JIANM

Journal of the International Academy of Neuromusculoskeletal Medicine

Volume 19

Issue 2

December 2022

JIANM

Journal of the International Academy of Neuromusculoskeletal Medicine

The Open Access, Peer-Reviewed, and Indexed Publication of the International Academy of Neuromusculoskeletal Medicine

Journal of the International Academy of Neuromusculoskeletal Medicine

December 2022 – Volume 19, Issue 2

Original Articles

Point-of-Care Musculoskeletal Ultrasound in the Diagnosis of Tibialis Posterior Partial Tendon Tear: A Case Report

Brandon Ramakko DC, MS, DIANM[1]

[1] DaVinci Wellness Centre, Georgetown, Grand Cayman

brandonramakko@gmail.com

Published: December 2022
Journal of the International Academy of Neuromusculoskeletal Medicine
Volume 19, Issue 2

ABSTRACT

Introduction: This report aims to illustrate the use of point-of-care ultrasound in aiding the diagnosis of a partial tibialis posterior tendon tear, a sometimes elusive diagnosis.

Case Presentation: A 31 year old male presented days after the acute onset of intense pain and edema of the medial ankle initiated by a "short jog across the street". Patient had a history of posterior and medial ankle pain following increased activity starting approximately 2 months prior.

Diagnosis: Point-of-care ultrasound showed edema surrounding the tibialis posterior tendon at the site of maximal tenderness located inferior to the medial malleolus and partial fiber disruption of the tendon was found more proximally, posterior to the medial malleolus, indicating a partial tendon tear.

Conclusion: Point-of-care ultrasound can identify a partial tendon tear of tibialis posterior and can be used for reassessment, as needed, throughout the recovery process.

Key Words: Tendinopathy, Tibialis Posterior, Point-of-care ultrasound, POCUS.

INTRODUCTION

Tibialis posterior tendinopathy is an often overlooked diagnosis.[1,2] Differential diagnosis of posterior tibialis tendinopathy can be challenging as pain along the tendon's path may be interpreted as Achilles tendinopathy, deltoid ligament sprain, or plantar fasciitis.[1] An acute full rupture requires surgical consult, but due to the difficulty in diagnosis, in one study, it was found that 88% of patients were initially misdiagnosed, and waited an average of 43 months to receive the correct diagnosis.[3] As the tibialis posterior muscle is partly responsible for maintaining the medial longitudinal arch, adult acquired pes planus may be due to tendon rupture or the more chronic degenerative condition, named "posterior tibial tendon dysfunction" (PTTD).[4] The clinical presentation of tibialis posterior tendinopathy may include: eversion ankle injury, generalized medial ankle pain, medial ankle swelling, flexible and asymmetric pes planus with forefoot pronation, and tenderness along the tendon's path.[2,4] It would be helpful to be able to screen patients with medial ankle pain for tibialis posterior tendinopathy in a cost-effective and time efficient manner and, in particular, rule out full rupture. Point-of-care musculoskeletal (MSK) ultrasound may be the solution to this problem.

The use of musculoskeletal (MSK) ultrasound is growing rapidly.[5] The advent of high frequency portable ultrasounds are making it easier for chiropractors and physiotherapists to offer point-of-care MSK diagnostic ultrasound. It does not irradiate the patient and it can provide imaging of joint effusion, edema, tendons, nerves, and muscles, which may have previously required sending the patient out for a costly MRI. Ultrasound is dynamic; you can apply pressure with the probe or move a nearby joint to assess the response of the tissue. Fiber disruption and/or visualizing the separation of tendon fibers with joint movement would indicate a tear. Increased blood flow can be assessed with color or power doppler settings. Increased thickness and a darker (hypoechoic) appearance could be used as an indicator of tendinosis. Tibialis posterior tendinopathy is well suited to evaluation via ultrasound.[6-8] With the appropriate ultrasound system and a sufficiently skilled technician/clinician, ultrasound may even be superior to MRI for the evaluation of the tibialis posterior tendon.[9]

CASE PRESENTATION

The patient initially presented for physiotherapy treatment of bilateral ankle pain which began after a return to tennis following several years of reduced activity. Pain was at its worst in the first 10-15 minutes of the day and after tennis. He found ¾ length insoles mildly helpful. The chart notes from the physical exam reported: R ankle edema, tenderness over all ligaments and soft tissues, and bilateral mid Achilles tenderness. Treatment over 2 visits consisted of calf raises, calf stretches, taping the midfoot, and new insoles. The patient reported notably reduced pain following treatment.

Approximately 1 month later, a "jog across the street" triggered intense pain and edema of the medial ankle. At the hospital he received a cortisone injection and a radiograph which, the patient reported, "found nothing wrong". Following the cortisone injection he found the swelling and pain had reduced almost entirely but partially returned a day later.

A couple days after the incident he presented for chiropractic care to address back and neck stiffness before flying out on vacation. Although he was not anticipating, nor searching for, aid with his ankle issue, a small amount of time was allocated to assessing the ankle, particularly via ultrasound imaging. He could bear weight and walk with mild pain at the medial ankle. The point of maximal tenderness was inferior to the medial malleolus. Ultrasound imaging was then performed with a handheld Clarius L15HD3 (Vancouver, BC, Canada), taking approximately 5 minutes to perform. The assessment began over the site of maximal tenderness, the inferior medial malleolus. Edema was noted between the tibialis posterior and flexor digitorum longus tendons (**Figure 1**). Toe flexion was tested and was strong and pain free. The tibialis posterior tendon was followed distal to the navicular which showed no pathology. Following the tendon more proximally to the posterior medial malleolus revealed some fiber disruption, indicating a partial tendon tear (**Figure 2**).

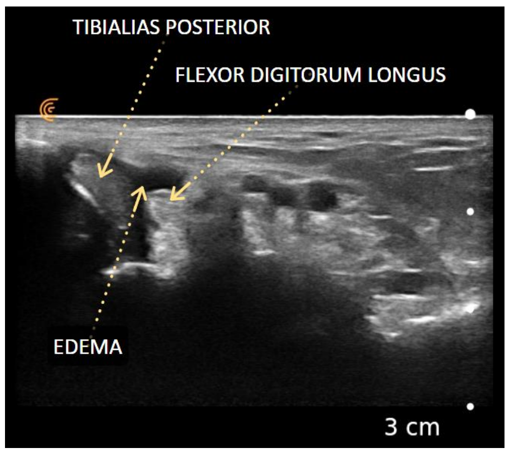

Figure 1: Transverse image of the tibialis posterior tendon in the tarsal tunnel.

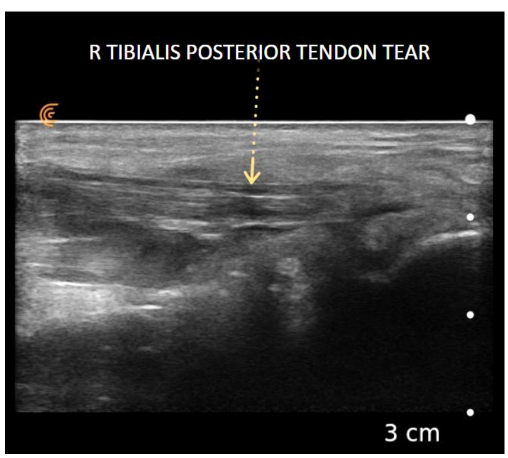

Figure 2: Longitudinal view of the right tibialis posterior tendon posterior to the medial malleolus. Note the fiber disruption indicating a partial tendon tear.

DISCUSSION

While a sudden increase in activities such as tennis could cause plantar fasciopathy or Achilles tendinopathy, the tibialis posterior tendon is another implicated structure. Any or all of the structures may be involved as well as relevant ligaments. The initial presentation to the physiotherapist matches well with the clinical picture of tibialis posterior tendinopathy and/or a combination of pathologies. In particular, the relief with an insole suggests the tibialis posterior tendon as it is partly responsible for maintaining the medial longitudinal arch.[6] The tibialis posterior tendon may have developed a tendinosis leaving it weakened such that a minor increase in load, such as a jog across the street, resulted in a sudden tear. Ultrasound can identify if a full rupture is present which helps ascertain the need for referral.[6]

This chiropractic visit was not an ideal scenario for an ankle assessment. The patient was seeking treatment for back and neck stiffness and not help with his ankle pain, making time spent diagnosing the ankle limited. MSK ultrasound, like radiographs, CT scans, or MRIs, are not a replacement for a proper history and physical exam. Considering how quickly and easily a scan can be performed with a handheld scanner, ultrasound was chosen, as it was deemed to be the single most valuable tool. Furthermore, it would be inappropriate to order an MRI, CT, or radiograph for every subsequent visit, but ultrasound is a safe, quick, and cost-effective imaging modality that can be used to reassess the state of the tendon, as needed, throughout the recovery process.[6-9]

CONCLUSION

Posterior tibialis tendinopathy can be challenging to diagnose, and the ability to evaluate the tendon within minutes via MSK ultrasound can aid in quickly identifying a partial tibialis posterior tendon tear. Point-of-care MSK ultrasound aids in increasing diagnostic accuracy which allows for proper management, and can be used, as needed, for reassessment.

CONSENT

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

COMPETING INTERESTS

The author declares no competing interests.

REFERENCES

1. Marcus RE, Pfister ME. The enigmatic diagnosis of posterior tibialis tendon rupture. *Iowa Orthop J.* 1993;13:171-7.

2. Foster AP, Thompson NW, Crone MD, Charlwood AP.. Rupture of the tibialis posterior tendon: an important differential in the assessment of ankle injuries. *Emergency Medical J.* 2005(4):915-6.

3. Mann FA, Thompson FM. Rupture of the posterior tibial tendon causing flatfoot. *J Bone Joint Surg* 1985;67A:556–61.

4. Johnson KA, Strom DE. Tibialis posterior tendon dysfunction. *Clin Orthop Relat Res.* 1989;(239):196-206.

5. Sharpe RE, Nazarian LN, Parker L, Rao VM, Levin DC. Dramatically increased musculoskeletal ultrasound utilization from 2000 to 2009, especially by podiatrists in private offices. *J Am Coll Radiol.* 2012 Feb;9(2):141-6. doi: 10.1016/j.jacr.2011.09.008.

6. Lhoste-Trouilloud A. The tibialis posterior tendon. *J Ultrasound.* 2012 Feb;15(1):2-6. doi: 10.1016/j.jus.2012.02.001.

7. Chen YJ, Liang SC. and Diagnostic efficacy of ultrasonography in stage I posterior tibial tendon dysfunction: sonographic-surgical correlation. *J Ultrasound Med.* 1997;16:417– 423. https://doi.org/10.7863/jum.1997.16.6.417

8. Neville C, Meyers K, Hojnowski MD. Ultrasound Assessment of the Tibialis Posterior Tendon. *J Ortho Sports Phys Ther.* 2010; 40(10):667-667. https://www.jospt.org/doi/10.2519/jospt.2010.0417

9. Arnoldner MA, Gruber M, Syré S, et al. Imaging of posterior tibial tendon dysfunction—comparison of high-resolution ultrasound and 3T MRI. *Eur J Radiol.* 2015;84:1777– 1781. https://doi.org/10.1016/j.ejrad.2015.05.021

Rapid Reduction of Gluteal Tendinopathy Symptomatology Utilizing Cold Laser and Sacroiliac Joint Manipulation: A Case Report

Sean Norkus DC, MS, DIBCN, DIBE[1]

Paige Lynch, DC[2]

[1] Palmer College of Chiropractic, Port Orange, FL
[2] Best Foot Forward Chiropractic, Daytona Beach, FL

sean.norkus@palmer.edu

Published: December 2022
Journal of the International Academy of Neuromusculoskeletal Medicine
Volume 19, Issue 2

ABSTRACT

Objective: The purpose of this case report is to describe the results of a woman with pain involving an acute gluteal tendinopathy who was treated with spinal manipulation and cold laser therapy.

Methods: A 27-year-old female sought care at a chiropractic clinic for acute pain that was located in the left gluteal region. This pain developed following the reintroduction of roller skating into her physical activity regimen and was believed to be due to overuse. Following the physical examination, the patient was diagnosed with an acute gluteal tendinopathy and joint restriction of the left sacroiliac joint. The patient was treated twice over a period of two weeks, involving the application of high-velocity low-amplitude manipulation directed at the left sacroiliac joint along with cold laser therapy.

Results: Following this short course of care, the patient reported a complete reduction of her symptoms and improvement on her Lower Extremity Functional Scale score. These changes appeared to be ahead of the timeframe associated with symptom reduction currently described in the literature.

Conclusion: Cold laser therapy and sacroiliac joint manipulation was associated with favorable outcomes in this case of a young adult female with gluteal tendinopathy.

Key Words: Tendinopathy, Neuromusculoskeletal, Diagnosis, Gluteal Myalgia, Cold Laser, Photobiomodulation

BACKGROUND

Establishing a specific tissue-based diagnosis in a patient experiencing deep gluteal pain is often unrealistic, even for clinicians with a great deal of experience in musculoskeletal diagnosis.[1] It is important that clinicians attempt to be as precise as possible when isolating a pain generator, as various tissues in this region can produce overlapping symptoms. One such example would be the consideration of referred pain from the lumbar region.[2] Adding to the confusion of diagnosis in this region would be the fact that gluteal pain itself may mimic additional pathologies, due in large part to the complexities of both gluteal and pelvic anatomy.[3] Although rare, one such example of viscerally-generated sacroiliac pain would be pseudoaneurysm of the gluteal artery.[4] More commonly encountered conditions are also capable of inducing pain in in sacroiliac region, including osteoarthritis, sacroiliac joint dysfunction, and even hyperparathyroidism.[5]

Central to the proper patient treatment is the correct diagnosis. This correct diagnosis not only allows for adequate patient communication but is also instrumental in outlining the best care appropriate at the time. As low back pain has developed into one of the world's largest healthcare issues, prompt identification of the causative factors of acute low back issues may help the patient's pain from entering the chronic realm.[6]

CASE PRESENTATION

A 27-year-old female presented to a chiropractic clinic with a chief complaint of localized pain in her left gluteal region and lateral hip following several days of roller skating. The patient also reported both subjective weakness of her left gluteal musculature and pain during the stance phase of walking, which is in line with previous publications of gluteal tendinopathy.[3] Utilizing the Numeric Rating Scale (NRS), the patient's pain was reported at 5/10 during walking, lower extremity abduction, and lower extremity extension, and as 0/10 at rest. This patient also reported a history of surgical repair of a previously torn anterior cruciate ligament (ACL) and both the medial and lateral menisci of her left knee. This item is potentially relevant as it has been reported that the gluteus medius muscles of fifty percent of the patients that underwent this procedure were moderately smaller post-surgery.[7]

While a diagnosis of gluteal strain was considered, this was disregarded due to the location and presentation of the patient's symptoms. Specifically, the patient reported both lateral hip pain as well as pain reproduction when the area surrounding the greater trochanter was palpated, indicating that the pain was not due to gluteal strain.[8,9] Static and motion palpation of the left sacroiliac joint elicited localized joint pain and reproduced the patient's chief complaint. These findings led to a diagnosis of gluteal tendinopathy and gluteal myalgia due to repetitive stress injury. In an effort to improve the validity of the findings within the case, and to aid in future patient communication, the patient also completed a Lower Extremity Functional Scale (LEFS) questionnaire before beginning treatment. In this case, the clinician utilized OrthoToolKit,[10] which showed the patient's pre-treatment LEFS score was 65/80 (81.3%). A figure of the patient's pre-treatment OrthoToolKit LEFS results have been included in **Figure 1** to provide a visual representation of the score.

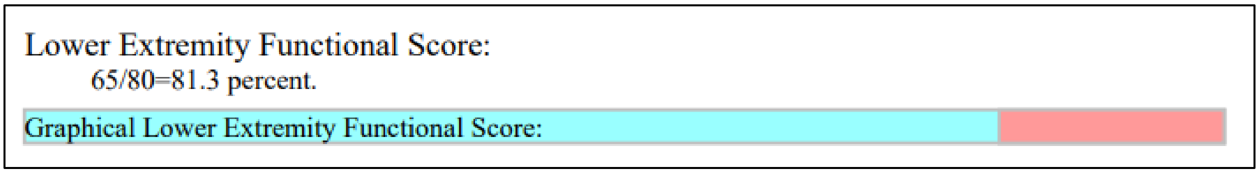

Figure 1 - Pre-treatment LEFS Score

The patient denied any recent trauma and no red flags were discovered that could suggest more serious pathology, so a trial of chiropractic care was initiated. At each treatment, the therapeutic interventions involved within this trial of care were high-velocity, low-amplitude sacroiliac joint manipulation and 10 W cold laser therapy with a LightCure LCT-1000 (New Castle, Delaware), which was applied directly over the regions of the gluteus maximus and medius insertions and the gluteal tuberosity of the femur. On each of two visits, the patient received left-sided sacroiliac joint manipulation and 3,000 joules of cold laser therapy (10W/5min). The sacroiliac joint manipulation was performed in a side-posture position and was intended to restore motion to the restricted joint. The patient tolerated all interventions well and denied adverse events following treatment. After each of the two treatments, the patient reported immediate improvement in her gluteal region pain. Following the patient's second visit, the NRS pain rating and Lower Extremity Functional Scale (**Figure 2**) outcomes were repeated. Due to substantial improvement the patient was released from care for this condition and instructed to return as-needed, should the condition return.

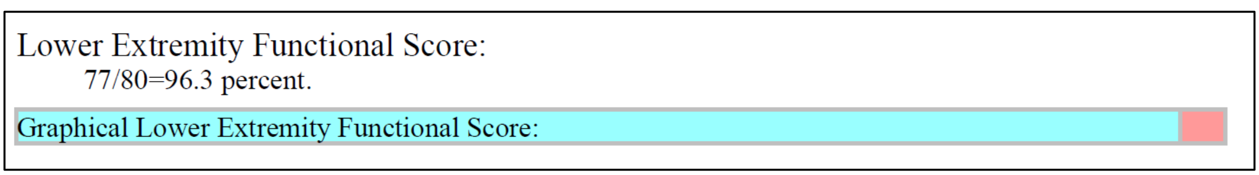

Figure 2 - Post-treatment LEFS score

DISCUSSION

Although the initial case presentation may have seemed straightforward, the treating physician was cautious in moving forward before considering the possibility of referred pain from more ominous and/or serious sources. As far back as 1982, literature can be found outlining the prevalence of roller-skating related injuries in both the experienced and non-experienced skater.[11] Specific to this case, gluteal tendinopathy is the most common of the lower limb tendinopathies.[8] Clinicians who are faced with the diagnosis of pain in the gluteal region should be cognizant of the wide variety of pain generators which have been found to cause pain in this region. These can include lumbar disc herniation, deep gluteal syndrome, and even vascular pathologies.[2,4,12] In line with the existing research, this case provides further evidence that cold laser can be a useful addition in the conservative management of tendinopathy.[13]

Of particular interest in this case was the rapid reduction of symptoms. Many individuals with gluteal pain have a long-term course of symptomatology due to difficulty in both location of the causative tissue and difficulty in therapeutic access of said tissue.[1] While the benefits of low level laser therapy have been documented in healthcare literature for decades, there is still a need for more documentation of the positive effects.[14] While minimal research exists on the use of cold laser as a therapy for gluteal tendinopathies, low level laser therapy and other types of photobiomodulation have been shown to promote healing by reducing swelling, inflammation, and pain in a variety of other musculoskeletal conditions.[15]

Through laying the anecdotal groundwork of the positive effect of some of these portal of entry accessible therapies, it is our hope that more researchers will be provided with the foundation to begin larger studies that can examine the mechanisms behind these changes at a more microscopic and quantifiable level. The Lower Extremity Functional Scale was created to objectively measure the functional abilities in those with a range of lower extremity conditions.[16] In this case, the utilization of the LEFS provided an opportunity to obtain objectively validated data to use as a measure of both treatment effectiveness and quantification of subjective patient reports.

CONCLUSIONS

Since at least 2015, tendinopathies of both the gluteus minimus and/or medius tendons has been established as the most frequently occurring pathology in those individuals with pain over the greater trochanteric region.[17] The authors hope that other practitioners who utilize low level laser therapy in their practices may be inspired to continue to help build the existing evidence for the use of this modality in other musculoskeletal conditions through publication of the treatment parameters that they have found to be most beneficial for a variety of conditions, both acute and chronic.

With the prevalence of validated outcome tools, it is becoming easier to chart the rapid improvement that patients may experience from the combination of therapies such as manipulation and cold laser. With the addition of outcome assessment tools to subjective patient reported information, case reports become a valid method of providing the background data to document the need for future larger-scale studies.

LIMITATIONS

Due to the fact that this is a single-patient case report, the results of this report cannot be generalized to other individuals presenting with similar conditions.

CONSENT

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the

Editor-in-Chief of this journal.

COMPETING INTERESTS

The authors declare that they have no competing interests.

ACKNOWLEDGMENTS

The authors would like to thank Sandra Lewis the Head Librarian at Palmer College of Chiropractic – Davenport Campus for her assistance in locating and retrieving some of the articles utilized in the writing of this case report.

REFERENCES

1. Gonzalez-Lomas G. Deep gluteal pain in orthopaedics: a challenging diagnosis. *JAAOS-Journal of the American Academy of Orthopaedic Surgeons*. 2021;29(24):e1282-e1290.

2. Wang Y, Yang J, Yan Y, et al. Possible pathogenic mechanism of gluteal pain in lumbar disc hernia. *BMC Musculoskeletal Disorders*. 2018-12-01 2018;19(1)doi:10.1186/s12891-018-2147-y

3. Franklyn-Miller A, Falvey E, McCrory P. The gluteal triangle: a clinical patho-anatomical approach to the diagnosis of gluteal pain in athletes. *British journal of sports medicine*. 2009;43(6):460-466.

4. Yurtseven T, Zileli M, Göker ENT, Tavmergen E, Hoscoskun C, Parildar M. Gluteal artery pseudoaneurysm, a rare cause of sciatic pain: case report and literature review. *Clinical Spine Surgery*. 2002;15(4):330-333.

5. Tuite MJ. Sacroiliac joint imaging. © Thieme Medical Publishers; 072-082.

6. Illes ST. Low back pain: when and what to do. *Orvosi Hetilap*. 2015;156(33):1315-1320.

7. Norte GE, Knaus KR, Kuenze C, et al. MRI-Based Assessment of Lower-Extremity Muscle Volumes in Patients Before and After ACL Reconstruction. *Journal of Sport Rehabilitation*. 2018;27(3):201-212.

8. Grimaldi A, Mellor R, Hodges P, Bennell K, Wajswelner H, Vicenzino B. Gluteal tendinopathy: a review of mechanisms, assessment and management. *Sports Medicine*. 2015;45(8):1107-1119.

