week of randomization visit.
- History or physical findings of lumbar disc disease, compression syndrome, local arthrosis, or neurological abnormality.
- Previous surgery for patellar tendinopathy.
- Thrombopathy, infection, tumor, or other severe systemic disease.
- Receiving systemic therapeutic anticoagulation.
- Pregnancy.
- Previous ESWT treatment.

All subjects who were selected provided written informed consent before entering the study.

Subjects were randomly assigned to two groups: the treatment group, designated TG (five males and five females), who received active treatment, and the control group, designated CG (five males and five females), who received placebo treatment. The TG received three to five treatment sessions of 2000 shocks each at maximum energy level 4 (0.17 mJ/mm²). The CG received three to five treatment sessions of 2000 shocks each at energy level 4, but with the use of an energy-absorbing pad. A maximum of two specific points of tenderness were treated at each treatment session. In both the treatment and control groups, the first three treatments were administered weekly.

The Siemens Sonocur machine was used for all treatments in this study. This machine is a low-energy device with medium footprint and accurate focusing, and is used without imaging. The machine generates shock waves using an electromagnetic acoustic source. The patient receives neither sedation nor anesthesia. The area to be treated is confirmed by palpation. A coupling gel is applied and the shock module coupling bellows are placed in contact with the painful site.

The method used to determine which subjects received a fourth and fifth treatment involved the subjects' subjective measure of their degree of improvement. The final two treatments were given no earlier than 3 weeks after the third treatment session. The fourth and fifth treatments were given 1 week apart.

The researchers and the subjects were not aware which subjects were assigned to the treatment group. The technician who administered the treatment randomly assigned the subjects to either the placebo or active treatment group, keeping this information encoded.

Evaluation

The age, activity level, frequency and type of training, presence of anterior knee pains, and history of injury of the subjects were evaluated with a questionnaire ([Figure 1](#)^[4]). [Requires Adobe Acrobat]

Real-time spatial compound imaging was done before treatment to aid the diagnosis of patellar tendinopathy and determine the severity.

Other methods of evaluation used were a vertical jump test and the Victorian Institute of Sport Assessment (VISA) test ([Figure 2](#)^[5]). [Requires Adobe Acrobat] Both of these assessments were

done before, during, and after the treatment sessions. The vertical jump score was used to analyze the functional impairment of the tendinopathy and the VISA score was used to assess the degree of pain as well as the functional impairment. The VISA has proven to be a reliable method of assessing the severity of tendinosis based on symptoms and function.[18] The test has been particularly useful given that patellar tendinopathy does not have any pathognomic symptoms other than tenderness with palpation, and its diagnosis often relies on patients' ratings of pain.[2,3]

Statistical analysis

This study is a pretest, midtest, and posttest randomized controlled study. Between-group differences on the VISA score and the vertical jump score were analyzed using analysis of variance with a statistical significance set at $P < .05$. The primary endpoint was the status of the patient on the VISA score at 12 weeks after the final ESWT treatment. Subject data were collected before treatment, 3 to 4 weeks after the third treatment, 3 to 4 weeks after the fifth treatment, when applicable, and at 12 weeks after the last treatment session. The subjects were not aware of their assignment to a particular group at the pretreatment and midtreatment point, and at 3 to 4 weeks posttreatment. However, they were aware of their assignments at 12 weeks when the final data were collected.

Results

The results of the study are based on the vertical jump scores, the VISA scores, anecdotal reports, and neovascularization as shown by ultrasound images. There were no significant differences between the groups at the start of the study.

Vertical jump score

The group change in vertical jump scores showed significance ($P < .05$). The mean change in vertical jump results for the control group was 0.0 inches and the mean change for the treatment group was 1.5 inches.

VISA score

Significant results were seen in the total VISA score versus group and time effects ($P < .05$). The mean VISA score for the control group was 49.9 pretreatment, 54.3 posttreatment at 5 weeks, and 53.2 at 12 weeks. The mean VISA score for the treatment group was 54.4 pretreatment, 65.6 posttreatment at 5 weeks, and 61.4 at 12 weeks.

Further analysis of the VISA score questions revealed VISA question 3 results to be significant ($P < .05$). The mean change for the control group was 0.85 and for the treatment group was 2.3.

The absolute group value for VISA question 6 was also significant ($P < .05$). The mean scores for the control group were 3.9 pretreatment, 4.2 posttreatment at 5 weeks, and 5.3 at 12 weeks, compared with 4.8 pretreatment, 7.3 posttreatment at 5 weeks, and 5.7 at 12 weeks for the treatment group.

Significant group effects were seen in VISA question 1 absolute group values ($P < .05$). The mean control group values were 6.3 pretreatment, 6.0 posttreatment at 5 weeks, and 6.6 at 12 weeks, compared with 8.1 pretreatment, 8.8 posttreatment at 5 weeks, and 7.4 at 12 weeks for the treatment group.

Other data

The pretreatment ultrasound results of 13 subjects were evaluated. These results showed that 6 of the 13 subjects had neovascularization in the area of the patellar tendon.

After treatment, anecdotal reports from the treatment group revealed five out of nine subjects had decreased pain and increased function; two subjects still experienced pain with stairs but felt they had improved overall, and two subjects reported still feeling pain with activity.

In the control group, six of seven subjects reported no improvement or change. One of the control subjects experienced some pain relief after the five treatment sessions, but this subject also reported decreased frequency of activity during the study.

Discussion

The primary etiologic factors for patellar tendinopathy include fatigue and overuse of the extensor mechanism, which together result in microtrauma to the patellar tendon.[2] Without proper rest and repair, collagen and matrix production is reduced and the tendon becomes more vulnerable to injury—a situation that can ultimately lead to a complete rupture of the tendon.[3] Furthermore, the intensity and the frequency of training are factors in the development of patellar tendinopathy, as are specific sport skills involving rapid acceleration, deceleration, jumping, and cutting (changing direction).[3]

Ferretti[1] found that extrinsic factors such as the intensity, frequency, and duration of training as well

as training surface were more consequential than intrinsic factors. Anatomical and biomechanical abnormalities that were assessed included high Q-angle (quadriceps angle from anterior inferior iliac spine to centre of patella to tibial tubercle), limb length discrepancy, pelvic asymmetry, patellar squinting (inward pointing patella from femoral neck anteversion), genu varum and genu valgum, femoral anteversion, and foot abnormalities. None of these abnormalities were significantly correlated with patellar tendinopathy; however, muscle and gait imbalances were related.[19]

Pathology of jumper's knee

Patellar tendinopathy, otherwise known as jumper's knee, has often been referred to as patellar tendinitis. The macroscopic pathology of patellar tendinitis is degeneration of the tendon with an inflammatory response, while the pathology of jumper's knee is noninflammatory degeneration, which requires replacing the term patellar "tendinitis" with patellar "tendinosis." [3]

Mucoid degeneration occurs in the patellar tendon of patients with patellar tendinosis. Macroscopically, the tendon's tissue is soft, yellow-brown, and disorganized. Microscopically, the tendon has increased ground substance, abnormal collagen, tenocytes, and vasculature, fibrocartilagenous metaplasia, fibroblast proliferation, capillary ingrowth, and fibrinoid necrosis.[2,3]

Recently, H. Alfredson of Sweden used perfusion microdialysis to investigate 20 patients with chronic Achilles tendinopathy. On the fluid analysis over a 2-hour period, there was a lack of E2 prostaglandin (indicating an absence of inflammation), increased ischemia with high lactate levels, and, for the first time, elevated levels of glutamate, a potent pain neurotransmitter.[20] This was followed by a biopsy that identified the glutamate receptor NMDAR1. Furthermore, Alfredson has used ultrasound color Doppler to identify neovascularization with vascular ingrowth in the area of nodular tendinosis in 28 of 28 patients with chronic tendinopathy. In a recent pilot study of 10 subjects, Ohberg and Alfredson reported elimination of pain in these patients for up to 6 months with sclerosing these new vessels with polidocanol as used in sclerosing varicose veins.[21]

Mechanism of ESWT

Several different theories attempt to explain how extracorporeal shock wave therapy works. Haist and von Keitz-Steeger have three hypotheses to explain the mechanism involved with shock wave therapy:

- "Shock waves damage cell membranes and therefore nociceptors cannot build up a potential to transmit pain signals."
- "Nociceptors which are stimulated by shock waves send a high frequency of impulses which are suppressed by a gate-control mechanism."
- "Shock wave-induced pericellular free radicals change the chemical milieu and pain-suppressing substances are released." [22]

Rompe and others suggest another hypothesis regarding the mechanism of ESWT in which the shock wave therapy alleviates pain by hyperstimulation analgesia.[9] According to this mechanism, intense stimulation controls pain via brain-stem mechanisms that exert descending inhibitory control over neural transmission through the dorsal horns and at higher levels in the somatic projection system.[9] As a result, no additional transmission of nociceptor information occurs.[12]