9. Allison K, Hall M, Wrigley TV, et al. Gluteal tendinopathy and hip osteoarthritis: Different pathologies, different hip biomechanics. *Gait and Posture*. 2018;61:459-465. doi:10.1016/j.gaitpost.2018.02.011

10. OrthoToolKit. Free Online LEFS Score Calculator - OrthoToolKit. Accessed May 4, 2022, https://orthotoolkit.com/lefs/

11. Ferkel RD, Mai LL, Ullis KC, Finerman GA. An analysis of roller skating injuries. *The American Journal of Sports Medicine*. 1982;10(1):24-30.

12. Martin HD, Gómez-Hoyos J. Deep gluteal syndrome. *Posterior Hip Disorders*. Springer; 2019:167-187.

13. Tumilty S, Munn J, McDonough S, Hurley DA, Basford JR, Baxter GD. Low level laser treatment of tendinopathy: a systematic review with meta-analysis. *Photomedicine and laser surgery*. 2010;28(1):3-16.

14. Farivar S, Malekshahabi T, Shiari R. Biological effects of low level laser therapy. *Journal of lasers in medical sciences*. 2014;5(2):58.

15. De Oliveira MF, Johnson DS, Demchak T, Tomazoni SS, Leal-Junior EC. Low-intensity LASER and LED (photobiomodulation therapy) for pain control of the most common musculoskeletal conditions. *European journal of physical and rehabilitation medicine*. 2022;58(2):282-289. doi:10.23736/S1973-9087.21.07236-1

16. Mehta SP, Fulton A, Quach C, Thistle M, Toledo C, Evans NA. Measurement properties of the lower extremity functional scale: a systematic review. *The Journal of orthopaedic and sports physical therapy*. 2016;46(3):200-216.

17. Grimaldi A, Fearon A. Gluteal Tendinopathy: Integrating Pathomechanics and Clinical Features in Its Management. *The Journal of orthopaedic and sports physical therapy*. 2015;45(11):910-22. doi:10.2519/jospt.2015.5829

Multimodal Treatment of Neck Pain with Radiculopathy: A Case Report

Whitney Amos McNary, DC[1]

Amanda Vozar, DC[2]

Lamar Dulin, DC, EP-C[3]

[1] Palmer College of Chiropractic, Port Orange, FL
[2] Palmer College of Chiropractic, Port Orange, FL
[3]North Florida Medical Center, Jacksonville, FL

whitney.amosmcnary@palmer.edu

Published: December 2022
Journal of the International Academy of Neuromusculoskeletal Medicine
Volume 19, Issue 2

ABSTRACT

Objective: The purpose of this case study is to discuss a positive outcome of conservative therapy on a patient with idiopathic, multi-level radiating neck pain.

Clinical Features: A 52-year-old male presented to an outpatient chiropractic clinic with severe radiating neck pain for ten days with unknown mechanism of injury. Patient states he is experiencing numbness and tingling in the left arm into the thumb and index finger as well as decreased and painful cervical spine range of motion. Affected activities of daily living include cooking, sleeping, eating, driving, and working overhead. Cervical spine radiographs and cervical spine MRI were performed revealing multi-level disc herniations with degeneration as well as left lateral recess stenosis and a stenotic left neural foramen at the level of C5/C6.

Intervention and Outcome: Treatment included cervical flexion/distraction, class IV cold laser, therapeutic ultrasound, axial decompression, shockwave, active rehabilitation, and thoracic spinal adjustments. After 6 months of treatment, the patient's VAS score decreased

from a 9/10 to a 2/10. Patient had visualized increased range of motion and no longer had any affected activities of daily living.

Conclusion: In this case report, a patient benefited from a multimodal approach to conservative care. This suggests that combination therapy can be effective for patients with multi-level disc herniations and associated radiculopathy. The result of decreased VAS, visualized increase in range of motion, along with improvement in activities of daily living demonstrate important functional improvement in this patient. Further study should be done on combinations of the chosen modalities to evaluate their combined effectiveness.

INTRODUCTION

Cervical radiculopathy is most prevalent in persons 50 to 54 years of age with an annual incidence of 107 per 100,000 men, and 64 per 100,000 women.[1] Previous instances of lumbar radiculopathy, cigarette smoking, and axial load bearing can increase the likelihood of experiencing cervical radiculopathy.[2] It is described as an impingement and/or irritation of cervical spinal nerve and/or nerve root typically characterized by unilateral shooting electric pain in the upper limb, neck pain and sensory, motor and/or reflex deficits.[3] Irritation and compression of cervical spinal nerve and/or nerve root can be due to any of the following: narrowing of intervertebral foramen (decreased disk height or degenerative joint changes), intervertebral disc herniation, infection, inflammatory exudate, physical injury or trauma, or spinal tumors.[1]

Patients presenting with cervical radiculopathy commonly experience painful neck movements and muscle spasms upon examination with or without accompanied motor, sensory, or reflex deficits. [1] The most common objective neurologic finding is diminished deep tendon reflexes, particularly of the triceps followed by weakness in the C6 and/or C7 innervated muscles.[1] However, clinicians need to keep in mind each patient may present differently. To confirm such diagnosis the Spurling test, shoulder abduction test, and the upper limb tension test can be performed.[1] Typically, imaging is not required unless there is a history of trauma, persistent symptoms, or red flags, however cervical spine magnetic resonance imaging (MRI) can be helpful to confirm diagnosis.[1,2] If peripheral neuropathy of the upper extremity is included in the differential diagnosis, electrodiagnostic testing can be considered.[1]

Nonoperative treatment options for patients with cervical radiculopathy include rest, cervical collar, physical therapy encouraging strengthening, stretching and consideration of traction therapy, massage, as well as oral medication such as corticosteroids dose-pack, non-steroidal anti-inflammatory drugs, Tramadol, and muscle relaxants.[1,2,4] For patients with red flag signs or symptoms that are persistent after four to six weeks of conservative care, an MRI may identify any pathology that may be considered appropriate for epidural steroid injection or surgery.[1] However, these treatment options have higher risks for complications compared to the nonoperative options.[1,2] This case reviews a nonoperative, multimodal treatment approach for a 52-year-old male with acute neck pain with radiating symptoms into the left upper extremity.

CASE PRESENTATION

A 52-year-old male sought care at an outpatient chiropractic clinic for neck pain with severe radiating numbness and tingling symptoms into the left upper extremity to the thumb and index finger with an 8/10 on the Visual Analogue Scale (VAS) during rest and 10/10 during activity. Patient reported the symptoms were present for approximately ten days with no known mechanism of injury. Patient was seen by another chiropractor three times for cervical region chiropractic manipulative therapy (CMT), resulting in no change in symptomatology. Patient reported he was a current smoker, employed as a heavy laborer with construction work, and a had history of IV drug use. **Table 1** indicates relevant cervical spine examination findings. Initial differential diagnosis included C5 nerve root irritation from facet encroachment or herniated disc.

Table 1	
Cervical Active Range of Motion (AROM)	Findings
Flexion: Extension: Right Lateral Flexion: Left Lateral Flexion: Right Rotation: Left Rotation:	normal and pain free with pain, severely decreased with pain, moderately decreased with pain, moderately decreased with pain, moderately decreased with pain, moderately decreased
Cervical Passive Range of Motion (PROM)	
Flexion: Extension: Right Lateral Flexion: Left Lateral Flexion: Right Rotation: Left Rotation:	normal and pain free with pain, moderately decreased with pain, moderately decreased with pain, moderately decreased with pain, moderately decreased with pain, moderately decreased
Neurologic	
Motor/Myotome: Deep Tendon/Pathological Reflexes: Sensory/Dermatome:	C5 left graded 2/5 Biceps (C5) left graded 1+ C5/C6 left decreased
Orthopedic	
Bakody's Sign: Foraminal Compression: Maximum Foraminal Compression: Cervical Distraction: Shoulder Depression:	+ left for radicular pain + left for radicular pain + left for radicular pain + left for radicular pain + left for radicular pain

Following a thorough history and examination with the presence of red flags including pain not improving with rest, over age 50, previous IV drug use, and high risk for psychosocial overlay, the patient was referred for Davis series radiographs **(Figures 1 and 2)** which revealed stenosis of the left C5/C6 neural foramen. After one month of treatment cervical MRI was ordered which revealed multi-level disc herniations with degeneration as well as left lateral recess stenosis and a stenotic left neural foramen at the level of C5/C6.

Figure 1: Oblique Cervical Spine

Figure 2: Lateral Cervical Spine

The first course of treatment consisted of cervical flexion/distraction (F&D) coupled with class IV cold laser over the C5/C6 region for eight visits; at which time both services were discontinued due to lack of positive response by the patient. Cervical axial decompression was then implemented with a gradual weight increase (19 - 36lbs of pressure) through two decompression protocols along with at-home cervical ROM exercises including cervical retractions, cervical flexion, extension, left and right rotation, and left and right lateral flexion. During this time, decompression was coupled with heat, which was replaced with therapeutic ultrasound, and then replaced with shockwave to help with muscle spasms in the left upper thoracic musculature. Patient began active rehabilitation with the addition of cervical retraction exercises with the goal to strengthen the anterior cervical musculature, which resulted in significant pain reduction. Treatments are described below in **Table 2**.

Table 2

Visit #	Cervical F&D	Laser Therapy	Axial Traction	Heat	Ultra-sound	Shock-wave	CMT	Rehab	E-Stim	Trigger Point Therapy
1,4,5	X	X								
Cervical F/D protocol 1 with 5 minutes of cold laser therapy over posterior cervical spine										
2,7,8	X									
Cervical F/D protocol 1										
3	X		X							
6	X								X	
9-17,19			X					X		
Cervical axial traction with 19 pounds of pressure that was incrementally increased to 36 pounds of pressure over 9 treatments and an angle of 19 degrees										
18 Re-eval							X			
20			X	X				X		
21-34			X	X						
Cervical axial traction with 36 pounds of pressure at 19 degrees with 10 minutes of moist heat to the cervical spine										
35-37			X	X	X					
Cervical axial traction with 36 pounds of pressure at 19 degrees with 8 minutes of therapeutic ultrasound to posterior cervical region and mid-scapular region bilaterally										
38-43, 45-47, 49-50, 53,55,62, 64,67			X		X	X				
Cervical axial traction with 36 pounds of pressure at 20 degrees for 15 minutes, 10 minutes therapeutic ultrasound to posterior cervical region and mid-scapular region bilaterally, 6 minutes extracorporeal shockwave therapy at 2000 pulses performed on middle and left lower trapezius										
44 Re-eval			X		X	X	X			X
48,52,54, 57,61,63, 68							X			X
Chiropractic manipulative therapy to thoracic spine using Gonstead technique with 8 minutes trigger point therapy to left upper, middle, and lower trapezius and left rhomboid										
51			X		X					X
56,58-60, 65-66,70			X		X	X		X		
Cervical axial traction with 36 pounds of pressure at 20 degrees for 15 minutes, 6 minutes extracorporeal shockwave therapy at 2000 pulses performed on middle and left lower trapezius, 3 sets of 15 supine cervical retractions, 3 sets of 15 all cervical range of motion exercises										
69			X			X				

Due to complicating factors such as the patient trying to work through the pain as a carpenter without not taking time off, patient age (55 years old), and past medical history (previous IV drug user and recent hospital stay for elevated ammonia levels), reduction of symptoms took longer than expected. After 6 months of treatment, the patient's VAS score decreased from a 9/10 to a 2/10. The patient had observed increased cervical range of motion and no longer had any affected activities of daily living. Treatment resulted in a reduction of symptoms and the patient was able to fully function at work.

DISCUSSION

Cervical radiculopathy symptoms typically occur unilaterally and are dependent on the involved nerve root.[2] The most commonly affected nerve roots are C6-C7 which can affect the first three digits of the hand.[2] Cervical radiculopathy may present with or without neck pain, can be accompanied by shoulder pain, and can even be absent of radiating pain. Pain is not always the presenting complaint as motor, sensory, and/or reflex deficits can present without pain.[2]

This particular case stands out due to the patient’s presenting symptoms of severe pain with neurological deficits along with a high risk of psychosocial overlay, current smoker, employed as a heavy laborer with construction work, and a history of IV drug use. Despite these complicating factors, the patient was able to steadily progress and eventually make a full resolution. The patient’s compliance with care is a strength in this case and allowed for consistent treatment with flexion distraction, cold laser, ultrasound, shockwave, decompression, and at-home exercises.

The effectiveness of flexion distraction therapy for cervical radiculopathy literature is limited to case studies and retrospective reviews with no recent studies.[5] However, with the studies that have been performed, clinical success has been found using the flexion distraction technique.[5] More scientific studies of efficacy and results from utilization of flexion distraction technique would provide a benefit in assessing long-term outcomes and safety.[5] Flexion distraction has been shown to decrease the intradiscal pressure, increase the intervertebral disc height and area of the intervertebral foramen, as well as restore physiologic motion.[5] A case series that followed 27 patients with similar treatment as this case report consisting of flexion distraction, CMT, physiotherapy, and rehabilitative exercises found 80% had a favorable clinical outcome with improvement of symptoms.[6]

A double-blind placebo controlled randomized study in 2010 showed that patients who received low-level cold laser therapy had greater improvement with measured flexion and extension of the cervical spine, better pain reduction, and greater improvement with quality of life than those who received a placebo procedure.[7] Another study in 2017 also showed therapeutic ultrasound to have similar results with a decrease in cervical radiculopathy symptoms and an improvement in quality of life.[8] These therapies were each performed individually, more studies with combined therapies are needed to demonstrate a possible increase in efficacy when performed together.

Cervical traction decompression therapy, manual therapy, shockwave therapy, and rehab exercises for cervical radiculopathy have little to no research to show their effectiveness individually. However, for this particular patient manual therapy and shockwave therapy were used to help decrease trigger points found the upper thoracic musculature. One systemic review and meta-analysis found that patients who received shockwave therapy in their trapezius musculature experienced pain relief in that region, however there is no evidence supporting that shockwave therapy is superior to other modalities in providing pain relief.[9] A case series suggests that implementing deep neck flexor and scapulothoracic strengthening exercises may be a suitable strategy for patients with cervical radiculopathy.[10] In this case report the patient did show improvement during the decompression protocol while receiving cervical exercises and manual therapy. This is similar to the results of a systemic review and meta-analysis from 2018 that found that mechanical traction had significant results with pain reduction when added to other modality procedures.[11]

There is no consensus for treatment of cervical radiculopathy with little high-quality evidence for the best treatment.[12,13] The literature on nonoperative treatment for cervical radiculopathy seems to point towards a favorable outcome.[12] The effectiveness of the individual treatments is disputable; however, a multimodal treatment approach may be beneficial for patients experiencing cervical radiculopathy.[12] The literature tends to align with the results of this case showing a positive outcome with flexion distraction, cervical mechanical traction/decompression, cold laser, and cervical strengthening exercises for cervical radiculopathy.

CONCLUSION

Cervical radiculopathy occurs in many different patient populations. It can have varying presentations and MRI should only be performed under certain circumstances. Not all patients with cervical radiculopathy present with the same symptoms or deficits. Effective management of cervical radiculopathy may include many modalities coupled throughout the treatment plan. The conservative care used in this case had positive outcomes with results consisting of decreased VAS, observed increase in range of motion, along with improvement in activities of daily living, constituting a successful treatment for this particular patient.

CONSENT

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

REFERENCES

1. Childress MA, Becker BA. Nonoperative Management of Cervical Radiculopathy. *Am Fam Physician*. 2016 May 1;93(9):746-54. PMID: 27175952. Accessed June 21, 2022. https://aafg.org/pubs/afp/issues/2016/0501/p746.html

2. Corey DL, Comeau D. Cervical Radiculopathy. *Medical Clinics of North America*. July 2014, Volume 98, Issue 4, Pages 791-799 ISSN 0025-7125, ISBN 9780323311656, https://doi.org/10.1016/j.mcna.2014.04.001.

3. DynaMed. Cervical Radicular Pain and Radiculopathy. EBSCO Information Services. Accessed June 21, 2022. https://www.dynamed.com/condition/cervical-radicular-pain-and-radiculopathy

4. Saal JS, Saal JA, Yurth EF. Nonoperative Management of Herniated Cervical Intervertebral Disc with Radiculopathy. *Spine*: August 15, 1996 - Volume 21 - Issue 16 - p 1877-1883. Accessed on June 24, 2022. https://journals.lww.com/spinejournal/Fulltext/1996/08150/Nonoperative_Management_of_Herniated_Cervical.8.aspx

5. Gudavalli S, Kruse RA. Foraminal stenosis with radiculopathy from a cervical disc herniation in a 33-year-old man treated with flexion distraction decompression manipulation. *Journal of manipulative and physiological therapeutics*. 2008;31(5):376-380. doi:10.1016/j.jmpt.2008.04.002

6. Eubanks JD. Cervical radiculopathy: nonoperative management of neck pain and radicular symptoms. *American family physician*. 2010;81(1):33-40. Accessed June 20, 2022. https://search-ebscohost-com.palmer.idm.oclc.org/login.aspx?direct=true&db=mdc&AN=20052961&site=ehost-live

7. Konstantinovic L, Cutovic M, Milovanovic A, Jovic S, Dragin A, Letic M, Miler V,. Low-Level Laser Therapy for Acute Neck Pain with Radiculopathy: A Double-Blind Placebo-Controlled Randomized Study, *Pain Medicine*, Volume 11, Issue 8, August 2010, Pages 1169–1178, https://doi.org/10.1111/j.1526-4637.2010.00907.x

8. Metin Ökmen B, Ökmen K., Altan L. Investigation of the effectiveness of therapeutic ultrasound with high-resolution ultrasonographic cross-sectional area measurement of cervical nerve roots in patients with chronic cervical radiculopathy: a prospective, controlled, single-blind study. *Journal of Medical Ultrasonics.* 45, 479–486 (2018). https://doi.org/10.1007/s10396-017-0855-9

9. Zhang Q, Fu C, Huang L, Xiong F, Peng L, Liang Z, Chen L, He C, Wei Q. Efficacy of Extracorporeal Shockwave Therapy on Pain and Function in Myofascial Pain Syndrome of the Trapezius: A Systematic Review and Meta-Analysis. *Archives of Physical Medicine and Rehabilitation.* 2020 Aug;101(8):1437-1446. doi: 10.1016/j.apmr.2020.02.013. Epub 2020 Mar 28. PMID: 32234411.

10. Cleland JA, Whitman JM, Fritz JM, Palmer JA. Manual physical therapy, cervical traction, and strengthening exercises in patients with cervical radiculopathy: a case series. *Journal of Orthopaedic and Sports Physical Therapy.* 2005 Dec;35(12):802-11. doi: 10.2519/jospt.2005.35.12.802. PMID: 16848101.

11. Romeo A, Vanti C, Boldrini V, Ruggeri M, Guccione AA, Pillastrini P, Bertozzi L. Cervical Radiculopathy: Effectiveness of Adding Traction to Physical Therapy-A Systematic Review and Meta-Analysis of Randomized Controlled Trials. *Physical Therapy Journal.* 2018 Apr 1;98(4):231-242. doi: 10.1093/physth/pzy001. Erratum in: Phys Ther. 2018 Aug 1;98(8):727. PMID: 29315428.

12. BenEliyahu DJ. Magnetic resonance imaging and clinical follow-up: study of 27 patients receiving chiropractic care for cervical and lumbar disc herniations. *Journal of manipulative and physiological therapeutics*.1996 Nov-Dec;19(9):597-606. PMID: 8976479. Accessed on July 12, 2022. https://pubmed.ncbi.nlm.nih.gov/8976479/

13. Akkan H, Gelecek N. The effect of stabilization exercise training on pain and functional status in patients with cervical radiculopathy. *Journal of Back Musculoskeletal Rehabilitation*. 2018;31(2):247-252. doi: 10.3233/BMR-169583. PMID: 28946516.

The Effect of Schroth Exercises on Cobb Angle, Quality of Life, and Functional Capacity in Adolescent Idiopathic Scoliosis: A Systematic Review of Randomised Controlled Trials

Joe H. Ghorayeb, DC, MHA[1]

Hiba S. Ayoub, HBSc[2]

Rashid Darwish, DC[3]

Pier-Paul Palombo, DC[4]

[1] University of Medicine & Health Sciences, New York, NY
[2]University of Toronto, Toronto, ON
[3]Private Practice, Richardson, TX
[4]Private Practice, London, ON

jghorayeb@umhs-sk.net

Published: December 2022
Journal of the International Academy of Neuromusculoskeletal Medicine
Volume 19, Issue 2

ABSTRACT

Background: Adolescent idiopathic scoliosis (AIS) is the most common form of spinal curve severity affecting individuals 10 to 18 years of age. Surgery is generally reserved for curves measuring >50°, whereas bracing and scoliosis-specific exercises are recommended for Cobb angles ranging from 10° to 45° in order to prevent curve progression. As the utility of Schroth exercises has emerged as a popular option among practising clinicians, the purpose of this systematic review is to determine the effect of Schroth exercises on Cobb angle progression, quality of life (QoL), and functional capacity in individuals with AIS.

Methods: Three databases, MEDLINE, PEDro, and CINAHL were systematically searched for English-language randomised controlled trials investigating the effect of Schroth exercises on individuals with AIS with Cobb angles ≥ 10°. The outcomes of interest were the effect of Schroth exercises on Cobb angle, QoL, and functional capacity when compared to standard nonoperative care.

Results: Five RCTs were appraised. Two studies showed no meaningful change in Cobb angle that may be accounted for outside of measurement error alone. One study showed some change beyond measurement error, but there was no follow-up beyond 12 weeks, and another showed some change at 26 weeks that may be attributable to the intervention (p=0.001). Only one study found significant improvements in QoL at 10 weeks (p<0.001). Of the two studies assessing functional ability, one study found no significant difference at 6 months follow-up (p=0.89), whereas the other study found significant improvement in 6-minute walk test (6MWT) at 6 months (p=0.001).

Conclusion: Insufficient evidence exists to suggest that engaging in Schroth exercises results in halting curve progression or regression of Cobb angle in AIS patients in the long-term. Improvement in quality of life and functional capacity may be realized in the short-term, though maintenance of such effects remains to be elucidated. Future studies with larger sample sizes and longer intervention and follow-up periods are necessary to determine the long-term effect of Schroth exercises on Cobb angle, QoL, and functional capacity in individuals with AIS.

Key words: Adolescent idiopathic scoliosis; Rehabilitation; Schroth exercise; Cobb angle; Quality of life; Functional capacity

INTRODUCTION

Adolescent idiopathic scoliosis (AIS) is the most common form of spinal curve asymmetry affecting individuals between 10 to 18 years of age.[1] AIS is characterised by lateral deviation, axial rotation, and abnormal sagittal curvature of the spine, and is defined as a coronal curvature measuring at least 10°. The prevalence of AIS has been reported to range between approximately 0.5 to 5% worldwide.[2-9] Girls and boys are equally likely to present with minor curves of roughly 10°, but females are much more likely to progress to more severe curvature.[10,11] The mean lifetime prevalence of low back pain in AIS patients is reportedly 40%.[12] The degree of curvature and accompanying truncal asymmetry can also lead to psychological disturbances in the adolescent population.[13]

Though the exact etiology of AIS remains to be elucidated, genetic factors have been implicated in the development and progression of the condition.[14-16] Standing posterior-anterior and lateral full spine radiographs, including the pelvis, are useful for measurement of the Cobb angle from the most tilted caudal and cephalic vertebrae toward the concavity in order to quantify the curvature. The main determinants of curve progression include curve location and magnitude, skeletal maturity based on age and Tanner stage, onset of menses, closure of the triradiate cartilage, Risser sign, and Sanders score.[17] Of these factors, the initial Cobb angle magnitude and skeletal maturity status show the highest correlation to curve progression.[18,19]

The International Scientific Society on Scoliosis Orthopaedic and Rehabilitation Treatment (SOSORT) classifies AIS by way of Cobb angle as follows: up to 20°, low; 21°-35°, moderate; 36°-40°, moderate to severe; 41°-50°, severe; 51°-55°, severe to very severe; and ≥56°, very severe.[20] The risk of Cobb angle progression in adulthood increases with baseline measurements over 26°.[18] This degree of curvature also increases the risk of health problems and reduction of quality of life (QoL). Reduced exercise tolerance may be evident in patients with moderate to severe curves.[21] Pulmonary impairment may be significant in individuals with Cobb angles >80° and in cases where thoracic rotation is present.[13]

Surgical intervention is generally reserved for curves measuring >50°, whereas bracing and scoliosis-specific exercises are recommended for Cobb angles ranging from 10° to 45° in order to help prevent curve progression.[22] Because bracing, either part-time or full-time, until skeletal maturity is reached is difficult and necessitates compliance[23], scoliosis-specific exercise therapy has been utilised as an adjunct to, and a means of increasing compliance with, brace treatment.[24] Of the various exercise protocols employed in the treatment of individuals with AIS, the utility of Schroth exercises has emerged as a popular option among practising clinicians.[25]

The Schroth method originated in Germany in the 1920s by Katharina Schroth, who developed curve-specific exercises to manage her own scoliosis.[24] In this method, the ideals of de-rotating, elongating, and stabilising the spine in all three anatomical planes are addressed in addition to the consideration of muscular symmetry, rotational angular breathing, and awareness of posture through stretching and exercising specific muscles.[26] Given the popularity and reported success of the Schroth Method in community practice[25], a previous systematic review and meta-analysis conducted by Burger et al.[27] concluded that Schroth exercises have a significant effect on reducing the Cobb angle and improving QOL in adolescents with idiopathic scoliosis, however the data presented in that review contradicts the aforementioned conclusion, with reported Cobb angle improvements of 1.2°, which do not reflect a minimal clinically important difference (MCID) in this context.[28] Moreover, contrary to the aim of the aforementioned review, the authors did not focus exclusively on randomised controlled trials, as they included a comparative trial by Kim and Hwangbo[29], which compared Schroth exercises to Pilates in the absence of a control group.

The aim of this systematic review is to provide a more accurate reflection of the effectiveness of the Schroth method by identifying, critically appraising, and evaluating the best available evidence regarding the impact, if any, of Schroth exercises on Cobb angle progression/reduction, in addition to the effects on QoL and functional capacity when compared to standard nonoperative care (e.g., observation and/or bracing) in patients with AIS.

METHODS

Registration and Reporting

We registered the review with the International Prospective Register of Systematic Reviews (PROSPERO) on May 10, 2022 (CRD42022329295). Our systematic review complies with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement.[30,31]

Search Strategy

A systematic search strategy was designed and executed from inception through May 6, 2022 using the following electronic databases: MEDLINE, PEDro, and CINAHL. The search terms included subject headings specific to each database (e.g., MeSH in MEDLINE) (**Appendix 1**) and free text words relevant to Schroth exercises as an intervention for AIS and the effect on Cobb angle and QoL in the other search databases. Search entries were exported to EndNote X9 for reference management and tracking of the screening process.

Study Selection

A two-phase approach to screening was used with two independent reviewers screening each citation and article. Phase one included screening of titles and abstracts for possible relevance. Phase two included full text screening of possibly relevant studies. Any disagreement was resolved by discussion between the paired reviewers. All relevant studies were independently critically appraised for risk of bias by the two reviewers using the PEDro scale. If there were disagreements in the risk of bias assessments between the reviewers, then a third reviewer independently assessed for risk of bias and discussed with the other two reviewers, until consensus was reached. Meta-analysis or statistical analysis of the studies reviewed was not conducted due to the heterogeneity of study participants, differences in reported outcome measures, and a limited number of study participants. Therefore, narrative analyses were conducted, focusing on participant characteristics, interventions employed, and pre- and post-treatment data pertaining to the outcomes of interest.

Inclusion Criteria

Studies were included if they met the following criteria: (1) published in English and in a peer-reviewed journal; (2) investigated the effect of Schroth exercise on Cobb angles measuring ≥ 10°, (3) investigated other outcome measures pertaining to QoL and functional capacity, (4) study designs were limited to randomised controlled trials (RCTs) only, (5) study populations were limited to adolescents with AIS who have not received prior bracing or operative care, and (6) comparison groups must have included age-matched participants with AIS who received nonoperative care, which entailed observation only or wearing a brace or engaging in an exercise regime that may include any form of stretching exercises, strengthening exercises, or mobilisation exercises.

Exclusion Criteria

RCTs involving individuals with AIS who received prior bracing or operative care in addition to cohort studies, case-control studies, case reports, opinions, clinical commentary, dissertations, conference posters, abstracts, letters to the editor, and articles without scientific data or a report of their methodology were excluded.

Outcomes

The primary outcome of interest was the effect of Schroth exercises on Cobb angle compared to standard nonoperative care. Secondary outcome measures include QoL and/or tests of functional capacity.

Data Extraction and Synthesis

The following data were extracted from each article: study design, participant characteristics, condition of interest, interventions employed, and pre- and post-treatment data pertaining to the outcomes of interest. Qualifying manuscripts were assessed using the PEDro scale. The PEDro scale has been validated as a measure of the methodological quality of clinical trials[32] assessing internal validity and sufficient statistical information for interpretability. The tool uses an 11-point scale based on items from the Delphi list developed by Verhagen et al.[33] The first point pertains to external validity and is not counted toward the overall score, leaving a possible total score of 10. If the trial did not report on a particular PEDro criterion, it was scored as if the criterion was not met. A PEDro score of 6 or greater is deemed high quality evidence, while 4-5 is deemed to be fair quality, and 3 or less is poor quality.[34]

Inter-observer agreement was calculated using the Kappa statistic.[35] Interpretation of Kappa values as described by Landis and Koch[36] are as follows: < 0 poor inter-rater agreement, 0.01 to 0.20 as slight agreement, 0.21 to 0.40 as fair agreement, 0.41 to 0.60 as moderate agreement, 0.61 to 0.80 as substantial agreement, and 0.81 to 1.0 as almost perfect agreement.

RESULTS

Figure 1 depicts the flow diagram of articles selected through the review. A total of 875 entries were identified after conducting the database searches, of which 34 were removed due to duplication. A total of 841 abstracts were then screened, and 29 full-text articles were selected for further assessment. Following this process, 5 RCTs[37-41] met the inclusion criteria for this review. The reviewers' Kappa value for the 5 articles was 1.0 (perfect agreement).

Study Quality and Characteristics

Table 1 depicts the PEDro score for each of the included studies. The average PEDro score of the studies assessed was 7. This is higher than the reported mean PEDro scores of musculoskeletal studies (5.08 ±1.7).[42] A total of 157 subjects participated across all 5 studies (141 female; 16 male). Two studies[37,39] include the same study participants but analyse different outcomes. Four studies[38-41] investigated the effect of Schroth exercises on Cobb angle, 3 studies[37-39] analysed QoL scores, and 2 studies[37,40] assessed changes in functional capacity. Study participants were similar in age, BMI, and baseline Cobb angle measurements. All primary intervention groups employed supervised Schroth exercises. In one study[38] a secondary intervention group employed the use of home-based Schroth exercises. In the remaining studies, control groups underwent observation only or bracing[39,40], supervised core exercises[40], or proprioceptive neuromuscular facilitation (PNF) exercises[41].

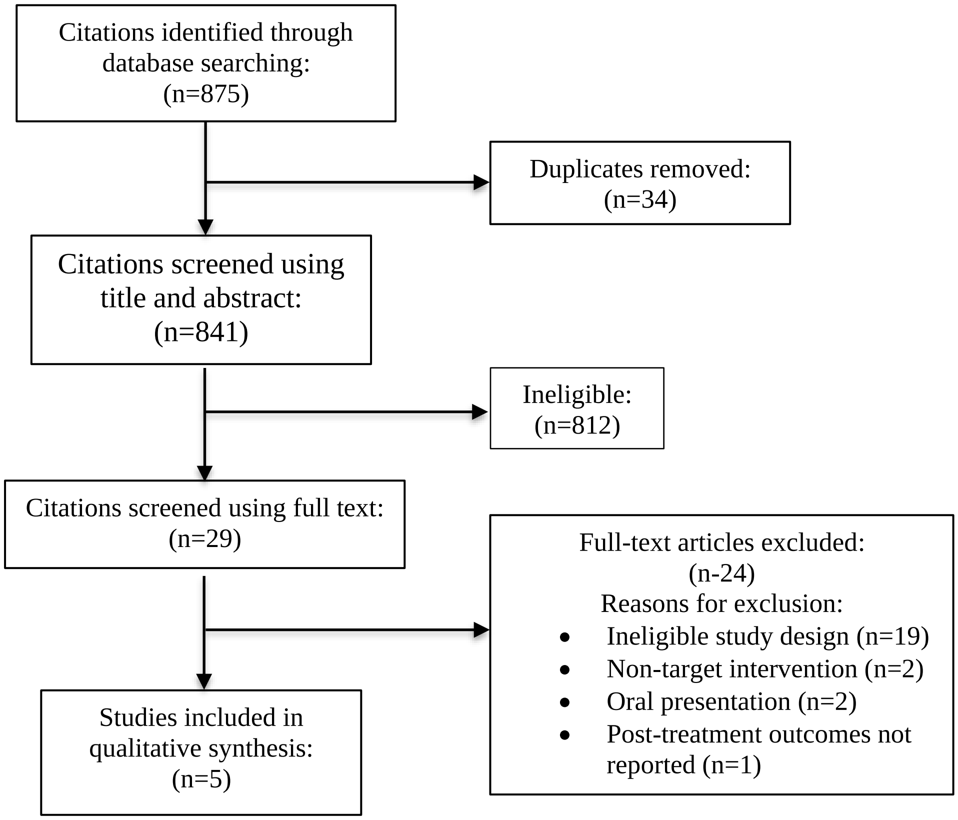

Figure 1. Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flowchart. Adolescent Idiopathic Scoliosis; Rehabilitation; Schroth exercise; Cobb angle; QoL

Description of Interventions

A full description of the interventions employed across the 5 studies is outlined in **Table 2**, where variations in the dosages of Schroth exercises are evident. The control groups were exposed to non-surgical management, which involved Pilates exercises, core exercises, PNF exercises, and observation or bracing.

Table 1. PEDro Scores for Qualified Studies

Study	Item 1	Item 2	Item 3	Item 4	Item 5	Item 6	Item 7	Item 8	Item 9	Item 10	Item 11	Total score
Schreiber 2015	Y	Y	Y	Y	N	N	Y	Y	Y	Y	Y	8
Kuru 2016	Y	Y	Y	Y	N	N	N	Y	Y	Y	N	6
Schreiber 2016	Y	Y	Y	Y	N	N	Y	Y	Y	Y	Y	8
Kocaman 2021	Y	Y	N	Y	N	N	Y	Y	Y	Y	Y	7
Mohamed 2021	Y	Y	Y	Y	N	N	N	Y	Y	Y	N	6

PEDro Criteria: Item 1 (Eligibility criteria), Item 2 (Subjects randomly allocated), Item 3 (Allocation concealed), Item 4 (Intervention groups similar), Item 5 (subjects were blinded), Item 6 (Therapists administering therapy blinded), Item 7 (All assessors blinded), Item 8 (At least 1 key outcome obtained from more than 85% of subjects initially allocated), Item 9 (All subjects received treatment or control intervention or an Intention-to-treat analysis performed), Item 10 (Between group comparison reported for at least one variable), Item 11 (study provides both point measures and measures of variability for at least one key outcome)

Study	Study Design	N	Study Participant Characteristics	Intervention Description	Control Description	Study Duration	F/U	Outcome Measures
Schreiber 2015	RCT	50 (47F, 3M)	23F, 2M with mean age of 13 years, mean BMI of 18 and mean baseline Cobb angle of 29° Control group 24F, 1M with mean age of 13 years, mean BMI of 20 and mean baseline Cobb angle of 28°	Supervised Schroth exercises included 5 1-hour long private sessions during the first two weeks, followed by weekly 1-hour long group classes combined with a 30±45 min daily home exercise program. Program duration was 6 months consisting of 5 private sessions and 22 group sessions.	Observation or bracing according to SRS recommendations.	26 wks	26 wks	SRS-22r SAQ Biering-Sorensen test
Kuru 2016	RCT	45 (39F, 6M)	Intervention group (Supervised Schroth exercise) 15 F, 1M with mean age of 13 years, mean BMI of 19 and mean baseline Cobb angle of 33° Home exercise group 12 F, 3M with mean age of 13 years, mean BMI of 20 and mean baseline Cobb angle of 30° Control group 13 F, 2 M with mean age of 13 years, mean BMI of 18 and mean baseline Cobb angle of 30°	Supervised Schroth exercises 3x/wk for 6 wks, 90-min in duration. The program was taught to caregivers and patients were asked to continue performing exercises at home for 6 months on completion of the 6 wk program. Home exercise group was taught Schroth exercises by a physiotherapist and were instructed to perform exercises at home for 6 months.	Observation only	6 wks	6 wks, 12 wks and, 24 wks Cobb angle F/U at 24 wks only	Cobb angle Angle of trunk rotation Height of hump Waist asymmetry SRS-23
Schreiber 2016	RCT	50 (47F, 3M)	Intervention group 23F, 2M with mean age of 13 years, mean BMI of 18 and mean baseline Cobb angle of 29° Control group 24F, 1M with mean age of 13 years, mean BMI of 20 and mean baseline Cobb angle of 28°	Supervised Schroth exercises included 5 1-hour long private sessions during the first two weeks, followed by weekly 1-hour long group classes combined with a 30±45 min daily home exercise program. Program duration was 6 months consisting of 5 private sessions and 22 group sessions.	Observation or bracing according to SRS recommendations.	26 wks	26 wks	Cobb angle

Kocaman 2021	RCT	28 (21F, 7M)	Intervention group 10F, 4M with mean age of 14 years, mean BMI of 20, mean baseline Cobb T of 18°, and mean baseline Cobb L of 16° Control group 11F, 3M with mean age of 14 years, mean BMI of 20, mean baseline Cobb T of 17°, and mean baseline Cobb L of 15°	Supervised Schroth exercises 3x/wk for 10 weeks, 90-min in duration. Total of 30 supervised sessions.	Supervised core exercises 3x/wk for 10 weeks, 90-min in duration. Total of 30 supervised sessions.	10 wks	10 wks	Cobb angle Angle of trunk rotation SRS-22
Mohamed 2021	RCT	34F	Intervention group 17F with mean age of 15 years, mean BMI of 19 and mean baseline Cobb angle of 20° Control group 17F with mean age of 15 years, mean BMI of 20 and mean baseline Cobb angle of 20°	Supervised Schroth exercises 3x/wk for 6 months, 60-min in duration. Total of 78 supervised sessions.	Supervised PNF exercises 3x/wk for 6 months, 60-min in duration. Total of 78 supervised sessions.	26 wks	26 wks	Cobb angle Angle of trunk rotation Plantar pressure distribution 6MWT

Table 2. Study Characteristics

F/U = follow-up; SRS = Scoliosis Research Society; SRS-22 = Scoliosis Research Society 22 questionnaire; SRS-22r = Scoliosis Research Society 22r questionnaire; SRS-23 = Scoliosis Research Society 23 questionnaire; SAQ = Spinal Appearance Questionnaire; BMI = Body mass index; PNF = proprioceptive neuromuscular facilitation exercises; 6MWT = 6-minute walk test

Description of Outcome Measures

The outcome measures and follow-up periods utilised in 4 of the 5 studies to assess Cobb angle are depicted in Table 3. Standard anterior-posterior full-spine radiographs were used to measure the Cobb angle in the included studies.[38-41] Three studies[37,38,41] assessed QoL measures utilising different versions of the SRS health-related QoL questionnaire (https://www.srs.org/professionals/online-education-and-resources/patient-outcome-questionnaires). Schreiber et al.[37] used the SRS22r, Kuru et al.[38] used the SRS23, and Kocaman et al.[40] used the SRS22. The questionnaire assesses scoliosis-specific health-related QoL and consists of five domains: function, pain, mental health, self-image, and satisfaction with management. The questionnaire has 22 (SRS-22 and SRS22r) or 23 items (SRS-23) and a score between 0 and 5 is given to each item, 0 being the worst outcome and 5 being the best outcome. Two studies[37,41] assessed functional capacity. Schreiber et al.[37] used the Biering-Sorensen test, a validated test to assess the isometric endurance of the trunk extensor muscles by measuring the duration in seconds in which a subject is able to hold the trunk in extension while fixed to a table. Mohamed and Yousef[41] used the six-minute walk test (6MWT) to ascertain cardiorespiratory capacity. Subjects were asked to walk at a steady pace, without running, over 20 metres on an unobstructed and rectangular pathway to cover as much distance as possible during 6 minutes. Standing rests may be taken but the timer must keep going. The score of the test is the distance walked in metres in 6 minutes.

Effect of Schroth Exercises on Cobb Angle

Table 3 depicts the reported means, medians, ranges, standard deviations, and *p*-values for Cobb angles measured at baseline, 10-12 weeks, and 24-26 weeks from four studies.[38-41] Among these studies, two studies[38,39] showed no meaningful change in the pre- and post-intervention Cobb angles that may be accounted for outside of measurement error alone. Kocaman et al.[40] show some change beyond measurement error, but there is no follow-up beyond 12 weeks. Mohamed and Yousef[41] showed some change in Cobb angle at 26 weeks that may be attributable to the intervention, but no reasonable follow-up is provided beyond this.

In the study by Kuru et al.[38], 45 participants (39 female; 6 male) of similar age, BMI, and baseline Cobb angle were randomised into 3 groups: 1) supervised Schroth exercise group, 2) home-based Schroth exercise group, and 3) control group, which entailed observation only. The supervised Schroth exercise group met 3 times per week for 6 weeks with each session lasting 90 minutes in duration. The program was then taught to their caregivers and the intervention group participants were asked to continue to perform the exercises at home for 6 months. The home exercise group was taught Schroth exercises by a physiotherapist and were asked to perform the exercises at home for 6 months. In comparison of the 3 groups, changes in Cobb angle were noted in favour of the supervised Schroth exercise group, though the changes were so small that they may be attributed to measurement error alone.

In the study by Schreiber et al.[39], 50 participants (47 female; 3 male) with AIS of similar age, BMI, and baseline Cobb angle were randomised into two groups; the intervention group, which entailed undergoing 5 1-hour-long private sessions with a physiotherapist to learn the Schroth exercises during the first two weeks of the study followed by engagement in weekly 1-hour-long group classes, concluding the study at 6 months. The control group underwent observation or bracing in accordance with Scoliosis Research Society (SRS) recommendations. Schreiber and colleagues measured the large curve between groups at 6 months and found a mean Cobb angle decrease by 1.2° in favour of the Schroth exercise group and a mean Cobb angle increase by 2.3° in the control group. Again, this change may be attributed to measurement error alone and not necessarily a reflection of any meaningful post-intervention change.

In the study by Kocaman et al.[40], 28 participants (21 female; 7 male) of similar age, BMI, and baseline Cobb angle were randomised into 2 groups: 1) supervised Schroth exercise group and 2) supervised core stabilisation exercise group. Both groups met 3 times per week for 10 weeks with each exercise session lasting 90 minutes in duration. In comparison of the groups, considerable changes in Cobb angle were noted in favour of the supervised Schroth exercise group compared to the core stabilisation exercise group ($p<0.001$). However, there is no follow-up beyond this point.

In the study by Mohamed and Yousef[41], 34 females with similar baseline characteristics were randomised into two groups: 1) supervised Schroth exercise group meeting 3 times per week to engage in 60 minutes of exercise over a period of 6 months and 2) supervised 60-minute sessions of proprioceptive neuromuscular facilitation (PNF) exercises 3 times per week over 6 months. Some change in Cobb angle was appreciated in both groups ($p<0.001$) with between-group comparison in favour of the Schroth group ($p=0.001$). However, again no reasonable follow-up is noted beyond this point to ascertain whether these effects were maintained.

Table 3. Reported means, medians, ranges, standard deviations and *p*-values for Cobb angles measured at baseline, 10-12 weeks, and 24-26 weeks

Study	Description	Baseline		10-12 weeks		24-26 weeks	
		mean ± SD or median (min-max)	*p*-value	mean ± SD	*p*-value	mean ± SD or median (min-max)	*p*-value
Kuru 2016	Schroth exercises	33.4±8.9 32.0 (20.0 – 50.0)	0.397	-	-	32 (20 – 45)	-
	Home exercise	30.3±7.6 30.0 (20.0 – 40.0)				35(20 – 45)	
	Control group	30.3±6.6 28.0 (20.0-45.0)				32 (22-46)	
Schreiber 2016	Schroth exercises	29.1±8.9 (11.3-44.3)	-	-	-	27.7±8.9 (14.4-43.9)	-
	Control group	27.9±8.8 (11.7-42.0)				29.1±8.8 (12.1-44.7)	
Kocaman 2021	Schroth exercises	T 17.64±4.1 L 15.80±3.42	-	T 9.71±3.47 L 9.40±2.61	<0.001	-	-
	Core exercises	T 17.29±3.45 L 15.17±4.02		T 13.57±5.03 L 12.33±4.37			
Mohamed 2021	Schroth exercises PNF exercises	20.42±2.57 20.21±2.80	0.40	-	-	14.11±1.96 17.46±2.37	0.001

T = Cobb angle of thoracic spine; L = Cobb angle of lumbar spine

Effect of Schroth Exercises on Quality of Life

Table 4 depicts the reported means, medians, ranges, standard deviations or standard errors, and *p*-values for QoL measured at baseline, 6 weeks, 10-12 weeks, and 24-26 weeks from three studies.[37,38,40] Kocaman et al.[40] was the only study that found significant improvements in QoL in favour of the Schroth exercise group at 10 weeks ($p<0.001$).