Other ESWT studies

The effects of ESWT on calcific rotator cuff tendinitis,[7,11-14,16] calcaneal spurs,[8,13,15] lateral epicondylitis,[9,10,12,13,17] Achilles tendinitis[23], plantar fasciitis,[24] and pseudarthrosis,[6,12,13,25] have been studied and ESWT has been found to have a success rate of approximately 80%.[13] However, the effects of shock wave therapy on patellar tendinopathy have been researched at only a preliminary level.[8,9,11,13,24-26]

The single previous study involving shock wave therapy specifically for the treatment of patellar tendinopathy was done by Lohrer and colleagues at the Institute of Sports Medicine in Germany. When the effectiveness of extracorporeal shock wave therapy was evaluated in 35 subjects with jumper's knee, ESWT was found to be an effective noninvasive method of treating patellar tendinopathy.[27]

Unfortunately, a comparison of the study described in this article with other studies is difficult for a number of reasons:

- Lack of research involving the effect of shock waves on patellar tendinopathy.
- Current research on other forms of tendinopathy uses different numbers of impulses and different shock waves (with the majority of studies using high-energy density shock waves that require the use of anesthetic).
- Some studies do not include a control group and are not blinded.[7,16]
- Follow-up times differ from study to study.

However, a comparison of results achieved with the higher shock wave frequency (0.28 mJ/mm²–0.8

mJ/mm²) used in most other studies and the results achieved in this study with a low frequency (0.17 mJ/mm²) may suggest that lower shock wave frequency provides more effective long-term results for decreasing pain and increasing function.[7,12,14,16]

Indicators of success

In this study, significant results were shown on analysis of VISA question 3 (pain with non-weight-bearing knee extension), question 6 (pain with 10 single-leg hops), and question 1 (number of minutes of pain-free sitting). The greatest improvements in subjects occurred at 3 weeks to 4 weeks after the five treatment sessions. This differs from other research on ESWT, which finds the most significant improvements at least 12 weeks after five treatment sessions.[12,15] This may suggest that more than five treatments should be administered and that a strength training program should be used in addition to ESWT to maintain the benefits of the shock wave therapy. Furthermore, subjects may have decreased their activity during the treatment period but then felt their improvements allowed them to increase their activity, meaning that at 12 weeks posttreatment they were stressing the knee to a greater extent.

Along with the VISA scores, the vertical jump test results and the anecdotal results suggest an increase in power and function in subjects due to a decrease in pain and, possibly, stimulation of fibroblastic activity, which strengthens the tendon. The ultrasound results support Alfredson's research, which showed evidence of neovascularization at the tendon as well as decreased levels of glutamate receptors.[19] Perhaps the improvement with ESWT is a result of the disruption of the new vessels and/or pain receptors.

There has only been one report of side effects with ESWT in this study. One subject reported that the intensity of knee pain increased during the 3 weeks of treatment. However, upon evaluation 4 weeks after the first three treatment sessions, the subject's pain had subsided.

There are potential sources of error that could account for unexpected results according to the hypothesis. The VISA score may not adequately reflect the improvement or lack of improvement in pain and functional impairment. One of the treatment group subjects did not show an improvement in VISA score; in fact the subject's score decreased. However, the subject also reported anecdotally that pain had decreased from a rating of 9 out of 10 (where 10 is the most pain) pretreatment to 1 out of 10 posttreatment. Furthermore, the subject reported that time to onset of pain during activity had significantly increased.

The most useful indicator in this study may be question 8 on the VISA, which refers to the time to onset of pain and the degree of pain experienced while undertaking sport. However, this question still raises some issues: it accounts for 30 out of a total 100 points and therefore heavily penalizes subjects who experience *any* pain with activity. Many athletes possess the ability to play through pain, and although the pain may be sufficient to cause some people to stop training or competing, these athletes continue. Thus, there may not be a true reflection of the degree and the consequences of this pain in the subject's response. In retrospect, adding a subjective visual analog scale question may have provided a useful indicator of pain relief and, therefore, improvement.

Conclusion

Although extracorporeal shockwave therapy appears to be a useful adjunct to the eccentric drop-squat strength protocol in the treatment of chronic patellar tendinopathy, further research is needed to determine the most efficient ESWT energy density, the ideal number of impulses, and the ideal follow-up time. In addition, more research is needed to determine the mechanism of pain relief of shock wave therapy on patellar tendinopathy. The next study should include a larger subject pool, a long-term follow-up, as well as a follow-up of successfully treated subjects to determine, using a color Doppler ultrasound, whether there is an elimination of the areas of neovascularization. Finally, a reliable objective measure of the microscopic and macroscopic improvement of the patellar tendon itself would be useful in evaluating the effects of shock waves on patellar tendinopathy.

Competing interests

Dr Taunton has a nominal investment in the local Sonocur machine.

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