Table 4. Reported means, medians, ranges, standard deviations or standard errors, and *p*-values for QoL measured at baseline, 6 weeks, 10-12 weeks, and 24-26 weeks

Study	Description	QoL Measure	Baseline		6 weeks		10-12 weeks		24-26 weeks	
			mean ± SD or (SE) or median (min-max)	*p*-value	mean ± SD	*p*-value	mean ± SD or (SE) or median (min-max)	*p*-value	mean ± SD or (SE) or median (min-max)	*p*-value
Schreiber 2015	Schroth exercises Control group	SRS22r	4.25(0.07) 4.15(0.07)	-	-	-	4.29(0.07) 4.18(0.07)	0.83	4.40(0.07) 4.15(0.07)	0.08
Kuru 2016	Schroth exercises	SRS23	3.9±0.6 4.2(2.7 - 4.7)	0.452	4.2(3.3 - 4.7)	-	4.3(3.3 - 4.8)	-	4.4(3.5 – 5.0)	-
	Home exercise group		3.9±0.4 4.0(3.2 - 4.5)		4.0(3.3 - 4.7)		4.1(3.5 - 4.8)		3.9(3.9 - 4.7)	
	Control group		4.1±0.4 4.1(3.3 - 4.6)		4.2(3.3 – 4.6)		4.1(3.5 – 4.7)		4.1(3.0 – 4.7)	
Kocaman 2021	Schroth exercises Core exercises	SRS22	3.49±0.13 3.48±0.24	-	-	-	4.56±0.13 4.30±0.17	<0.001	-	-

SRS-22 = Scoliosis Research Society 22 questionnaire; SRS-22r = Scoliosis Research Society 22r questionnaire; SRS-23 = Scoliosis Research Society 23 questionnaire

Effect of Schroth Exercises on Functional Capacity

Table 5 depicts the reported means, standard deviations, and *p*-values for functional capacity measured at baseline, 3 months, and 6 months from two studies.[37,41] Schreiber et al.[37] found that the Schroth exercise group had a significantly longer isometric hold time over the control group at 3 months (*p*=0.04). Though this effect was not maintained at 6 months follow-up (*p*=0.89). Mohamed and Yousef[41] found significant improvement in 6MWT in favour of the Schroth exercise group at 6 months follow-up (*p*=0.001).

Table 5. Reported means, standard deviations, and *p*-values for functional capacity measured at baseline, 3 months, and 6 months

Study	Description	Test	Baseline	3 months		6 months	
			mean ± SD	mean ± SD	*p*-value	mean ± SD	*p*-value
Schreiber 2015	Schroth exercises Control group	Biering-Sorensen test (sec)	117.38±12.19 120.96±12.30	149.63±12.40 125.77±12.97	0.04	154.10±12.58 132.09±12.83	0.89
Mohamed 2021	Schroth exercises PNF exercises	6MWT (m)	288±9.73 283.70±9.45	-	-	328±9.63 284.94±11.29	0.001

6MWT = 6-minute walk test

DISCUSSION

Our review included five articles with an average PEDro score of 7/10. One study investigated the effect of Schroth exercises on Cobb angle only.[39] Two studies investigated the effect on Cobb angle and QoL[38,40]. One study investigated the effect on QoL and functional capacity[36], and one study investigated the effect on Cobb angle and functional capacity[41].

Clinicians and researchers consider a 5° change in Cobb angle to be clinically important.[28] The evidence depicted from the included studies suggests that clinical improvement in the Cobb angle may be appreciated in as little as 10 weeks[40] to as long as 26 weeks[41] with engagement in supervised Schroth exercises when compared to standard nonoperative care.

In regard to QoL, while MCID values exist for the SRS-22, this is related to the appearance, activity, and pain domains after surgical correction of AIS[43], and thus would not be applicable with the patient population identified in this review who do not undergo operative care. Moreover, the use of MCID has been criticized as an inaccurate, and sometimes unattainable, measure among individuals with AIS in this context, prompting Kelly et al.[44] to propose the utilization of the minimum detectable measurement difference (MDMD) as an alternative to the MCID in outcomes research. However, the proposed values offered by Kelly et al. also apply to AIS patients who undergo surgical correction, rendering a suitable means of detecting MCID or MDMD for AIS patients engaged in scoliosis-specific exercises untenable at this juncture. The same paucity of information related to MCID in functional capacity among AIS patients who undergo nonoperative care also applies.

The variability in study protocols including the study population, type of interventions employed in the control groups, dosage, and outcome measures make it difficult to determine the optimal treatment protocol of Schroth exercises. In a meta-analysis conducted by Park et al.[45], the authors determined that treatment duration of 6 months or longer has a large effect size on Cobb angle. Kocaman et al.[40] reported regression in Cobb angle within 10 weeks, which may owe to the dosage of supervised Schroth exercise engagement; 30 90-minutes sessions over 10 weeks when compared to the dosage applied by Kuru et al.[38] (18 90-minute sessions over 6 weeks). Though, this discrepancy may also be explained by the smaller baseline Cobb angles of study participants in the studies by Kocaman et al.[40] and Mohamed and Yousef[41] when compared to the participants in the other studies[38,39] who presented with larger baseline Cobb angles. This line of reasoning comports with the findings reported by Park et al.[45], where in their observation the success of Schroth exercises was more readily appreciated in individuals with a baseline Cobb angle of less than 30°.

Kuru et al.[38], Schreiber et al.[39], and Mohamed and Yousef[41] demonstrated that exercise consistency maintains Cobb angle improvements at 24 to 26 weeks despite considerable differences in baseline Cobb angles. Therefore, it would appear that baseline Cobb angle measurement and treatment duration and consistency may serve as primary factors in determining the success of Schroth exercises on Cobb angle. This notion comports with what we presently understand to be the natural history of AIS.[17-19,46]

Given the nature of RCTs, it is not possible to blind the participants or therapists when administering the interventions. This may increase the risk of detection bias.[47] Moreover, Kocaman et al.[40] did not employ allocation concealment, thus decreasing the internal validity of their studies and increasing the risk of selection bias.[48]

LIMITATIONS

There are several limitations in this systematic review. The small number of articles included indicates that there is little evidence on the use of Schroth exercises for the management of AIS. Secondly, the studies reviewed had relatively small sample sizes ranging from 24 to 52 subjects. The studies reviewed had short follow-up periods when compared to the normative duration of scoliosis treatment of 2-3 years. Moreover, individuals with AIS represent a complex heterogeneous group with differing anatomic features and health states, making it difficult to define a target population in this context. Lastly, the literature search only included articles published in English, excluding available evidence from non-English studies that may pertain to some aspect of conservative management of AIS.

CONCLUSION

In conclusion, there is very low-quality evidence in favour of Schroth exercises engagement on Cobb angle, between 10 to 26 weeks, when compared to standard treatment for AIS. Improvement in quality of life and functional capacity may also be realized in the short term, though maintenance of such effects in the long-term remain to be elucidated. Future high-quality studies with larger sample sizes and longer intervention and follow-up periods, ideally lasting a minimum of 2 years in duration beyond skeletal maturity, would help to further and more confidently ascertain the long-term effect of Schroth exercises on Cobb angle, QoL, and functional capacity in individuals with AIS.

ACKNOWLEDGEMENT

The authors would like to thank Jim Eubanks, MD, DC, MS of the Department of Physical Medicine and Rehabilitation at the University of Pittsburgh Medical Center for sharing his insight regarding the production of this review.

AUTHOR CONTRIBUTIONS

Conceptualization, J.G., H.A., R.D., P.P.P.; methodology, J.G., H.A., R.D., P.P.P.; formal analysis, J.G., H.A., R.D., P.P.P.; investigation, J.G., H.A.; resources, J.G., H.A.; writing—original draft preparation, J.G., H.A., R.D., P.P.P.; writing—review and editing, J.G., H.A., R.D., P.P.P. All authors have read and agreed to the published version of the manuscript.

FUNDING

This research received no external funding.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

REFERENCES

1. Weinstein SL, Dolan LA, Cheng JC, Danielsson A, Morcuende JA. Adolescent idiopathic scoliosis. *Lancet*. 2008;371(9623):1527–37.

2. Kamtsiuris P, Atzpodien K, Ellert U, Schlack R, Schlaud M. Prävalenz von somatischen Erkrankungen bei Kindern und Jugendlichen in Deutschland. Ergebnisse des Kinder- und Jugendgesundheitssurveys (KiGGS) [Prevalence of somatic diseases in German children and adolescents. Results of the German Health Interview and Examination Survey for Children and Adolescents (KiGGS)]. *Bundesgesundheitsblatt Gesundheitsforschung Gesundheitsschutz*. 2007;50(5-6):686-700. doi:10.1007/s00103-007-0230-x

3. Suh SW, Modi HN, Yang JH, Hong JY. Idiopathic scoliosis in Korean schoolchildren: a prospective screening study of over 1 million children. *Eur Spine J*. 2011;20(7):1087-1094. doi:10.1007/s00586-011-1695-8

4. Nery LS, Halpern R, Nery PC, Nehme KP, Stein AT. Prevalence of scoliosis among school students in a town in southern Brazil. *Sao Paulo Med J*. 2010;128(2):69-73. doi:10.1590/s1516-31802010000200005

5. Daruwalla JS, Balasubramaniam P, Chay SO, Rajan U, Lee HP. Idiopathic scoliosis. Prevalence and ethnic distribution in Singapore schoolchildren. *J Bone Joint Surg Br*. 1985;67(2):182-184. doi:10.1302/0301-620X.67B2.3980521

6. Wong HK, Hui JH, Rajan U, Chia HP. Idiopathic scoliosis in Singapore schoolchildren: a prevalence study 15 years into the screening program. *Spine (Phila Pa 1976)*. 2005;30(10):1188-1196. doi:10.1097/01.brs.0000162280.95076.bb

7. Cilli K, Tezeren G, Taş T, et al. Sivas il merkezinde skolyoz için okul taramasi [School screening for scoliosis in Sivas, Turkey]. *Acta Orthop Traumatol Turc*. 2009;43(5):426-430. doi:10.3944/AOTT.2009.426

8. Soucacos PN, Soucacos PK, Zacharis KC, Beris AE, Xenakis TA. School-screening for scoliosis. A prospective epidemiological study in northwestern and central Greece. *J Bone Joint Surg Am*. 1997;79(10):1498-1503. doi:10.2106/00004623-199710000-00006

9. Weinstein SL, Dolan LA, Wright JG, Dobbs MB. Effects of bracing in adolescents with idiopathic scoliosis. *N Engl J Med*. 2013;369(16):1512-1521. doi:10.1056/NEJMoa1307337

10. Lonstein JE, Carlson JM. The prediction of curve progression in untreated idiopathic scoliosis during growth. *J Bone Joint Surg Am*. 1984;66(7):1061-1071.

11. Tan KJ, Moe MM, Vaithinathan R, Wong HK. Curve progression in idiopathic scoliosis: follow-up study to skeletal maturity. *Spine (Phila Pa 1976)*. 2009;34(7):697-700.

12. Balagué F, Pellisé F. Adolescent idiopathic scoliosis and back pain. *Scoliosis Spinal Disord*. 2016;11(1):27. Published 2016 Sep 9. doi:10.1186/s13013-016-0086-7

13. Asher MA, Burton DC. Adolescent idiopathic scoliosis: natural history and long term treatment effects. *Scoliosis*. 2006;1(1):2. Published 2006 Mar 31. doi:10.1186/1748-7161-1-2

14. Sharma S, Londono D, Eckalbar WL, et al. A PAX1 enhancer locus is associated with susceptibility to idiopathic scoliosis in females. *Nat Commun*. 2015;6:6452. Published 2015 Mar 18. doi:10.1038/ncomms7452

15. Gao X, Gotway G, Rathjen K, Johnston C, Sparagana S, Wise CA. Genomic Analyses of Patients with Unexplained Early-Onset Scoliosis. *Spine Deform*. 2014;2(5):324-332. doi:10.1016/j.jspd.2014.04.014

16. Gao W, Peng Y, Liang G, et al. Association between common variants near LBX1 and adolescent idiopathic scoliosis replicated in the Chinese Han population. *PLoS One*. 2013;8(1):e53234. doi:10.1371/journal.pone.0053234

17. Weinstein, Stuart L. MD The Natural History of Adolescent Idiopathic Scoliosis. *Journal of Pediatric Orthopaedics*: July 2019 - Volume 39 - Issue - p S44-S46 doi: 10.1097/BPO.0000000000001350

18. Lee CF, Fong DY, Cheung KM, Cheng JC, Ng BK, Lam TP, Yip PS, Luk KD (2012) A new risk classification rule for curve progression in adolescent idiopathic scoliosis. *Spine J Off J North Am Spine Soc*. 12(11):989–995. https://doi.org/10.1016/j.spinee.2012.05.009

19. Charles YP, Canavese F, Dimeglio A (2017) Curve progression risk in a mixed series of braced and nonbraced patients with idiopathic scoliosis related to skeletal maturity assessment on the olecranon. *J Pediatr Orthop B*. 26(3):240–244. https://doi.org/10.1097/BPB.0000000000000410

20. Negrini S, Donzelli S, Aulisa AG, et al. 2016 SOSORT guidelines: orthopaedic and rehabilitation treatment of idiopathic scoliosis during growth. *Scoliosis Spinal Disord*. 2018;13:3. Published 2018 Jan 10. doi:10.1186/s13013-017-0145-8

21. Agabegi SS, Kazemi N, Sturm PF, Mehlman CT. Natural History of Adolescent Idiopathic Scoliosis in Skeletally Mature Patients: A Critical Review. *J Am Acad Orthop Surg*. 2015;23(12):714-723. doi:10.5435/JAAOS-D-14-00037

22. Gomez JA, Hresko MT, Glotzbecker MP. Nonsurgical Management of Adolescent Idiopathic Scoliosis. *J Am Acad Orthop Surg*. 2016;24(8):555-564. doi:10.5435/JAAOS-D-14-00416

23. Canavese F, Kaelin A. Adolescent idiopathic scoliosis: Indications and efficacy of nonoperative treatment. *Indian J Orthop*. 2011;45(1):7-14. doi:10.4103/0019-5413.73655

24. Romano M, Minozzi S, Zaina F, et al. Exercises for adolescent idiopathic scoliosis: a Cochrane systematic review. *Spine (Phila Pa 1976)*. 2013;38(14):E883-E893. doi:10.1097/BRS.0b013e31829459f8

25. Berdishevsky H, Lebel VA, Bettany-Saltikov J, et al. Physiotherapy scoliosis-specific exercises - a comprehensive review of seven major schools. *Scoliosis Spinal Disord*. 2016;11:20. Published 2016 Aug 4. doi:10.1186/s13013-016-0076-9

26. Weiss HR. The method of Katharina Schroth - history, principles and current development. *Scoliosis*. 2011;6:17. Published 2011 Aug 30. doi:10.1186/1748-7161-6-17

27. Burger M, Coetzee W, du Plessis LZ, et al. The effectiveness of Schroth exercises in adolescents with idiopathic scoliosis: A systematic review and meta-analysis. *S Afr J Physiother*. 2019;75(1):904. Published 2019 Jun 3. doi:10.4102/sajp.v75i1.904

28. Richards BS, Bernstein RM, D'Amato CR, Thompson GH. Standardization of criteria for adolescent idiopathic scoliosis brace studies: SRS Committee on Bracing and Nonoperative Management. *Spine (Phila Pa 1976)*. 2005;30(18):2068-2077. doi:10.1097/01.brs.0000178819.90239.d0

29. Kim G, HwangBo PN. Effects of Schroth and Pilates exercises on the Cobb angle and weight distribution of patients with scoliosis. *J Phys Ther Sci*. 2016;28(3):1012-1015. doi:10.1589/jpts.28.1012

30. Moher D, Liberati A, Tetzlaff J, Altman DG, The PRISMA Group. Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement. *BMJ*. 2009; 339:b2535. PMID: 19622551.

31. Liberati A, Altman DG, Tetzlaff J, Mulrow C, Gtzsche PC, et al. The PRISMA Statement for Reporting Systematic Reviews and Meta-Analyses of Studies That Evaluate Health Care Interventions: Explanation and Elaboration. *BMJ*. 2009; 339:b2700. PMID: 19622552.

32. de Morton NA. The PEDro scale is a valid measure of the methodological quality of clinical trials: a demographic study. *Aust J Physiother*. 2009; 55: 129-133.

33. Verhagen AP, de Vet HC, de Bie RA, Kessels AG, Boers M, Bouter LM, et al. The Delphi list: a criteria list for quality assessment of randomized clinical trials for conducting systematic reviews developed by Delphi consensus. *J Clin Epidemiol*. 1998; 51: 1235-1241.

34. Foley NC, Bhogal SK, Teasell RW, Bureau Y, Speechley MR. Estimates of quality and reliability with the physiotherapy evidence-based database scale to assess the methodology of randomized controlled trials of pharmacological and nonpharmacological interventions. *Phys Ther*. 2006;86(6):817-824.

35. Viera AJ, Garrett JM. Understanding interobserver agreement: the kappa statistic. *Fam Med*. 2005;37(5):360-363.

36. Landis JR, Koch GG. The measurement of observer agreement for categorical data. *Biometrics*. 1977;33(1):159-174.

37. Schreiber S, Parent EC, Moez EK, et al. The effect of Schroth exercises added to the standard of care on the quality of life and muscle endurance in adolescents with idiopathic scoliosis-an assessor and statistician blinded randomized controlled trial: "SOSORT 2015 Award Winner". *Scoliosis*. 2015;10:24. Published 2015 Sep 18. doi:10.1186/s13013-015-0048-5

38. Kuru T, Yeldan İ, Dereli EE, Özdinçler AR, Dikici F, Çolak İ. The efficacy of three-dimensional Schroth exercises in adolescent idiopathic scoliosis: a randomised controlled clinical trial. *Clin Rehabil*. 2016;30(2):181-190. doi:10.1177/0269215515575745

39. Schreiber S, Parent EC, Khodayari Moez E, et al. Schroth Physiotherapeutic Scoliosis-Specific Exercises Added to the Standard of Care Lead to Better Cobb Angle Outcomes in Adolescents with Idiopathic Scoliosis - an Assessor and Statistician Blinded Randomized Controlled Trial. *PLoS One*. 2016;11(12):e0168746. Published 2016 Dec 29. doi:10.1371/journal.pone.0168746

40. Kocaman H, Bek N, Kaya MH, Büyükturan B, Yetiş M, Büyükturan Ö. The effectiveness of two different exercise approaches in adolescent idiopathic scoliosis: A single-blind, randomized-controlled trial. *PLoS One*. 2021;16(4):e0249492. Published 2021 Apr 15. doi:10.1371/journal.pone.0249492

41. Mohamed RA, Yousef AM. Impact of Schroth three-dimensional vs. proprioceptive neuromuscular facilitation techniques in adolescent idiopathic scoliosis: a randomized controlled study. *Eur Rev Med Pharmacol Sci*. 2021;25(24):7717-7725. doi:10.26355/eurrev_202112_27618

42. Moseley AM, Elkins MR, Janer-Duncan L, Hush JM. The Quality of Reports of Randomized Controlled Trials Varies between Subdisciplines of Physiotherapy. *Physiother Can*. 2014;66(1):36-43. doi:10.3138/ptc.2012-68

43. Carreon LY, Sanders JO, Diab M, et al. The minimum clinically important difference in Scoliosis Research Society-22 Appearance, Activity, And Pain domains after surgical correction of adolescent idiopathic scoliosis. *Spine (Phila Pa 1976)*. 2010;35(23):2079-2083. doi:10.1097/BRS.0b013e3181c61fd7

44. Kelly MP, Lenke LG, Sponseller PD, et al. The minimum detectable measurement difference for the Scoliosis Research Society-22r in adolescent idiopathic scoliosis: a comparison with the minimum clinically important difference. *Spine J*. 2019;19(8):1319-1323. doi:10.1016/j.spinee.2019.04.008

45. Park JH, Jeon HS, Park HW. Effects of the Schroth exercise on idiopathic scoliosis: a meta-analysis. *Eur J Phys Rehabil Med*. 2018;54(3):440-449. doi:10.23736/S1973-9087.17.04461-6

46. Dimeglio A, Canavese F. Progression or not progression? How to deal with adolescent idiopathic scoliosis during puberty. *J Child Orthop*. 2013;7(1):43-49. doi:10.1007/s11832-012-0463-6

47. Hróbjartsson A, Thomsen AS, Emanuelsson F, et al. Observer bias in randomised clinical trials with binary outcomes: systematic review of trials with both blinded and non-blinded outcome assessors. *BMJ*. 2012;344:e1119. Published 2012 Feb 27. doi:10.1136/bmj.e1119

48. Poolman RW, Struijs PA, Krips R, et al. Reporting of outcomes in orthopaedic randomized trials: does blinding of outcome assessors matter? *J Bone Joint Surg Am*. 2007;89(3):550-558. doi:10.2106/JBJS.F.00683

APPENDIX 1

MEDLINE search terms

(("adolescent"[MeSH Terms] OR "adolescent"[All Fields] OR adolescent[All Fields]) AND idiopathic[All Fields] AND ("scoliosis"[MeSH Terms] OR "scoliosis"[All Fields] OR scoliosis[All Fields])) AND (Schroth[All Fields] AND ("exercise"[MeSH Terms] OR "exercise"[All Fields] OR exercise[All Fields])) AND (Cobb[All Fields] AND angle[All Fields]) AND ("quality of life"[MeSH Terms] OR ("quality"[All Fields] AND "life"[All Fields]) OR "quality of life"[All Fields] OR quality of life[All Fields]) OR qol[All Fields]

Exercise Prescription for Low Back Pain Management in Parkinson's Disease

Joe H. Ghorayeb, DC, MHA[1]

Gregory Grigoropoulos, BS[2]

Elsa George, MD[3]

Evan Arbit, MD[3]

Hong Wu, MD, MS[3]

[1] University of Medicine & Health Sciences, New York, NY
[2] Loyola University Chicago Stritch School of Medicine, Maywood, IL
[3] Department of Physical Medicine and Rehabilitation, Rush University, Chicago, IL

Hong_Wu@rush.edu

Published: December 2022
Journal of the International Academy of Neuromusculoskeletal Medicine
Volume 19, Issue 2

ABSTRACT

People with Parkinson's disease (PD) frequently experience low back pain (LBP). LBP often causes significant disability and reduced quality of life. A body of literature exists supporting the utility of exercise for the management of LBP in the general population. However, no such guideline exists to aid clinicians in exercise prescription for the management of LBP for individuals with PD. This descriptive literature review summarizes the current knowledge with respect to the mechanism and utility of physical exercise (PE) interventions, issues related to exercise prescription, the influence of patient expectations on exercise adherence and outcomes, and the process of encouraging behavior modification in the context of managing LBP in individuals with PD. The effects of the interventions prescribed may vary depending on content-related factors (i.e., type of exercises, dosage, frequency, stage of disease progression, etc.) and contextual factors (i.e., treatment setting,

access to organized programs, etc.). Patient expectations also significantly influence adherence to, and outcomes of, PE. Treatment goals and timing are also important as they relate to the amount and type of PE to be prescribed within each stage of the disease and the expectation of outcomes of both clinicians and patients, respectively. Despite the need for more quantitative and qualitative investigations to further clarify the formulation and dosage of PE for patients with PD who experience LBP, this article outlines tailored PE prescription recommendations in this context based on the current body of knowledge available at this time.

Key words: Parkinson's disease; low back pain; exercise

Key Points:

- The prevalence of Parkinson's disease (PD) is on the rise and is expected to continue to increase over the next 20 years.

- Low back pain (LBP) is highly prevalent in individuals with PD with a longer duration and higher pain intensity when compared to the general population.

- Traditional treatment of PD including pharmacotherapy and neurosurgical procedures can be effective in treating PD-related symptoms, but cannot slow disease progression.

- Engagement in exercise and physical activity has demonstrated evidence of disease-modifying capabilities in PD in addition to LBP management.

- A sound exercise program to manage LBP in the context of PD ought to include a combination of aerobic and resistance training, flexibility exercises, gait and balance training, postural control exercises, and adjunct therapy, and should be individually tailored to the abilities, disease stage, precautions, and goals of each person.

INTRODUCTION

Parkinson's disease (PD) is a progressive neurodegenerative movement disorder defined by loss of dopaminergic neurons and the presence of alpha-synuclein within the midbrain.[1] PD is one of the fastest growing neurologic disorders[2], affecting 2-3% of the population over 65 years of age[3] with a yearly incidence estimated to be as high as 35 in 100,000[3], and is estimated to affect more than 12 million people worldwide by 2040[4]. PD peak prevalence is between 85-89 years of age, with the relative risk being 1.4 times higher in men than in women[2]. Beta-adrenergic antagonist, pesticide exposure, and dairy product consumption have been identified as potential risk factors in the development of PD. Coffee consumption and a history of engagement in physical activity and competitive sports have been shown to slow motor and cognitive decline with reduced mortality in those with PD.[5]

Pain is a common symptom of PD that negatively impacts quality of life. Patients with PD have been reported to experience five distinct types of pain: 1) musculoskeletal pain as a result of rigidity and/or skeletal deformity, 2) radicular-neuropathic pain due to either a root lesion, focal or peripheral neuropathy, 3) dystonia-related pain, 4) central pain, and 5) akathitic pain.[6,7] Of the many musculoskeletal pain conditions affecting individuals with PD, low back pain (LBP) is the most prevalent (87.6%) with a longer duration and higher pain intensity when compared to the general population.[8] Back pain ranks highest in terms of disability according to the Global Burden of Disease Study[9] and accounts for $134 billion in public and private healthcare spending in the United States[10].

Physical Deficits and PD Staging

The loss of dopaminergic neurons leads to the cardinal physical deficits associated with PD, beginning with early and prominent tremors of the hands followed by bradykinesia, whereby patients notice that simple everyday tasks take longer to perform. Rigidity proves to be the main obstacle to ambulation for the PD patient, resulting in shorter steps, an altered walking pace, reduced arm swing, and postural alterations. Spinal abnormalities and gait disturbance appear as a late manifestation of PD with characteristic festination when standing, initiating a step, and/or turning.[11]

PD may be staged descriptively for impairment and disability using the Hoehn & Yahr (H&Y) scale[12], which outlines an approximation of symptom progression by assessing impairment and disability, with the former equating to objective indicators of the disease and the latter being the apparent functional deficits as described in **Table 1**. The Unified Parkinson's Disease Rating Scale (UPDRS) is another rating tool used to gauge the severity and progression of PD.[13]

Table 1. Stages of PD Progression

Stage	Hoehn and Yahr Scale	Modified Hoehn and Yahr Scale
1	Unilateral involvement only, usually with minimal or no functional disability	Unilateral involvement only
1.5	-	Unilateral and axial involvement
2	Bilateral or midline involvement without impairment of balance	Bilateral involvement without impairment of balance
2.5	-	Mild bilateral disease with recovery on pull test
3	Bilateral disease: mild to moderate disability with impaired postural reflexes; physically independent	Mild to moderate bilateral disease; some postural instability; physically independent
4	Severely disabling disease; still able to walk or stand unassisted	Severe disability; still able to walk to stand unassisted
5	Confined to bed or wheelchair unless aided	Wheelchair-bound or bedridden unless aided

Mechanisms of Low Back Pain Development in PD

In addition to age-related degenerative changes that contribute to the development of LBP, two prevailing theories have been proposed as mechanisms that describe the increased prevalence of LBP among individuals with PD; altered posture and aberrant muscle tone, intensifying stress on the soft tissues and bony structures of the lumbar spine.[14] Watanabe et al. established that increased thoracic and/or thoracolumbar kyphosis as well as reduced lumbar range of motion occur in conjunction with progressive sagittal malalignment in the absence of sufficient compensatory postural alterations such as reduced thoracic kyphosis and pelvic retroversion, correlating strongly with the complaint of LBP and reduced walking ability.[15] Parkinsonian central pain is also implicated in the development and persistence of PD-related LBP due to diminished descending inhibitory control[16] and the influence of dopaminergic pathways on the basal ganglia and pain pathways[17].

PD-related rigidity has also been implicated with higher pain frequency and intensity. Allen et al. found that increased rigidity is strongly associated with pain and interfered with engaging in work and activities of daily living, even after adjustment for age and gender.[18] Some evidence suggests that changes in muscle structure and quality may contribute to the development of LBP in PD. Margraf and colleagues interpreted focal myopathy among patients with PD with camptocormia by way of identifying fatty infiltration in the paravertebral muscles as a potential reason for disease progression and muscle degeneration.[19] Though, the mechanism behind how these findings might contribute to the clinical presentation of LBP in the context of PD remains to be understood.

Treatment of PD and the Role of Exercise in PD

Pharmacologically, PD is typically managed with Levodopa in combination with Carbidopa to reduce adverse effects and improve CNS bioavailability. Anticholinergics or Amantadine may be employed if tremors are the primary symptom to be controlled. Selegiline, a selective MAO-B inhibitor, is typically used to treat early-stage PD and offers mild symptom relief.[20] While most anti-Parkinson medications may provide effective symptom control, the disease eventually progresses and becomes resistant to pharmacotherapy. In such instances, deep brain stimulation is an effective therapy to treat severe motor fluctuations or tremors in advanced PD.[21,22] Though, neither pharmacotherapy nor neurosurgical procedures confer disease-modifying capabilities in PD.[23,24] On the other hand, exercise, among other lifestyle factors, has been shown to slow PD progression.[25] In animal models of PD, regular exercise produces neuroprotective effects against the neurotoxins 6-hydroxydopamine (6-OHDA) and 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP)[26,27], which decidedly cause a loss of dopaminergic neurons[28]. Exercise further induces the accumulation of the ketone body D-b-hydroxybutyrate (DBHB) in the hippocampus, which in turn promotes the expression of brain-derived neurotrophic factor (BDNF); an important protein involved in plastic changes related to learning and memory.[29-32] Exercise dose has also been shown to play a factor, with greater physical activity conferring greater protection.[33] In addition to the aforementioned observations, attenuation of α-synuclein expression in conjunction with reduced α-synuclein-mediated inflammation via downregulation of toll-like receptor 2 and decreased NADPH oxidase expression has been observed in MPTP-induced mice following an 8-week treadmill exercise program.[34]

With respect to the effect of exercise on individuals with PD, Shulman et al.[35] conducted an important randomized clinical trial (RCT) of three types of exercise in order to determine whether superiority of outcomes exists in favor of one exercise modality compared to another. The authors randomized study participants with a diagnosis of PD (H&Y1-3) with at least two of the three cardinal signs of tremor, bradykinesia or rigidity in addition to the presence of mild to moderate gait impairment across three exercise groups: 1) high-intensity treadmill training; consisting of 30 minutes at 70%-80% of heart rate reserve (HRR), 2) low-intensity treadmill training (50 minutes at 40%-50% of HRR), and 3) stretching and resistance training (2 sets of 10 repetitions on each leg on 3 resistance machines [leg press, leg extension, and curl]). All three groups engaged in exercise 3 times per week for 12 weeks, for a total of 36 sessions. At the conclusion of the interventions, it was determined that both types of treadmill training improved cardiovascular fitness with no difference between groups. Unsurprisingly, stretching and resistance training resulted in the greatest increases in muscle strength and were the only group that improved in the UPDRS motor subscale ($p<0.05$). Moreover, the study authors reported no adverse effects with the engagement of either of the aforementioned exercise interventions, and no changes of antiparkinsonian medications were made during the trial.

The aim of this article is to summarize how exercise therapy exerts its effects on pain control, postural correction and control, flexibility, general fitness, gait performance, and balance in the context of PD, in addition to offering exercise prescription recommendations for this patient population.

METHODS

Search Strategy

A literature search using MEDLINE was conducted from inception to June 2022. The search terms included (e.g., MeSH in MEDLINE) are depicted in **Appendix 1**.

Study Selection

A two-phase approach to screening was used with two independent reviewers screening each citation and article. Phase one included screening of titles and abstracts for possible relevance. Phase two included full-text screening of possibly relevant studies. Any disagreement was resolved by discussion between the paired reviewers.

Inclusion Criteria

Studies were included if they met the following criteria: 1) published in English and in a peer-reviewed journal; 2) study designs included experimental (Randomised Clinical Trials) and observational (Cohort and Case-Control) studies; 3) study populations included adults (19+years old); 4) study populations confined to individuals with PD (H&Y 1-5); and 5) studies addressed one or more of the physical deficits and/or symptoms associated with PD such as aerobic fitness, muscle strength, gait performance, postural correction, flexibility, balance and pain control with some form of exercise as the primary intervention.

Exclusion Criteria

Studies which did not include some form of exercise as the primary intervention along with perspective articles, opinions, comments, letters to the editor, and articles without scientific data or a report of their methodology were excluded.

RESULTS

A total of 618 entries were identified after conducting the database searches, of which 44 were removed due to duplication. A total of 574 abstracts were then screened, and 48 full-text articles were selected for full-text review. Following this process, 22 studies were included in the review. Nine articles related to enhancing general fitness[51-56,61,69,74], 4 related to improving postural correction[77,79-81], 2 related to increasing flexibility[85,86], and 7 related to balance improvement[90-96] in the context of PD. Experimental studies describing the mechanisms in which exercise exerts its pain modulating effects in individuals with PD were unavailable. Therefore, a broad summary regarding the analgesic properties of exercise is offered based on review of 7 articles.[38-44]

Pain Control

Exercise has been shown to be a powerful intervention to lessen the perception of pain as well as improve mood and reduce stress.[36] The Center for Disease Control and Prevention's opioid prescribing guideline, published in 2016, recommends exercise as a first-line treatment in pain management.[37] Three distinct mechanisms explain how exercise modulates pain symptoms. The first is the reconceptualization of pain-related fear via the development of new inhibitory associations that compete with and suppress the original conditioned response to movement and physical activity in the individual with pain.[38-40] The second mechanism describes the effect of painful exercise on the activation of the conditioned pain modulation response, where an individual may report a lower pain rating of a primary pain complaint in the presence of a secondary painful stimulus.[41] Finally, the third mechanism highlights exercise's role in blunting the immune system's function in exacerbating pain states, in turn promoting an anti-inflammatory state with long-term engagement in exercise and physical activity.[42-44] Various modalities of exercise are capable of exerting pain modulating effects in addition to developing favorable phenotypic and functional adaptations as described below.

General Fitness and Gait Performance

Aerobic training improves general fitness[45,46] as well as gait performance and speed in patients with PD.[47-49] The risk of all-cause and cause-specific mortality is also greatly reduced in individuals who engage in both aerobic and muscle strengthening activities at the levels recommended by the 2018 physical activity guidelines for Americans.[50] Of the various modes of aerobic training to enhance cardiorespiratory fitness for individuals with PD, treadmill training, overground walking, and cycling have consistently produced the greatest improvements in gait performance, speed, stride length, and cadence.[51,52] Moreover, the addition of auditory cueing by way of rhythmic auditory stimulation (RAS) may be a useful adjunct to gait training, as evidenced by the finding of an RCT conducted by Calabro et al.[53], in which 50 patients with PD (H&Y 2&3) were randomly allocated to one of two groups: 1) treadmill gait training with RAS; where patients were instructed to synchronize their footsteps to the beat of the music played during the training sessions, and 2) treadmill gait training without the assistance of RAS. Each session lasted 30 minutes in duration and was completed 5 days per week over an 8-week period. While both groups demonstrated improvements in gait performance at the conclusion of the study, the RAS group demonstrated superior improvement in functional gait assessment (FGA), falls efficacy scale (FES), timed up-and-go test (TUG), and UPDRS ($p<0.001$). Both moderate-intensity aerobic exercise (50% of HRR) and high-intensity training (HIIT) (85% of HRR) have been shown to improve pain symptoms. However, HIIT also improves cardiorespiratory fitness. A 6-week moderate-intensity treadmill walking program (50% of HRR) for 52 sedentary patients with chronic LBP was compared to a 6-week program involving specific strengthening exercises targeting the trunk and upper and lower limbs.[54] Walking and resistance training were shown to be equally effective in reducing LBP symptoms with no significant difference between the two exercise modalities.

Twenty study subjects with chronic LBP (CLBP) and similar baseline characteristics were enrolled in an RCT conducted by Chatzitheodorou et al.[55] and were randomly allocated to an intervention group consisting of a 12-week high-intensity exercise treadmill program (85% of HRR) or a passive treatment group, which included the administration of 10 minutes of shortwave diathermy, 5 minutes of continuous pulse therapeutic ultrasound, 10 minutes of low-level laser therapy laser, and 12 minutes of electrotherapy at varying intensities per each subject’s tolerance. Study subjects in the high-intensity exercise program reported significant reductions in LBP, disability, and psychological strain compared to no improvement in the passive treatment group. These findings are further supported by a subsequent RCT carried out by Chatzitheodorou and colleagues[56], with a larger sample size (n=64), in which study subjects with CLBP were randomized into a dexamethasone suppression group (dex+) and a control group without dexamethasone suppression, and both groups engaged in high-intensity aerobic exercise. Subjects in the dex+ group reported a significant reduction in pain complaints and anxiety/depression, in addition to improved physical function and adrenocortical responsiveness ($p<0.001$) when compared to subjects in the control group. These findings suggest that for individuals with CLBP and associated psychological strain, engagement in high-intensity aerobic exercise may be a preferred strategy to induce a cortisol response, as a direct relationship between the strength of the cortisol response to a stressor (e.g., exercise) and developing a higher pain threshold has been reported in a number of studies.[57-60]

A more recent RCT by Harvey et al.[61] found that 36 HIIT sessions (≥85% of maximal heart rate) over a 12-week period among patients with PD (H&Y 1-3) with a mean age of 68.5 years resulted in an increase in peak oxygen consumption by $2.8 mLkg^{-1}min^{-1}$ in the intervention group compared to $1.5 mLkg^{-1}min^{-1}$ in the control group, suggesting that HIIT may be a feasible and acceptable exercise strategy in people with early to mid-stage Parkinson’s disease.

Because muscle strength declines in almost all patients with PD[62], the general physical requirements necessary to execute a controlled gait pattern, rise from a chair, and negotiate stairs are compromised, in turn increasing the risk of falls. If an individual with PD also presents with concomitant low bone mass, the risk of fractures with falls increases, resulting in a medical emergency. Strength training, characterized as contracting muscles against resistance, is accomplished using free weights, resistance machines, elastic bands, and resistance against water or one's own body weight is not only an effective hypoalgesic in patients with PD[63] but also derives desirable adaptations that counteract the development and progression of chronic disease in aging populations[64] in addition to reducing all-cause mortality.[65-68] In individuals with mild to moderate PD, high-force eccentric resistance training is both a safe and feasible strategy to bring about favorable adaptations in muscle structure and mobility with clinically insignificant serum CK and muscle pain levels following training sessions.[69] In this landmark study, 10 patients with PD (H&Y 1-3) between the ages of 40 and 85 engaged in a 12-week course of high-force eccentric resistance training on an eccentric ergometer 3 times per week[69] and responded similarly to both young[70] and frail elderly people[71,72] without PD who were exposed to high-force eccentric exercise. These findings illustrate the benefits of high force eccentric training for patients with PD due to the high levels of muscle force generated in eccentric exercise with associated low metabolic demands.[73]

Corcos et al.[74] conducted a two-year RCT involving pairs of patients with PD who were matched by age, sex, disease stage, and off-medication UPDRS-III, and engaged in either progressive resistance exercise (PRE), which consisted of a weight lifting program, or the Fitness Counts (FC) exercise program, recommended by the Parkinson’s Foundation[75], which focuses on performing stretches, balance exercises, breathing, and non-progressive strengthening exercises. Study subjects engaged in exercise twice per week for 24 months at a gym under the supervision of a personal trainer, and were followed for 24 months at 6-month intervals. At the conclusion of the study, the mean off-medication UPDRS-III score decreased more with the PRE group than with the FC group (mean difference: - 7·3 points; 95% CI: -11·3 to -3·6; $p<0.001$), denoting a significant improvement in motor signs, strength, and movement speed, and physical function.

Postural Correction

Common postural abnormalities occurring among patients with PD include camptocormia, antecollis, Pisa syndrome, and scoliosis.[76] While no formal treatment has been established to correct postural abnormalities associated with PD, which may contribute to LBP symptoms, several studies have demonstrated positive clinical outcomes with conservative care.

A retrospective pilot study by Lee et al.[77] involving 9 patients with PD (6 inpatient, 3 outpatient) (H&Y stages 2-4) with camptocormia who engaged in core strengthening and back extensor strengthening exercises of varying frequencies (once per week for 3 months v twice daily for 5 weeks) in addition to walking with a weighted low-slung backpack showed that 8/9 patients achieved a mean camptocormia flexion angle reduction of 20º ($p=0.012$) at 3 months post-treatment, in addition to mean changes of 2.788 ($p=0.011$) and 3.788 ($p=0.012$) in UPDRS II and III scores, respectively, reflecting a clinically important difference.[78]

Ye et al.[79] reported a case of a 70-year-old man with PD and hyperflexion of the thoracolumbar supine, which would resolve on recumbent positioning. The patient was instructed to perform neck and back extensor exercises daily for 30 minutes and was fitted with a cruciform anterior spinal hyperextension brace (CASH). After four months, his functional ambulation categories (FAC) score improved from 3 to 5 and his TUG improved from 16 seconds to 9 seconds. At the end of a 5-month follow-up, he was reportedly able to maintain an improved posture without the brace.

A pilot study conducted by Lena et al.[80] followed 6 patients with PD (H&Y 2-3) with Pisa syndrome, and lateral flexion of the trunk (LFT) >10º, who engaged in 10 consecutive 90-minute exercise sessions, under physiotherapist supervision, to correct postural trunk deviation. The patients presented with one of two patterns of muscle hyperactivity, confirmed by EMG; either ipsilateral or contralateral to the bending side. At the conclusion of the intervention, all participants demonstrated improvement in UPDRS-II, UPDRS-III, back pain rating, and LFT degree. Only 1 participant did not show improvement on UPDRS-III.

Gandolfi et al.[81] conducted a single-blind RCT involving 37 patients with PD (H&Y ≤4) and ≥ 5º of forward trunk flexion (FTF) to determine whether a trunk-specific rehabilitation program was more efficacious than conventional exercise at improving both postural orientation and FTF severity. The experimental group (n=19) engaged in 60-minute individualized sessions consisting of active self-correction exercises, trunk stabilization exercises, and functional tasks. The control group (n=18) intervention consisted of joint mobilization, muscle strengthening and stretching, and overground training and balance exercises. Participants in both groups engaged in individualized 60-minute sessions with a physiotherapist twice daily for 4 weeks. FTF significantly improved in the experimental group at the end of the intervention course ($p=0.003$) and at the 1-month follow-up ($p=0.004$).

Flexibility

Engaging in regular stretching is widely accepted as an essential component of a sound PD exercise program[82,83], though an optimal dose of stretch therapy remains unknown. When coupled with resistance exercise, stretching helps diminish muscle rigidity.[84] Some evidence demonstrates positive acute effects of stretching, as seen in a recent RCT by Vialleron et al.[85] In this study, 19 age-matched patients with PD (H&Y 2&3) were randomly allocated to a stretching group (n=10); who were exposed to 4 minutes (4 sets x 60 seconds) of triceps surae stretching, a sham stretching group (n=9); who were exposed to 4 minutes of forearm stretching, and a control group consisting of 10 age-matched healthy older adults who were not exposed to any form of stretching. The acute effects of the triceps surae stretching intervention demonstrated appreciable improvements in ankle mobility and gait initiation with large effect sizes ($d \geq 0.8$). The authors concluded that triceps surae stretching ought to be integrated into a multicomponent exercise program to include strength, balance, and gait training. This notion comports with the findings reported in an RCT by Santos and colleagues[86], in which patients with PD (H&Y 1&2) with akinesia and rigidity (AR-subtype), also defined as the "postural instability gait difficulty subtype" (PIGD- subtype)[87], engaged in 16 PRE training sessions over 8 weeks, beginning with workloads at 40-50% of their tested 1 repetition maximum (1RM) and progressing to 80-85% of 1RM. The PRE group realized significant improvements in static posturography, gait, and quality of life when compared to AR-subtype patients with PD with matched baseline characteristics in the control group.

Balance

PD-related axial rigidity also causes various biomechanical impairments that result in unfavorable postural alterations such as abnormally stooped posture, which impacts range of motion, brings about low back pain, and negatively impacts postural control[88,89]. Because stooped posture has a destabilizing effect on postural control, and the condition may not always be fully corrected with exercise or adjunct therapy, patients with PD need to retrain their spatial awareness with the postural alterations that they have acquired in order to improve their balance and gait so as to prevent falls. Balance training, of various forms, has been studied in isolation and in combination with resistance training for individuals with early-to-mid stage PD with the general consensus being that highly challenging balance exercises yield the greatest positive effects[90-94]. More recently, Capato and colleagues conducted an RCT to evaluate the effectiveness of balance training in individuals with advanced-stage PD (H&Y 4). The study results demonstrated that patients who engaged in 10 45-minute balance training sessions twice weekly, over a 5-week period, supported by RAS, realized significant improvements in balance and fall prevention when compared to age- and stage-matched controls who engaged in the same balance training program but without RAS support. Furthermore, the improvements were retained at 1-month and 6-month follow-ups.[95] These findings are encouraging and suggest that improving balance and preventing falls is still possible and a worthwhile goal at this stage of PD progression.

An RCT conducted by Silva-Batista and colleagues[96] compared the effects of three types of exercise on balance improvement in patients with PD (H&Y 2&3). Patients were randomized into a resistance training group (RT) (n=13), a resistance training with instability group (RTI) (n=13), and a placebo-control group (n=13). Study participants in the RT and RTI groups engaged in challenging exercises that demanded standing and balance for execution twice per week for 12 weeks. The RTI group also utilized various devices such as a BOSU ball and balance discs to progressively induce instability that challenged balance and postural control during resistance exercise. The control group engaged in upper body machine-based exercises that did not require a standing or balance component for execution, at the same frequency and duration as the other groups. While both the RT and RTI groups showed improvements in muscle strength, only the RTI group produced significant improvements in balance testing per the BESTest; a widely used clinical balance examination with good validity and reliability.[97-99] The RTI group showed improved post-intervention balance test scores with a mean difference of 18% (95% CI=12.7-23.4, $p < 0.001$), while the RT group showed no significant changes in balance performance (MD=2.9%; CI=−2.4 to 8.2; $p=0.579$), and the control group showed a significant decrease in balance performance (MD=−6.0%; CI=−11.4 to −0.7; $p=0.018$). The authors concluded that the balance improvements realized by the RTI group came as a result of increased force production capacity of the plantar flexors and knee extensors, in addition to enhanced anticipatory postural adjustments. The RTI group also demonstrated a reduced fear of falling ($p<0.05$), supporting the notion that challenging balance training in combination with resistance training can decrease barriers to exercise and allow individuals with PD to become more confident in executing demanding motor tasks.

Complementary Physical Interventions

In addition to the aforementioned exercise modalities, various complementary physical interventions have been shown to confer improvements in both motor and non-motor PD symptoms. Tai Chi and Qigong, mind-body intervention with roots in martial arts training, have demonstrated small-to-medium effects on motor function and balance.[100-106] Various styles of dance have been shown to improve balance and motor symptom severity in individuals with mild to moderate PD.[107-110] Technologies that promote healthy behaviors by combining video games and exercise have also been employed in the context of PD rehabilitation, with evidence demonstrating some effectiveness in the utilization of these technologies to improve balance, functional mobility, and cognitive skills.[111-114] Aerobic boxing programs for patients with PD have surged in popularity and have been promoted as an effective means to improve PD motor symptoms.[115,116] Though, recent reports suggest that the utilization of boxing as an established intervention for PD symptom management has expanded beyond the current evidence to substantiate its utilization.[117]

DISCUSSION

Exercise Prescription for LBP in Patients with PD

While no evidence currently exists to suggest that specific exercises or a specific exercise program is superior to another in the context of managing low back pain in the context of PD, strong evidence supports the recommendation of programs prescribed for patients with PD that involve at least 3 hours of exercise per week, as prescribed by the American College of Sports Medicine's Physical Activity Guidelines for Adults[118] and the U.S. Department of Health and Human Services Physical Activity Guidelines for Americans[119], which recommend engagement in a minimum of 150 to 300 minutes per week of moderate-intensity aerobic physical activity or 75 to 150 minutes per week of vigorous-intensity aerobic physical activity or a combination of both and muscle building and bone strengthening exercises a minimum of 2 days per week. In addition to the aforementioned recommendations, we have modified and adapted our exercise prescriptions to also include flexibility and balance training as supported by several high-quality studies[120-122], as well as prescription exercises for postural correction in those patients who present with postural deformities.[77,79-81] It is the responsibility of clinicians to tailor the specific exercise programs chosen for each patient, based on individual physical deficits and impairments. Therefore, the exercises selected must be meaningful to each patient and relevant for them to carry out their activities of daily living. Working with experienced rehabilitation professionals at the outset in a skilled exercise facility is crucial to reduce the risk of falls and to ensure that the prescribed exercises are appropriate and well-tolerated. Providing patient and family education regarding exercise therapy, precautions, and injury/fall prevention, in addition to enhancing family and community support and advice regarding exercise adherence will help to improve clinical outcomes.

We have stratified general exercise prescriptions to improve pain control, general fitness, and gait performance, in addition to enhancement of balance and flexibility, and improved postural correction, where necessary, across the 5 H&Y stages of PD.

For individuals with early-stage PD (H&Y 1-2)

The general goals of treatment during the early stage of PD are to educate patients about the disease, promote increased activity and improve strength, endurance, balance, and flexibility to enhance functional capacity. Exercise recommendations doses for patients with PD in these stages are aligned with the American College of Sports Medicine's Physical Activity Guidelines for Adults[123] with modifications to include daily engagement in stretching exercises, and balance training performed 3 days per week. Complete exercise prescription recommendations for patients with PD in these stages (H&Y 1-2) are summarized in **Table 2**. These recommendations are modified and adapted to also include balance training and postural correction exercises, where necessary.

For individuals with mid-stage PD (H&Y 3)

The general goals of treatment during the mid-stage of PD are to preserve early-stage goals in addition to improving transfer, gait, and reaching performance, improving posture, improving steadiness, freezing control, and fall prevention in addition to caregiver education.

For individuals with late-stage PD (H&Y 4-5)

The general goals of treatment during the late stage of PD are to preserve mid-stage goals in addition to maintaining vital functions, preventing contractures and pressure sores, and educating caregivers about bed mobility, transfer, ADLs, and exercise.

Complete exercise prescription recommendations for patients with mid-to-late-stage PD (H&Y 3-5) are summarized in **Table 3**. These recommendations are modified and adapted to also include balance training and postural correction exercises, where necessary, and align with the most recent recommendations made by the ACSM's Guidelines for Persons with Chronic Diseases and Disabilities (Basic CDD4 Recommendations).[124]

Table 2. Exercise Prescription for PD H&Y 1&2 modified and adapted from American College of Sports Medicine Physical Activity Guidelines for Adults
RPE based on modified RPE scale

Mode	Frequency	Duration	Intensity	Progression
Warm-up and cool-down	Before and after each session	10-15 min	Easy RPE <3/10	Should be maintained as transition phase, especially for those doing higher-intensity physical activity
Aerobic Training • Large-muscle easily accessible activities such as walking as the basic program • Types include walking, jogging, cycling, swimming/water-based exercise, or other enjoyable physical activities that may be sustained over a prolonged period of time	5+ days/ week	Start at any duration, as tolerated Ultimate weekly goal is to build up to a minimum of 150 to 300 minutes per week of moderate-intensity aerobic physical activity or 75 to 150 minutes per week of vigorous-intensity aerobic physical activity or a combination of both	Light (casual walk) RPE of 2 Moderate (brisk walk) RPE of 3 Vigorous (like jogging) RPE of 4-6	Progression week over week in either intensity, volume, or a combination of both should not exceed 10%
Strength Straining • High force eccentric training • Functional gravity-based exercises • Weight training is an alternative for those who are interested and motivated to do it	2-3 days/ week	8-12 repetitions x 2-4 sets	RPE of 3-7	Build gradually to as many sets a day as tolerated
Stretch Therapy Major muscle groups and calf stretches	Daily	60 s/stretch, repeat 3x Entire stretching session ~ 30 min	Maintain stretch below discomfort point	Discomfort point should occur at a ROM that does not cause instability. This discomfort point will vary between people and with different joints in each person.
Balance Training	3 days/ week	~45 min	Somewhat challenging	Supervised side to side sways and weight shifting, along with variable footwork exercises with the support of walkers and other assistive devices
Postural Correction	1-7 days/week	20-90 min/session 1-2x/day per PT recommendation	To tolerance	Supervised exercises to correct postural trunk deviation, individually tailored to the patient in addition to wearing a weighted low-slung backpack or brace, as needed

Table 3. Exercise Prescription for PD H&Y 3-5 modified and adapted from Basic CDD4 Recommendations
RPE based on modified RPE scale

Mode	Frequency	Duration	Intensity	Progression
Warm-up and cool-down	Before and after each session	10-15 min	Easy RPE <3/10	Should be maintained as transition phase, especially for those doing higher-intensity physical activity
Aerobic Training • Large-muscle easily accessible activities such as walking as the basic program • Types include walking, jogging, cycling, swimming/water-based exercise, or other enjoyable physical activities that may be sustained over a prolonged period of time • Walking under supervision with assistive devices and safety harness	5+ days/ week	Start at any duration, as tolerated Ultimate weekly goal is to build up to a minimum of 150 to 300 minutes per week of moderate-intensity aerobic physical activity or 75 to 150 minutes per week of vigorous-intensity aerobic physical activity or a combination of both	Light (casual walk) RPE of 2 Moderate (brisk walk) RPE of 3 Vigorous (like jogging) RPE of 4-6	Progression week over week in either intensity, volume, or a combination of both should not exceed 10%
Strength Straining • High force eccentric training • Functional gravity-based exercises (avoid free weights in advanced PD) • Weight training is an alternative for those who are interested and motivated to do it	2-3 days/ week	8-12 repetitions x 2-4 sets	RPE of 3-7	Build gradually to as many sets a day as tolerated
Stretch Therapy Major muscle groups and calf stretches Independent and assisted stretching exercises	Daily	60 s/stretch, repeat 3x Entire stretching session ~ 30 min	Maintain stretch below discomfort point	Discomfort point should occur at a ROM that does not cause instability. This discomfort point will vary between people and with different joints in each person.
Balance Training	3 days/ week	~45 min	Somewhat challenging	Supervised side to side sways and weight shifting, along with variable footwork exercises with the support of walkers and other assistive devices
Postural Correction	1-7 days/week	20-90 min/session 1-2x/day per PT recommendation	To tolerance	Supervised exercises to correct postural trunk deviation, individually tailored to the patient in addition to wearing a weighted low-slung backpack or brace, as needed

Barriers to Exercise Engagement and Strategies to Enhance Exercise Adherence

Perceived barriers to exercise engagement are predictive of exercise behavior. The most commonly reported perceived barriers to exercise in healthy older adults include lack of interest, poor health, weakness, fear of falling, pain, bad weather, lack of time, and limited access to exercise resources.[125] In the context of PD, these aforementioned concerns in addition to scheduling issues, commuting challenges, medical comorbidities, hospitalizations, family demands, and low outcome expectations appear to be the major perceived barriers to exercise.[126] Moreover, a lack of structured community-based PD-specific exercise programs may further reduce the motivation for engagement, though efforts are being made to adapt and scale such exercise programs to serve the needs of patients with PD.[127]

Working with an experienced physical therapist, rehabilitation-oriented chiropractor, and/or physical trainer who understands how to optimally dose exercise prescription for each individual in order to ensure that engaging in exercise is tolerable and sustainable has been shown to enhance exercise adherence in this patient population.[128] Once individuals are able to engage in exercise independently, booster supervised sessions have been shown to improve exercise adherence.[129]

Exercise snacking, a strategy that calls for breaking up daily exercise recommendations into several shorter bouts of physical activity or “exercise snacks” throughout the day has demonstrated improvements in cardiorespiratory fitness, metabolic markers of health and muscle mass and function in small proof of concept studies[130-132]. This strategy may prove to be worthwhile in enhancing exercise adherence among people with PD.

Autoregulation

Autoregulation describes the means of selecting and adjusting training intensity based on one’s performance during a given training session. Because consistency of effort during every exercise session is unlikely for a variety of reasons, in order to maintain adherence to exercise, rate of perceived exertion (RPE), a validated and reliable psycho-physical tool may be taught to patients for them to assess their subjective perception of effort exerted during physical activity in order to allow them to adjust their level of exertion during physical training based on real-time psychological and physiological feedback (**Table 4**).[133]

Table 4. Modified RPE Scale Reproduced from (Haddad 2017)

Rating	Descriptor
0	Rest
1	Very, Very Easy
2	Easy
3	Moderate
4	Somewhat Hard
5	Hard
6	
7	Very Hard
8	
9	
10	Maximal

CONCLUSION

Low back pain in the context of PD is a complex condition that may be influenced by pathology, pain intensity, physical impairment, and psychosocial reinforcements, resulting in disability. To date, multiple lines of empirical evidence demonstrate that activities and exercises that challenge physical impairments result in improvements in low back pain and quality of life among individuals with PD. The exercise prescription recommendations outlined in this article exemplify an approach that combines strength training, aerobic training, stretching, and balance training, in addition to postural correction exercises, where necessary, that is modified across the stages of PD progression based on the current body of knowledge available at this time. Future high-quality studies with longer intervention and follow-up periods would help to further ascertain the effects of exercise dose and type on managing both LBP and PD progression in addition to exploring strategies to enhance behavior modification in order to improve adherence, and ultimately, outcomes.

AUTHOR CONTRIBUTIONS

Conceptualization, J.G., H.W..; methodology, J.G., H.W.; formal analysis, J.G., G.G.; investigation, J.G., G.G.; original draft preparation, J.G., G.G., H.W.; writing—review and editing, J.G., G.G., H.W., E.G., E.A. All authors have read and agreed to the published version of the manuscript.

FUNDING

This research received no external funding.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

REFERENCES

1. Edinoff A, Sathivadivel N, McBride T, et al. Chronic Pain Treatment Strategies in Parkinson's Disease. *Neurol Int*. 2020;12(3):61-76. Published 2020 Nov 18. Doi:10.3390/neurolint12030014
2. GBD 2016 Parkinson's Disease Collaborators. Global, regional, and national burden of Parkinson's disease, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016 [published correction appears in Lancet Neurol. 2021 Dec;20(12):e7]. *Lancet Neurol*. 2018;17(11):939-953. Doi:10.1016/S1474-4422(18)30295-3
3. Pringsheim T, Jette N, Frolkis A, Steeves TD. The prevalence of Parkinson's disease: a systematic review and meta-analysis. *Mov Disord*. 2014;29(13):1583-1590. Doi:10.1002/mds.25945
4. Dorsey ER, Sherer T, Okun MS, Bloem BR. The Emerging Evidence of the Parkinson Pandemic. *J Parkinsons Dis*. 2018;8(s1):S3-S8. Doi:10.3233/JPD-181474

5. Belvisi D, Pellicciari R, Fabbrini G, Tinazzi M, Berardelli A, Defazio G. Modifiable risk and protective factors in disease development, progression and clinical subtypes of Parkinson's disease: What do prospective studies suggest?. *Neurobiol Dis*. 2020;134:104671. Doi:10.1016/j.nbd.2019.104671
6. Ford B. Pain in Parkinson's disease. *Clin Neurosci*. 1998;5(2):63-72.
7. Beiske AG, Loge JH, Ronningen A, Svensson E. Pain in Parkinson's disease: prevalence and characteristics. *PAIN* 2009;141:173–7.
8. Galazky I, Caspari C, Heinze HJ, Franke J. The prevalence of chronic low back pain and lumbar deformities in patients with Parkinson's disease: implications on spinal surgery. *Eur Spine J*. 2018;27(11):2847-2853. Doi:10.1007/s00586-018-5748-0
9. Global Burden of Disease Study 2013 Collaborators. Global, regional, and national incidence, prevalence, and years lived with disability for 301 acute and chronic diseases and injuries in 188 countries, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet*. 2015;386(9995):743-800. Doi:10.1016/S0140-6736(15)60692-4
10. Dieleman JL, Cao J, Chapin A, et al. US Health Care Spending by Payer and Health Condition, 1996-2016. *JAMA*. 2020;323(9):863-884. Doi:10.1001/jama.2020.0734
11. Zafar S, Yaddanapudi SS. Parkinson Disease. [Updated 2021 Aug 11]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK470193/
12. Goetz CG, Poewe W, Rascol O, et al. Movement Disorder Society Task Force report on the Hoehn and Yahr staging scale: status and recommendations. *Mov Disord*. 2004;19(9):1020-1028. doi:10.1002/mds.20213
13. Fahn S, Marsden CD, Calne DB, Goldstein M. Recent Developments in Parkinson's Disease, Vol 2. Florham Park, NJ. Macmillan Health Care Information 1987, pp 15 3-163, 293-304
14. Hallett M. Parkinson revisited: pathophysiology of motor signs. *Adv Neurol*. 2003;91:19-28.
15. Watanabe K, Hirano T, Katsumi K, et al. Characteristics of spinopelvic alignment in Parkinson's disease: Comparison with adult spinal deformity. *J Orthop Sci*. 2017;22(1):16-21. Doi:10.1016/j.jos.2016.09.013
16. Burkey AR, Carstens E, Jasmin L. Dopamine reuptake inhibition in the rostral agranular insular cortex produces antinociception. *J Neurosci*. 1999;19(10):4169-4179. Doi:10.1523/JNEUROSCI.19-10-04169.1999
17. Chudler EH, Dong WK. The role of the basal ganglia in nociception and pain. *PAIN*. 1995;60(1):3-38. Doi:10.1016/0304-3959(94)00172-B
18. Natalie E. Allen, PhD, Cassandra M. Wong, BappSc (Phty) Hons, Colleen G. Canning, PhD, Niamh Moloney, PhD, The Association Between Parkinson's Disease Motor Impairments and Pain, *Pain Medicine*, Volume 17, Issue 3, March 2016, Pages 456–462, https://doi.org/10.1111/pme.12898
19. Margraf NG, Wrede A, Rohr A, et al. Camptocormia in idiopathic Parkinson's disease: a focal myopathy of the paravertebral muscles. *Mov Disord*. 2010;25(5):542-551. Doi:10.1002/mds.22780
20. Yuan H, Zhang ZW, Liang LW, et al. Treatment strategies for Parkinson's disease. *Neurosci Bull*. 2010;26(1):66-76. Doi:10.1007/s12264-010-0302-z

21. Rogers G, Davies D, Pink J, Cooper P. Parkinson’s disease: summary of updated NICE guidance [published correction appears in BMJ. 2019 Feb 28;364:l961]. *BMJ*. 2017;358:j1951. Published 2017 Jul 27. Doi:10.1136/bmj.j1951
22. Rosenquist PB, Youssef NA, Surya S, McCall WV. When All Else Fails: The Use of Electroconvulsive Therapy for Conditions Other than Major Depressive Episode. *Psychiatr Clin North Am*. 2018;41(3):355-371. Doi:10.1016/j.psc.2018.04.002
23. Mari Z, Mestre TA. The Disease Modification Conundrum in Parkinson’s Disease: Failures and Hopes. *Front Aging Neurosci*. 2022;14:810860. Published 2022 Feb 28. Doi:10.3389/fnagi.2022.810860
24. Ellis TD, Colón-Semenza C, DeAngelis TR, et al. Evidence for Early and Regular Physical Therapy and Exercise in Parkinson’s Disease. *Semin Neurol*. 2021;41(2):189-205. Doi:10.1055/s-0041-1725133
25. Paul KC, Chuang YH, Shih IF, et al. The association between lifestyle factors and Parkinson’s disease progression and mortality. *Mov Disord*. 2019;34(1):58-66. Doi:10.1002/mds.27577
26. Cotman CW, Berchtold NC, Christie LA. Exercise builds brain health: key roles of growth factor cascades and inflammation [published correction appears in Trends Neurosci. 2007 Oct;30(10):489]. *Trends Neurosci*. 2007;30(9):464-472. Doi:10.1016/j.tins.2007.06.011
27. Monteiro-Junior RS, Cevada T, Oliveira BR, et al. We need to move more: Neurobiological hypotheses of physical exercise as a treatment for Parkinson’s disease. *Med Hypotheses*. 2015;85(5):537-541. Doi:10.1016/j.mehy.2015.07.011
28. Smeyne RJ, Jackson-Lewis V. The MPTP model of Parkinson’s disease. *Brain Res Mol Brain Res*. 2005;134(1):57-66. Doi:10.1016/j.molbrainres.2004.09.017
29. Sleiman SF, Henry J, Al-Haddad R, et al. Exercise promotes the expression of brain derived neurotrophic factor (BDNF) through the action of the ketone body β-hydroxybutyrate. *Elife*. 2016;5:e15092. Published 2016 Jun 2. Doi:10.7554/eLife.15092
30. Palasz E, Niewiadomski W, Gasiorowska A, Mietelska-Porowska A, Niewiadomska G. Neuroplasticity and Neuroprotective Effect of Treadmill Training in the Chronic Mouse Model of Parkinson’s Disease. *Neural Plast*. 2019;2019:8215017. Published 2019 Apr 3. Doi:10.1155/2019/8215017
31. Gerecke KM, Jiao Y, Pagala V, Smeyne RJ. Exercise does not protect against MPTP-induced neurotoxicity in BDNF haploinsufficient mice. *PloS One*. 2012;7(8):e43250. Doi:10.1371/journal.pone.0043250
32. Real CC, Ferreira AF, Chaves-Kirsten GP, Torrão AS, Pires RS, Britto LR. BDNF receptor blockade hinders the beneficial effects of exercise in a rat model of Parkinson’s disease [published correction appears in Neuroscience. 2013 Sep 5;247:319-20]. *Neuroscience*. 2013;237:118-129. Doi:10.1016/j.neuroscience.2013.01.060
33. Gerecke KM, Jiao Y, Pani A, Pagala V, Smeyne RJ. Exercise protects against MPTP-induced neurotoxicity in mice. *Brain Res*. 2010;1341:72-83. Doi:10.1016/j.brainres.2010.01.053
34. Koo JH, Jang YC, Hwang DJ, et al. Treadmill exercise produces neuroprotective effects in a murine model of Parkinson’s disease by regulating the TLR2/MyD88/NF-κB signaling pathway. *Neuroscience*. 2017;356:102-113. Doi:10.1016/j.neuroscience.2017.05.016

35. Shulman LM, Katzel LI, Ivey FM, et al. Randomized clinical trial of 3 types of physical exercise for patients with Parkinson disease. *JAMA Neurol*. 2013;70(2):183-190. Doi:10.1001/jamaneurol.2013.646
36. Lima LV, Abner TSS, Sluka KA. Does exercise increase or decrease pain? Central mechanisms underlying these two phenomena. *J Physiol*. 2017 Jul 1;595(13):4141-4150. Doi: 10.1113/JP273355. Epub 2017 May 26. PMID: 28369946; PMCID: PMC5491894.
37. Dowell D, Haegerich TM, Chou R. CDC Guideline for Prescribing Opioids for Chronic Pain—United States, 2016. *JAMA*. 2016;315(15):1624-1645. Doi:10.1001/jama.2016.1464
38. McNally RJ. Mechanisms of exposure therapy: how neuroscience can improve psychological treatments for anxiety disorders. *Clin Psychol Rev*. 2007;27(6):750-759. doi:10.1016/j.cpr.2007.01.003
39. Schmid J, Theysohn N, Ga F, et al. Neural mechanisms mediating positive and negative treatment expectations in visceral pain: a functional magnetic resonance imaging study on placebo and nocebo effects in healthy volunteers. *PAIN*. 2013;154(11):2372-2380. doi:10.1016/j.pain.2013.07.013
40. Tryon WW. Possible mechanisms for why desensitization and exposure therapy work. *Clin Psychol Rev*. 2005;25(1):67-95. doi:10.1016/j.cpr.2004.08.005
41. Kapitzke D, Vetter I, Cabot PJ. Endogenous opioid analgesia in peripheral tissues and the clinical implications for pain control. *Ther Clin Risk Manag*. 2005;1(4):279-297.
42. Petersen AM, Pedersen BK. The anti-inflammatory effect of exercise. *J Appl Physiol (1985)*. 2005;98(4):1154-1162. doi:10.1152/japplphysiol.00164.2004
43. Svensson M, Lexell J, Deierborg T. Effects of Physical Exercise on Neuroinflammation, Neuroplasticity, Neurodegeneration, and Behavior: What We Can Learn From Animal Models in Clinical Settings. *Neurorehabil Neural Repair*. 2015;29(6):577-589. doi:10.1177/1545968314562108
44. Steensberg A, Fischer CP, Keller C, Møller K, Pedersen BK. IL-6 enhances plasma IL-1ra, IL-10, and cortisol in humans. *Am J Physiol Endocrinol Metab*. 2003;285(2):E433-E437. doi:10.1152/ajpendo.00074.2003
45. Oliveira de Carvalho A, Filho ASS, Murillo-Rodriguez E et al (2018) Physical exercise for Parkinson's disease: clinical and experimental evidence. *Clin Pract Epidemiol Ment Health* 14:89–98. https://doi.org/10.2174/1745017901814010089
46. Kim Y, Lai B, Mehta T et al (2019) Exercise training guidelines for multiple sclerosis, stroke, and Parkinson disease: rapid review and synthesis. *Am J Phys Med Rehabil* 98:613–621. https://doi.org/10.1097/PHM.0000000000001174
47. McGraw SM, Hoover DL, Shirey MP (2013) Exercise guidelines for patients with Parkinson's disease. *Home Health Care Manag Pract* 26:167–174. https://doi.org/10.1177/1084822313514977
48. Nadeau A, Pourcher E, Corbeil P (2014) Effects of 24 wk of treadmill training on gait performance in Parkinson's disease. *Med Sci Sports Exerc* 46:645–655. https://doi.org/10.1249/MSS.0000000000000144
49. Bello O, Fernandez-Del-Olmo M. How does the treadmill affect gait in Parkinson's disease? *Curr Aging Sci*. 2012;5:28-34.
50. Zhao M, Veeranki SP, Magnussen CG, Xi B. Recommended physical activity and all cause and cause specific mortality in US adults: prospective cohort study. *BMJ*. 2020;370:m2031. Published 2020 Jul 1. doi:10.1136/bmj.m2031

51. Bello O, Marquez G, Camblor M, Fernandez-Del-Olmo M. Mechanisms involved in treadmill walking improvements in Parkinson’s disease. *Gait Posture*. 2010;32:118-123.
52. Arcolin I, Pisano F, Delconte C, et al. Intensive cycle ergometer training improves gait speed and endurance in patients with Parkinson’s disease: a comparison with treadmill training. *Restorative Neurol Neurosci*. 2015;34:125-138.
53. Calabrò RS, Naro A, Filoni S, et al. Walking to your right music: a randomized controlled trial on the novel use of treadmill plus music in Parkinson's disease. *J Neuroeng Rehabil*. 2019;16(1):68. Published 2019 Jun 7. doi:10.1186/s12984-019-0533-9
54. Shnayderman I, Katz-Leurer M. An aerobic walking programme versus muscle strengthening programme for chronic low back pain: a randomized controlled trial. *Clin Rehabil*. 2013;27(3):207-214. doi:10.1177/0269215512453353
55. Chatzitheodorou D, Kabitsis C, Malliou P, Mougios V. A pilot study of the effects of high-intensity aerobic exercise versus passive interventions on pain, disability, psychological strain, and serum cortisol concentrations in people with chronic low back pain. *Phys Ther*. 2007;87(3):304-312. doi:10.2522/ptj.20060080
56. Chatzitheodorou D, Mavromoustakos S, Milioti S. The effect of exercise on adrenocortical responsiveness of patients with chronic low back pain, controlled for psychological strain. *Clin Rehabil*. 2008;22(4):319-328. doi:10.1177/0269215507079858
57. Timmers I, Kaas AL, Quaedflieg CWEM, Biggs EE, Smeets T, de Jong JR. Fear of pain and cortisol reactivity predict the strength of stress-induced hypoalgesia. *Eur J Pain*. 2018;22(7):1291-1303. doi:10.1002/ejp.1217
58. Bagnato G, Cordova F, Sciortino D, et al. Association between cortisol levels and pain threshold in systemic sclerosis and major depression. *Rheumatol Int*. 2018;38(3):433-441. doi:10.1007/s00296-017-3866-3
59. Chen C, Nakagawa S, An Y, Ito K, Kitaichi Y, Kusumi I. The exercise-glucocorticoid paradox: How exercise is beneficial to cognition, mood, and the brain while increasing glucocorticoid levels. *Front Neuroendocrinol*. 2017;44:83-102. doi:10.1016/j.yfrne.2016.12.001
60. Hill EE, Zack E, Battaglini C, Viru M, Viru A, Hackney AC. Exercise and circulating cortisol levels: the intensity threshold effect. *J Endocrinol Invest*. 2008;31(7):587-591. doi:10.1007/BF03345606
61. Harvey M, Weston KL, Gray WK, et al. High-intensity interval training in people with Parkinson's disease: a randomized, controlled feasibility trial. *Clin Rehabil*. 2019;33(3):428-438. doi:10.1177/0269215518815221
62. Salgado S, Williams N, Kotian R (2013) An evidence-based exercise regimen for patients with mild to moderate Parkinson’s disease. *Brain Sci* 3:87–100. https ://doi.org/10.3390/brain sci3010087
63. Nguy V, Barry BK, Moloney N, et al. Exercise-induced hypoalgesia is present in people with Parkinson's disease: Two observational cross-sectional studies. *Eur J Pain*. 2019;23(7):1329-1339. doi:10.1002/ejp.1400
64. Mcleod JC, Stokes T, Phillips SM. Resistance Exercise Training as a Primary Countermeasure to Age-Related Chronic Disease. *Front Physiol*. 2019;10:645. Published 2019 Jun 6. doi:10.3389/fphys.2019.00645
65. Saeidifard F, Medina-Inojosa JR, West CP, et al. The association of resistance training with mortality: A systematic review and meta-analysis. *Eur J Prev Cardiol*. 2019;26(15):1647-1665. doi:10.1177/2047487319850718

66. Kamada M, Shiroma EJ, Buring JE, Miyachi M, Lee IM. Strength Training and All-Cause, Cardiovascular Disease, and Cancer Mortality in Older Women: A Cohort Study. *J Am Heart Assoc*. 2017;6(11):e007677. Published 2017 Oct 31. doi:10.1161/JAHA.117.007677
67. Momma H, Kawakami R, Honda T*, et al* Muscle-strengthening activities are associated with lower risk and mortality in major non-communicable diseases: a systematic review and meta-analysis of cohort studies. *British Journal of Sports Medicine* Published Online First: 28 February 2022. doi: 10.1136/bjsports-2021-105061
68. Emmanuel Stamatakis, I -Min Lee, Jason Bennie, Jonathan Freeston, Mark Hamer, Gary O'Donovan, Ding Ding, Adrian Bauman, Yorgi Mavros, Does Strength-Promoting Exercise Confer Unique Health Benefits? A Pooled Analysis of Data on 11 Population Cohorts With All-Cause, Cancer, and Cardiovascular Mortality Endpoints. *American Journal of Epidemiology*. Volume 187, Issue 5, May 2018, Pages 1102–1112, https://doi.org/10.1093/aje/kwx345
69. Dibble LE, Hale T, Marcus RL, Gerber JP, Lastayo PC. The safety and feasibility of high-force eccentric resistance exercise in persons with Parkinson's disease. *Arch Phys Med Rehabil*. 2006;87(9):1280-1282. doi:10.1016/j.apmr.2006.05.016
70. Roig M, O'Brien K, Kirk G, et al. The effects of eccentric versus concentric resistance training on muscle strength and mass in healthy adults: a systematic review with meta-analysis. *Br J Sports Med*. 2009;43(8):556-568. doi:10.1136/bjsm.2008.051417
71. LaStayo PC, Ewy GA, Pierotti DD, Johns RK, Lindstedt S. The positive effects of negative work: increased muscle strength and decreased fall risk in a frail elderly population. *J Gerontol A Biol Sci Med Sci*. 2003;58(5):M419-M424. doi:10.1093/gerona/58.5.m419
72. LaStayo P, McDonagh P, Lipovic D, et al. Elderly patients and high force resistance exercise--a descriptive report: can an anabolic, muscle growth response occur without muscle damage or inflammation?. *J Geriatr Phys Ther*. 2007;30(3):128-134. doi:10.1519/00139143-200712000-00008
73. Peñailillo L, Blazevich AJ, Nosaka K. Factors contributing to lower metabolic demand of eccentric compared with concentric cycling. *J Appl Physiol (1985)*. 2017;123(4):884-893. doi:10.1152/japplphysiol.00536.2016
74. Corcos DM, Robichaud JA, David FJ, et al. A two-year randomized controlled trial of progressive resistance exercise for Parkinson's disease. *Mov Disord*. 2013;28(9):1230-1240. doi:10.1002/mds.25380
75. Fitness Counts – Parkinson's Foundation. Available from: https://www.parkinson.org/sites/default/files/documents/fitness-counts.pdf
76. Doherty KM, van de Warrenburg BP, Peralta MC, et al. Postural deformities in Parkinson's disease. *Lancet Neurol*. 2011;10(6):538-549. doi:10.1016/S1474-4422(11)70067-9
77. Lee KH, Kim JM, Kim HS. Back Extensor Strengthening Exercise and Backpack Wearing Treatment for Camptocormia in Parkinson's Disease: A Retrospective Pilot Study. *Ann Rehabil Med*. 2017;41(4):677-685. doi:10.5535/arm.2017.41.4.677
78. Shulman LM, Gruber-Baldini AL, Anderson KE, Fishman PS, Reich SG, Weiner WJ. The clinically important difference on the unified Parkinson's disease rating scale. *Arch Neurol*. 2010;67(1):64-70. doi:10.1001/archneurol.2009.295

79. Ye BK, Kim HS, Kim YW. Correction of camptocormia using a cruciform anterior spinal hyperextension brace and back extensor strengthening exercise in a patient with Parkinson disease. *Ann Rehabil Med*. 2015;39(1):128-132. doi:10.5535/arm.2015.39.1.128
80. Lena F, Iezzi E, Etoom M, et al. Effects of postural exercises in patients with Parkinson's disease and Pisa syndrome: A pilot study. *NeuroRehabilitation*. 2017;41(2):423-428. doi:10.3233/NRE-162033
81. Gandolfi M, Tinazzi M, Magrinelli F, et al. Four-week trunk-specific exercise program decreases forward trunk flexion in Parkinson's disease: A single-blinded, randomized controlled trial. *Parkinsonism Relat Disord*. 2019;64:268-274. doi:10.1016/j.parkreldis.2019.05.006
82. Gallo P, Ewing Garber C. PARKINSON’S DISEASE: A Comprehensive Approach to Exercise Prescription for the Health Fitness Professional. *ACSM’s Health & Fitness Journal*: July/August 2011 - Volume 15 - Issue 4 - p 8-17 doi: 10.1249/FIT.0b013e31821eca84
83. Ellis T, Rochester L. Mobilizing Parkinson's Disease: The Future of Exercise. *J Parkinsons Dis*. 2018;8(s1):S95-S100. doi:10.3233/JPD-181489
84. Ellis T, DeAngelis TR, Dalton D, Venne J (2016) Be active & beyond—a guide to exercise and wellness for people with Parkinson’s Disease. APDA. Available from: https://www.apdaparkinson.org/wp-content/uploads/pdf_publications/Be-Active-Book_For-Web-90o.pdf
85. Vialleron T, Delafontaine A, Millerioux I, Memari S, Fourcade P, Yiou E. Acute effects of short-term stretching of the triceps surae on ankle mobility and gait initiation in patients with Parkinson's disease. *Clin Biomech (Bristol, Avon)*. 2021;89:105449. doi:10.1016/j.clinbiomech.2021.105449
86. Santos L, Fernandez-Rio J, Winge K, et al. Effects of progressive resistance exercise in akinetic-rigid Parkinson's disease patients: a randomized controlled trial. *Eur J Phys Rehabil Med*. 2017;53(5):651-663. doi:10.23736/S1973-9087.17.04572-5
87. Zaidel A, Arkadir D, Israel Z, Bergman H. Akineto-rigid vs. tremor syndromes in Parkinsonism. *Curr Opin Neurol*. 2009;22(4):387-393. doi:10.1097/WCO.0b013e32832d9d67
88. Bloem BR, Beckley DJ, van Dijk JG. Are automatic postural responses in patients with Parkinson's disease abnormal due to their stooped posture?. *Exp Brain Res*. 1999;124(4):481-488. doi:10.1007/s002210050644
89. Jacobs JV, Dimitrova DM, Nutt JG, Horak FB. Can stooped posture explain multidirectional postural instability in patients with Parkinson's disease?. *Exp Brain Res*. 2005;166(1):78-88. doi:10.1007/s00221-005-2346-2
90. Albrecht F, Pereira JB, Mijalkov M, et al. Effects of a Highly Challenging Balance Training Program on Motor Function and Brain Structure in Parkinson's Disease. *J Parkinsons Dis*. 2021;11(4):2057-2071. doi:10.3233/JPD-212801
91. Joseph C, Brodin N, Leavy B, Hagströmer M, Löfgren N, Franzén E. Cost-effectiveness of the HiBalance training program for elderly with Parkinson's disease: analysis of data from a randomized controlled trial. *Clin Rehabil*. 2019;33(2):222-232. doi:10.1177/0269215518800832
92. Conradsson D, Löfgren N, Nero H, et al. The Effects of Highly Challenging Balance Training in Elderly With Parkinson's Disease: A Randomized Controlled Trial. *Neurorehabil Neural Repair*. 2015;29(9):827-836. doi:10.1177/1545968314567150

93. Leavy B, Joseph C, Löfgren N, Johansson H, Hagströmer M, Franzén E. Outcome Evaluation of Highly Challenging Balance Training for People With Parkinson Disease: A Multicenter Effectiveness-Implementation Study. *J Neurol Phys Ther*. 2020;44(1):15-22. doi:10.1097/NPT.0000000000000298
94. Sparrow D, DeAngelis TR, Hendron K, Thomas CA, Saint-Hilaire M, Ellis T. Highly Challenging Balance Program Reduces Fall Rate in Parkinson Disease. *J Neurol Phys Ther*. 2016;40(1):24-30. doi:10.1097/NPT.0000000000000111
95. Capato TTC, Nonnekes J, de Vries NM, IntHout J, Barbosa ER, Bloem BR. Effects of multimodal balance training supported by rhythmical auditory stimuli in people with advanced stages of Parkinson's disease: a pilot randomized clinical trial. *J Neurol Sci*. 2020;418:117086. doi:10.1016/j.jns.2020.117086
96. Silva-Batista C, Corcos DM, Kanegusuku H, et al. Balance and fear of falling in subjects with Parkinson's disease is improved after exercises with motor complexity. *Gait Posture*. 2018;61:90-97. doi:10.1016/j.gaitpost.2017.12.027
97. Horak FB, Wrisley DM, Frank J. The Balance Evaluation Systems Test (BESTest) to differentiate balance deficits. *Phys Ther*. 2009;89(5):484-498. doi:10.2522/ptj.20080071
98. Leddy AL, Crowner BE, Earhart GM. Functional gait assessment and balance evaluation system test: reliability, validity, sensitivity, and specificity for identifying individuals with Parkinson disease who fall. *Phys Ther*. 2011;91(1):102-113. doi:10.2522/ptj.20100113
99. Leddy AL, Crowner BE, Earhart GM. Utility of the Mini-BESTest, BESTest, and BESTest sections for balance assessments in individuals with Parkinson disease. *J Neurol Phys Ther*. 2011;35(2):90-97. doi:10.1097/NPT.0b013e31821a620c
100. Yu X, Wu X, Hou G, Han P, Jiang L, Guo Q. The Impact of Tai Chi on Motor Function, Balance, and Quality of Life in Parkinson's Disease: A Systematic Review and Meta-Analysis. *Evid Based Complement Alternat Med*. 2021;2021:6637612. Published 2021 Jan 11. doi:10.1155/2021/6637612
101. Zhong D, Xiao Q, Xiao X, et al. Tai Chi for improving balance and reducing falls: An overview of 14 systematic reviews. *Ann Phys Rehabil Med*. 2020;63(6):505-517. doi:10.1016/j.rehab.2019.12.008
102. Kedzior KK, Kaplan I. Tai Chi and Parkinson's disease (PD): A systematic overview of the scientific quality of the past systematic reviews. *Complement Ther Med*. 2019;46:144-152. doi:10.1016/j.ctim.2019.08.008
103. Aras B, Seyyar GK, Fidan O, Colak E. The effect of Tai Chi on functional mobility, balance and falls in Parkinson's disease: A systematic review and meta-analysis of systematic reviews. *Explore (NY)*. 2022;18(4):402-410. doi:10.1016/j.explore.2021.12.002
104. Song R, Grabowska W, Park M, et al. The impact of Tai Chi and Qigong mind-body exercises on motor and non-motor function and quality of life in Parkinson's disease: A systematic review and meta-analysis. *Parkinsonism Relat Disord*. 2017;41:3-13. doi:10.1016/j.parkreldis.2017.05.019
105. Chen S, Zhang Y, Wang YT, Liu X, Song W, Du X. The effect of Qigong-based therapy on patients with Parkinson's disease: a systematic review and meta-analysis. *Clin Rehabil*. 2020;34(12):1436-1448. doi:10.1177/0269215520946695
106. Kamieniarz A, Milert A, Grzybowska-Ganszczyk D, Opara J, Juras G. Tai Chi and Qi Gong therapies as a complementary treatment in Parkinson's disease - a systematic review. *Complement Ther Med*. 2021;56:102589. doi:10.1016/j.ctim.2020.102589

107. Carapellotti AM, Stevenson R, Doumas M. The efficacy of dance for improving motor impairments, non-motor symptoms, and quality of life in Parkinson's disease: A systematic review and meta-analysis. *PLoS One*. 2020;15(8):e0236820. Published 2020 Aug 5. doi:10.1371/journal.pone.0236820
108. Kalyani HHN, Sullivan K, Moyle G, et al. Effects of Dance on Gait, Cognition, and Dual-Tasking in Parkinson's Disease: A Systematic Review and Meta-Analysis. *J Parkinsons Dis*. 2019;9(2):335-349. doi:10.3233/JPD-181516
109. Barnish MS, Barran SM. A systematic review of active group-based dance, singing, music therapy and theatrical interventions for quality of life, functional communication, speech, motor function and cognitive status in people with Parkinson's disease. *BMC Neurol*. 2020;20(1):371. Published 2020 Oct 10. doi:10.1186/s12883-020-01938-3
110. Hidalgo-Agudo RD, Lucena-Anton D, Luque-Moreno C, Heredia-Rizo AM, Moral-Munoz JA. Additional Physical Interventions to Conventional Physical Therapy in Parkinson's Disease: A Systematic Review and Meta-Analysis of Randomized Clinical Trials. *J Clin Med*. 2020;9(4):1038. Published 2020 Apr 7. doi:10.3390/jcm9041038
111. Garcia-Agundez A, Folkerts AK, Konrad R, et al. Recent advances in rehabilitation for Parkinson's Disease with Exergames: A Systematic Review. *J Neuroeng Rehabil*. 2019;16(1):17. Published 2019 Jan 29. doi:10.1186/s12984-019-0492-1
112. Li R, Zhang Y, Jiang Y, Wang M, Ang WHD, Lau Y. Rehabilitation training based on virtual reality for patients with Parkinson's disease in improving balance, quality of life, activities of daily living, and depressive symptoms: A systematic review and meta-regression analysis. *Clin Rehabil*. 2021;35(8):1089-1102. doi:10.1177/0269215521995179
113. Perrochon A, Borel B, Istrate D, Compagnat M, Daviet JC. Exercise-based games interventions at home in individuals with a neurological disease: A systematic review and meta-analysis. *Ann Phys Rehabil Med*. 2019;62(5):366-378. doi:10.1016/j.rehab.2019.04.004
114. Dockx K, Bekkers EM, Van den Bergh V, et al. Virtual reality for rehabilitation in Parkinson's disease. *Cochrane Database Syst Rev*. 2016;12(12):CD010760. Published 2016 Dec 21. doi:10.1002/14651858.CD010760.pub2
115. Combs SA, Diehl MD, Chrzastowski C, et al. Community-based group exercise for persons with Parkinson disease: a randomized controlled trial. *NeuroRehabilitation*. 2013;32(1):117-124. doi:10.3233/NRE-130828
116. *Rock Steady Boxing*. Available from: https://www.rocksteadyboxing.org Accessed July 10, 2022.
117. Morris ME, Ellis TD, Jazayeri D, et al. Boxing for Parkinson's Disease: Has Implementation Accelerated Beyond Current Evidence?. *Front Neurol*. 2019;10:1222. Published 2019 Dec 4. doi:10.3389/fneur.2019.01222
118. American College of Sports Medicine's Physical Activity Guidelines for Adults. ACSM. 2018. Available from: https://www.exerciseismedicine.org/assets/page_documents/EIM%20Prescription%202018%20e-form.pdf
119. U.S. Department of Health and Human Services. *Physical Activity Guidelines for Americans, 2nd edition*. Washington, DC: U.S. Department of Health and Human Services; 2018. Available from: https://health.gov/sites/default/files/2019-09/Physical_Activity_Guidelines_2nd_edition.pdf

120. Osborne JA, Botkin R, Colon-Semenza C, et al. Physical Therapist Management of Parkinson Disease: A Clinical Practice Guideline From the American Physical Therapy Association. *Phys Ther*. 2022;102(4):pzab302. doi:10.1093/ptj/pzab302
121. Martignon C, Pedrinolla A, Ruzzante F, et al. Guidelines on exercise testing and prescription for patients at different stages of Parkinson's disease. *Aging Clin Exp Res*. 2021;33(2):221-246. doi:10.1007/s40520-020-01612-1
122. Uhrbrand A, Stenager E, Pedersen MS, Dalgas U. Parkinson's disease and intensive exercise therapy--a systematic review and meta-analysis of randomized controlled trials. *J Neurol Sci*. 2015;353(1-2):9-19. doi:10.1016/j.jns.2015.04.004
123. American College of Sports Medicine’s Physical Activity Guidelines for Adults. ACSM. 2018. Available from: https://www.exerciseismedicine.org/assets/page_documents/EIM%20Prescription%202018%20e-form.pdf
124. Moore, G., Durstine, J. and Painter, P., 2016. *ACSM’s exercise management for persons with chronic diseases and disabilities: 4th. Ed.*. 4th ed. Champaign, IL: Human Kinetics, p.21.
125. Schutzer KA, Graves BS. Barriers and motivations to exercise in older adults. *Prev Med*. 2004;39(5):1056-1061. Doi:10.1016/j.ypmed.2004.04.003
126. Ellis T, Boudreau JK, DeAngelis TR, et al. Barriers to exercise in people with Parkinson disease. *Phys Ther*. 2013;93(5):628-636. doi:10.2522/ptj.20120279
127. Domingos J, Radder D, Riggare S, et al. Implementation of a Community-Based Exercise Program for Parkinson Patients: Using Boxing as an Example. *J Parkinsons Dis*. 2019;9(3):615-623. Doi:10.3233/JPD-191616
128. Schootemeijer S, van der Kolk NM, Ellis T, et al. Barriers and Motivators to Engage in Exercise for Persons with Parkinson’s Disease. *J Parkinsons Dis*. 2020;10(4):1293-1299. Doi:10.3233/JPD-202247
129. Collado-Mateo D, Lavín-Pérez AM, Peñacoba C, et al. Key Factors Associated with Adherence to Physical Exercise in Patients with Chronic Diseases and Older Adults: An Umbrella Review. *Int J Environ Res Public Health*. 2021;18(4):2023. Published 2021 Feb 19. Doi:10.3390/ijerph18042023
130. Jenkins EM, Nairn LN, Skelly LE, Little JP, Gibala MJ. Do stair climbing exercise “snacks” improve cardiorespiratory fitness?. *Appl Physiol Nutr Metab*. 2019;44(6):681-684. Doi:10.1139/apnm-2018-0675
131. Rafiei H, Omidian K, Myette-Côté É, Little JP. Metabolic Effect of Breaking Up Prolonged Sitting with Stair Climbing Exercise Snacks. *Med Sci Sports Exerc*. 2021;53(1):150-158. Doi:10.1249/MSS.0000000000002431
132. Perkin OJ, McGuigan PM, Stokes KA. Exercise Snacking to Improve Muscle Function in Healthy Older Adults: A Pilot Study. *J Aging Res*. 2019;2019:7516939. Published 2019 Oct 3. Doi:10.1155/2019/7516939
133. Haddad M, Stylianides G, Djaoui L, Dellal A, Chamari K. Session-RPE Method for Training Load Monitoring: Validity, Ecological Usefulness, and Influencing Factors. *Front Neurosci*. 2017;11:612. Published 2017 Nov 2. Doi:10.3389/fnins.2017.00612

APPENDIX 1

MEDLINE search terms

("exercise"[MeSH Terms] OR "exercise"[All Fields]) AND ("parkinson disease"[MeSH Terms] OR ("parkinson"[All Fields] AND "disease"[All Fields]) OR "parkinson disease"[All Fields] OR ("parkinson's"[All Fields] AND "disease"[All Fields]) OR "parkinson's disease"[All Fields]) AND ("back pain"[MeSH Terms] OR ("back"[All Fields] AND "pain"[All Fields]) OR "back pain"[All Fields]) AND ("pain"[MeSH Terms] OR "pain"[All Fields]) OR ("pliability"[MeSH Terms] OR "pliability"[All Fields] OR "flexibility"[All Fields]) OR strength[All Fields] OR ("posture"[MeSH Terms] OR "posture"[All Fields]) OR "balance"[All Fields] OR endurance[All Fields]

Conservative Management of Peroneal Nerve Entrapment: A Case Report

Shawn LaCourt, DC[1]

Tara Cheuvront, DC[1]

Corey McCurdy[2]

[1] Palmer College of Chiropractic, Port Orange, FL
[2] Student, Palmer College of Chiropractic, Port Orange, FL

shawn.lacourt1@palmer.edu

Published: December 2022
Journal of the International Academy of Neuromusculoskeletal Medicine
Volume 19, Issue 2

ABSTRACT

Background

The peroneal nerve is the most common compressive nerve entrapment in the lower extremity. Symptoms of peroneal nerve entrapment include foot drop, weakness of the ankle and toe dorsiflexion and eversion, and paresthesia over the lateral leg below the knee and dorsum of the foot. The purpose of this case report is to discuss the treatment and outcome of peroneal nerve entrapment utilizing manipulation, kinesiology tape, and manual therapy.

Methods

A 23-year-old female patient presented for chiropractic care for a chief complaint of numbness and tingling over the anterior aspect of her left foot. After a history and exam, the patient was diagnosed with peroneal nerve entrapment. The patient completed 12 visits over two months using manipulation, manual therapy, and kinesiology tape.

Results

The patient had a complete resolution of symptoms after the seventh visit. The patient reported that after being discharged from care, she could complete all her normal activities of daily living without reproducing the symptoms.

Conclusion

A 23-year-old female patient was successfully treated by manipulation, manual therapy, and kinesiology tape for the diagnosis of peroneal nerve entrapment.

Keywords

Peroneal nerve entrapment, kinesiology tape, manipulation, manual therapy

INTRODUCTION

The peroneal nerve is the most common compressive nerve entrapment in the lower extremity.[1] The primary cause of peroneal nerve entrapment is a traumatic injury such as knee dislocation, fibular fractures, or a direct blow to the area.[1] Nontraumatic risk factors include behavior issues related to sitting for long periods with crossed legs and repetitive stretches from squatting.[1] Interestingly, weight loss may also produce acute peroneal nerve entrapment in some patients.[1] Knee arthroplasty is a surgical risk factor for developing peroneal nerve entrapment.[1]

The common symptoms of peroneal nerve entrapment are foot drop, ankle and toe dorsiflexion and foot eversion weakness, along with paresthesia over the lateral leg below the knee and dorsum of the foot.[1-2] Individuals with peroneal nerve entrapment may also experience a distinctive steppage gait.[2] The common peroneal nerve branches off the sciatic nerve proximal to the popliteal fossa.[2] The common peroneal nerve winds around the fibular head, making it vulnerable to compression injuries or excessive stretching injuries against the fibular head.[2] Distal to the fibular head, the common peroneal nerve branches out to the superficial and deep peroneal nerves.[2] The superficial peroneal nerve provides sensation to the dorsum of the foot and the outside portion of the lower extremity.[2] The superficial nerve also provides motor function to the peroneus longus muscle.[2] The deep peroneal nerve provides sensory information to the first digit of the lower extremity as well as the webbing of the foot.[2] The motor function of the tibialis anterior, extensor hallicus longus and extensor hallicus brevis is provided by the deep peroneal nerve.[2]

Conservative treatment for peroneal nerve entrapment is based on removing anything that may be compressing the area, stabilizing unstable joints, and reducing inflammation.[1] Many patients that experience peroneal nerve entrapment will have relief with conservative treatment; however, surgical intervention is often necessary when conservative treatment fails to resolve the symptoms.[1]

Villafane et al[3] discussed a case involving a 24-year-old male patient and the use of spinal manipulation, fibular head manipulation, and neurodynamic mobilization, including soft tissue treatment to the psoas and hamstrings. The treatment outcome was an increase in patient function and a decrease in symptoms. Villafane et al[3] is one of only a few case reports that utilized manipulation as part of the treatment plan. The purpose of the case report is to discuss the treatment and outcome of peroneal nerve entrapment utilizing manipulation, kinesiology tape, and manual therapy.

CASE PRESENTATION

A 23-year-old female patient presented for chiropractic care for a chief complaint of numbness and tingling over the anterior aspect of her left foot. The patient reported that the pain had come on over the previous two weeks and had been a relatively constant sensation since the onset. The patient explained that she felt most of the numbness over the anterior aspect of her left talus. When she would walk, it would occasionally radiate into the medial aspect of her left foot. The patient was instructed to walk down the hallway a few times to establish when the pain radiated the most in her gait. It was established that the radiating numbness and tingling to the medial foot would occur during the "pre-swing" and "toe off" phases of the gait cycle. The patient also noted numbness and tingling when she wore heels, so it was concluded that the radiating numbness and tingling sensation would occur when the patient's left foot was in plantar flexion. It was also noted that when the patient first noticed the numbness and tingling, she felt some slight numbness in her shin, but that has since diminished, and she has not experienced that sensation since the initial onset. The patient rated the severity of the numbness and tingling sensation at rest as 3/10 and when she was walking or in heels as 5/10 on a VAS pain scale. There is no known mechanism of injury, but the patient first acknowledged the pain when she was walking on the treadmill at the gym, and the pain has remained since that day, two weeks before her initial visit for the issue. The patient's prior history reveals corrective scoliosis surgery and spinal fusion surgery five years prior for correcting a 72-degree levoscoliosis in her thoracic spine. The patient has no other significant surgeries, traumas, or complaints in her accompanying history.

Upon examination of the left foot, the range of motion in dorsiflexion, plantar flexion, eversion, inversion, abduction, and adduction were all within normal limits; however, the patient's complaint of numbness and tingling over the anterior and medial aspect of the left foot was elicited with active and passive plantar flexion of the right foot. Tenderness, numbness, and tingling were elicited with direct palpation of the left talus, along with a slight referral to the medial aspect of the foot. Palpation of the lower left leg also revealed trigger points which are hypertonic spots or a somewhat palpable nodule found in taut bands of skeletal muscle, as well as muscle spasms throughout the patient’s anterior tibialis muscle, peroneus longus, and peroneus brevis muscles. The patient's numbness and tingling on the anterior part of the foot were also elicited when the peroneus longus and brevis muscles were palpated. The patient reported feeling it in the profound plantar aspect of her left foot. Anterior and posterior drawer tests of the left ankle were negative, along with the Thompson and Tinel’s Tarsal Tunnel tests. Duchenne's sign was positive on the left foot and negative on the right, indicative of peroneal nerve entrapment on the left. The patient's lumbar spine and hip were also examined and revealed no significant findings, positive orthopedic tests, or diminished motor, sensory, or deep tendon reflex neurological deficits.

Initial chiropractic treatment began with a review of the patient's findings. It explained the different aspects where the peroneal nerve could become entrapped and cause the paresthesia she was experiencing on the anterior and medial aspects of her left foot. Initial patient treatment began with myofascial release and trigger point therapy of the left anterior tibialis muscle, peroneus longus, and peroneus brevis muscles. The myofascial release of the patient's peroneus longus and brevis muscles consisted of first shortening the muscle by plantar-flexing and everting the left foot, then taking a thumb contact on the distal aspect of the muscle belly and passively dorsi-flexing and inverting the left foot with the other hand while the thumb pins the muscle. This process was repeated, working proximally up both muscle bellies until the furthermost proximal end of the muscle belly was reached. The same process was repeated for the left anterior tibialis muscle except the foot was first dorsi-flexed and inverted to shorten the muscle and was plantar-flexed and everted to lengthen the muscle after the thumb contact in the muscle belly was taken. Trigger point therapy consisted of applying constant ischemic pressure with a reinforced thumb contact over the previously palpated trigger points in each of the peroneus longus, brevis, and anterior tibialis muscles until the trigger point was released.

Treatment continued with a high-velocity low amplitude (HVLA) chiropractic adjustment of the patient's left talus when palpation findings indicated the need. For the talus adjustment, in this case, the patient is positioned supine with the right leg on the table and the left leg hanging off the side of the table. The doctor stands at the foot of the table facing the head of the table on the side of the patient's left leg. The segmental contact point is the anterior dome of the talus; the doctor's contact point is the left and right middle fingers overlapped on the anterior dome of the talus, with the left middle finger being the most superior of the two. The thumbs stabilize on the foot's plantar side, and the ring and pinky fingers stabilize on the posterior aspect of the Achilles tendon. The left foot is first tractioned superior to inferior. Then, a short, quick thrust is applied anterior to posterior by the middle fingers on the anterior dome of the talus. Further into the treatment plan, a low force-impulse adjustment was performed on the patient's left fibular head with a chiropractic instrument assisted adjustment. It was added to the patient's care plan going forward. For this adjustment, the patient is lying prone with both legs on the table. The doctor is positioned in a scissored stance facing the head of the table on the patient's left side. The posterior aspect of the patient's left fibular head is contacted with the instrument and a thrust is administered with the instrument in a posterior to anterior line of correction.

Treatment also included decompression strips of kinesiology tape, which were applied to the anterior dome of the left talus and the posterior aspect of the left fibular head. The kinesiology tape was applied in a bandage-like fashion where the backing of the tape is first ripped in the middle to expose the adhesive side of the kinesiology tape. The backing is then slightly peeled back and roughly an inch of the tape is exposed. Once the adhesive side of the tape is exposed, it is stretched to about 25% of the tape's total stretch ability and the center of the one inch of tape that is exposed is applied to the head of the left fibula and to the anterior dome of the left talus individually. The rest of the backing is then removed from the remainder of one side of the tape strand and applied to the skin as an anchor strip, with no stretch involved. This is then repeated for the other side of the tape with the backing still on.

The patient's treatment plan consisted of ten visits over eight weeks. The patient did miss weeks three and four due to traveling and work but returned to a consistent treatment schedule after her travel. The patient no longer experienced numbness and tingling throughout the last three visits. At the conclusion of the patient's care plan, she no longer had any symptoms from her original chief complaint and could walk on a treadmill and wear high heels without the symptoms being elicited at any time.

DISCUSSION

Peroneal nerve palsy is the most common entrapment neuropathy in the lower extremity, and the presentation of foot drop is a frequent initial manifestation of this disorder.[4] This condition can have a marked influence on the patient's activities of daily living. In a referenced case study, a similar treatment approach to ours utilizing chiropractic manipulation of the lower extremity was chosen for a 14-year-old female with an insidious onset of foot drop.[5]

After the history and examination, clinical reasoning suggested that the lesion exclusively involved temporary compression of the peroneal nerve at the fibular head, as evidenced by weak dorsiflexion and spared plantar flexion, inversion, and intact sensation of the lower leg, with no suspicion of higher sources of neuropathy based upon exam.[5] Other etiologies such as synovial cysts, diabetic mononeuropathy, infection, tumor, and aneurysm were discounted due to the patient's lack of associated symptoms, pain, relevant clinical history, and negative examination and imaging findings.[5] There was no indication of other associated pathologies; therefore, clinical rationale led the chiropractor to an association between tibiofibular joint dysfunction secondary to a sports sprain injury and residual post-meniscectomy scar tissue at the knee joint affecting the peroneal nerve at the level of the fibular head.[5] Strictly a mechanical etiology was considered based on lack of foot dorsiflexion, moderate posterior and lateral proximal tibiofibular joint restriction on active and passive range of motion, and essentially negative history, examination, and imaging findings to consider more complex or higher source etiologies.[5]

The chiropractic physician assessed the knee joint and identified a lateral to medial fixation compatible with segmental joint dysfunction (restriction) at the right proximal tibiofibular joint.[5] A manipulative procedure was performed with the metacarpophalangeal joint of the index finger in contact with the fibular head and stabilization on the opposite side of the joint.[5] A lateral to medial HVLA thrust was delivered, addressing the tibiofibular joint restriction.[5] Immediately after, the patient could dorsiflex her right foot approximately 1 to 1½ inches with noticeably increased strength.[5] It is possible that the joint addressed had residual scar tissue irritating the nerve, which was released after the manipulation. The patient was released from care following a reexamination two weeks after her initial presentation, where she presented asymptomatic with no significant findings on examination.[5] She was advised to avoid leg-crossing habits, continue self-directed lower extremity rehabilitative exercises for re-conditioning and muscle strengthening, and consider follow-up with physical therapy.[5]

Tibiofibular joint manipulation has been shown to improve ankle dorsiflexion, decrease joint pain, and improve balance.[5] HVLA manipulation of joint dysfunctions may break down adhesions from previous micro-traumatic injuries and scar tissue, which is known to be one cause of temporary peroneal nerve palsy.[5] Thus, manual manipulation may release these fibrous entrapments and restore functionality.[5] This study is unique regarding foot drop and utilization of CMT. There are few to no published similar cases in the current literature. This emphasizes the importance of differential diagnosis and management of peroneal nerve palsy.[5]

Another research study was found that demonstrates the use of instrument-assisted manipulation to improve symptoms of lower leg neuropathy.[6] Based on the study's results, a course of chiropractic care using Torque Release Technique (TRT) and Activator Methods Chiropractic Technique (AMCT) to correct segmental dysfunction was associated with the resolution of chronic lower limb neuropathy and improvements in physical functioning and quality of life.[6]

Additional research was found utilizing Kinesio taping (KT) and proprioceptive neuromuscular facilitation (PNF) interventions for patients with reduced ankle dorsiflexion range of motion due to foot drop and stroke.[7] The study concluded that PNF-KT applied to the affected side improved ankle dorsiflexion range of motion and gait parameters in hemiplegic stroke patients better than no taping.[7] Thus, lower-leg PNF-KT may be a helpful intervention in a rehabilitation program to improve ankle dorsiflexion range of motion and gait parameters in chronic stroke patients with foot drop in clinical settings.[7]

Similarly, another study aimed to determine the effect of a proprioceptive neuromuscular facilitation (PNF) pattern Kinesio taping (KT) application on the ankle dorsiflexion range of motion (DF-ROM) and balance ability in patients with chronic stroke.[8] This study demonstrates the application of PNF-KT may be a feasible therapeutic method for improving ankle movement and balance in patients with chronic stroke.[8] Additional research is recommended to identify the long-term effects of PNF-KT.[8]

Lastly, a referenced case report describes a 40-year-old male who presented with complaints of pain in the left lower lateral one-third of the leg.[9] Physical diagnosis for entrapment of the superficial peroneal nerve at the site of the peroneal tunnel was entertained based on clinical examination and three positive provocation tests. Conventionally, treatment for this type of entrapment has been surgical decompression by splitting the crural fascia, with successful outcomes. This study demonstrates physical therapy management of peroneal nerve entrapment with pain modalities, soft tissue mobilization, and neural mobilization. Reduction of pain was noted in this patient with complete pain resolution maintained at a six-month follow-up.[9]

The prior referenced studies demonstrate the clinical rationale and effectiveness of the treatments utilized in this case. The patient was managed successfully by conservative care consisting of manual and instrument-assisted manipulations, manual therapy, and kinesiology tape. However, one limitation of this study is the patient did not receive a diagnostic ultrasound to confirm the diagnosis of peroneal nerve entrapment due to the fact that the patient’s symptoms improved with conservative management prior to the need for further advanced testing. The utilization of diagnostic ultrasound has become increasingly popular in diagnosing nerve entrapment. Jung et al. reported that the diagnostic ultrasound was cost-effective and reliable for the diagnosis of nerve entrapment.[10] Chang & Kim demonstrated that the diagnostic ultrasound is reliable for large peripheral nerves of the extremities; however, there are inconsistencies with the smaller peripheral nerves.[11]

CONCLUSION

This case report describes the resolution of peroneal nerve entrapment by utilizing manipulation, manual therapy, and kinesiology tape. It describes conservative treatment without adverse reactions to treat the condition, and suggests the application of kinesiology tape may be beneficial to patients with peroneal nerve entrapment.

COMPETING INTERESTS

The authors declare that they have no competing interests.

REFERENCES

1. Fortier LM, Markel M, Thomas BG, Sherman WF, Thomas BH, Kaye AD. An update on peroneal nerve entrapment and neuropathy. *Orthopedic Reviews*. 2021;13(2). doi:10.52965/001c.24937

2. Bowley MP, Doughty CT. Entrapment neuropathies of the lower extremity. *Medical Clinics of North America*. 2019;103(2):371-382. doi:10.1016/j.mcna.2018.10.013

3. Villafañe JH, Pillastrini P, Borboni A. Manual therapy and neurodynamic mobilization in a patient with peroneal nerve paralysis: A case report. *Journal of Chiropractic Medicine*. 2013;12(3):176-181. doi:10.1016/j.jcm.2013.10.007

4. Masakado Y, Kawakami M, Suzuki K, et al. Clinical neurophysiology in the diagnosis of peroneal nerve palsy. *Keio J. Med*. 57 (2008), pp. 84-85.

5. Francio T. Chiropractic care for Foot Drop due to peroneal nerve neuropathy. *Journal of Bodywork and Movement Therapies*. 2014;18(2):200-203. doi:10.1016/j.jbmt.2013.08.004

6. Russell D, Doyle M. Resolution of lower limb neuropathy and improved physical functioning in an 18-year-old male cricketer receiving chiropractic care: a case report. *Chiropractic Journal of Australia*. 2018;*46*(2):123–133. https://www.cjaonline.com.au/index.php/cja/article/view/205

7. Park D, Bae Y. Proprioceptive neuromuscular facilitation Kinesio taping improves range of motion of ankle dorsiflexion and balance ability in chronic stroke patients. *Healthcare*. 2021;9(11):1426. doi:10.3390/healthcare9111426

8. Bae Y, Park D. Immediate effect of lower-leg Kinesio taping on ankle dorsiflexion and gait parameters in chronic stroke with Foot Drop. *Journal of Stroke and Cerebrovascular Diseases*. 2022;31(5):106425. doi:10.1016/j.jstrokecerebrovasdis.2022.106425

9. Anandkumar S. Physical therapy management of entrapment of the superficial peroneal nerve in the lower leg: a case report. *Physiother Theory Pract*. 2012 Oct;28(7):552-61. doi: 10.3109/09593985.2011.653709. Epub 2012 Feb 2. PMID: 22299636

10. Jung J-H, Kim K-H, Choi S-K, Shim J-H. Usefulness of ultrasound for detecting suspected peripheral nerve lesions in diagnosis of peripheral neuropathy: Case report and brief review of the literature. *Journal of Korean Neurosurgical Society*. 2013;53(2):132. doi:10.3340/jkns.2013.53.2.132

11. Chang K-V, Kim SB. Editorial: Use of ultrasound in diagnosis and treatment of peripheral nerve entrapment syndrome. *Frontiers in Neurology*. 2020;10. doi:10.3389/fneur.2019.01348