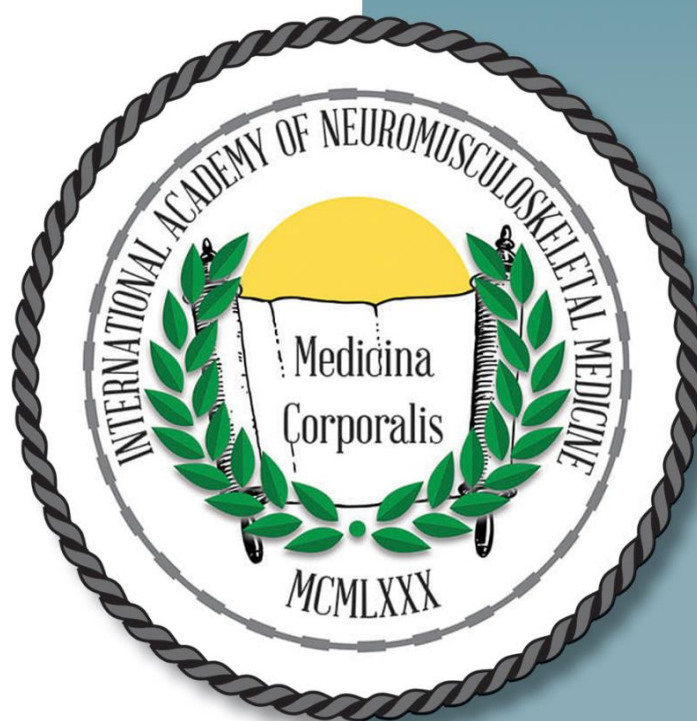


JIANM

**Journal of the International
Academy of
Neuromusculoskeletal
Medicine**



Volume 19

Issue 1

June 2022



IANM

Journal of the International Academy of Neuromusculoskeletal Medicine

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

Editorial Board

Editor-in-Chief

Marc Lucente, DC, MA, DIANM

Associate Editors

Jeffrey P. Krabbe DC, MPH, MS, DACBN, FACN, CISSN, CSCS
Tracey Littrell, DC, DACBR, DIANM

Editorial Advisory Board

James Brandt, DC, MS, DIANM
Ronald Evans, DC, DIANM
Bruce Gundersen, DC, DIANM

Editorial Review Board

Rishi T. Bodalia, DC, MS, DACBR, RMSK
Robert Cooperstein, MA, DC
Donald S. Corenman, MD, DC, DIANM
J. Donald Dishman, DC, MS, DIBCN, FIACN, FIBE
Jo Eash, DC
Ralph Kruse, DC, DIANM
Heather Meeks, DC

Sean Norkus, DC, MS, DIBCN, DIBE
Casey S. Okamoto, DC
Christopher Roecker, DC, MS, DIANM, DACSP
Alec Schielke, DC
Brandon Steele, DC, DIANM
John Stites, DC, DACBR, DIANM
Cliff Tao, DC, DACBR

Articles, abstracts, opinions, and comments appearing in this journal are the work of submitting authors, have been reviewed by members of the editorial board and do not reflect the positions, opinions, endorsements, or consensus of the Academy.

Journal of the International Academy of Neuromusculoskeletal Medicine

June 2022 – Volume 19, Issue 1

Editor's Desk

- ❖ Marc Lucente, DC, MA, DIANM

Original Articles

- ❖ Shaw T, Sergeant A, Lorentz D. Resistance Training for Fall Prevention: A Narrative Review of the Literature. *JIANM*. 2022;19(1):3-9.
- ❖ Cheuvront T, Sergeant A. Lower Extremity Multiple Thromboses Presenting as Sciatic Leg Pain: A Case Report. *JIANM*. 2022;19(1):10-19.
- ❖ McKnight R. Ozone Injection Treatment for Unremitting Head and Neck Pain Post Motor Vehicle Accident: A Case Report. *JIANM*. 2022;19(1):20-26.
- ❖ Gundersen B. Regenerative/Injection Management of a Patient with Achilles Tendinopathy: A Case Report. *JIANM*. 2022;19(1):27-34.
- ❖ Hodges J, Beck N. Chiropractic Care and Rehabilitation Combined with Myofascial Release Technique After Double Mastectomy: A Case Report. *JIANM*. 2022;19(1):35-40.

Editor's Desk

Marc Lucente, DC, MA, DIANM
Editor-in-Chief



Welcome to the June 2022 edition of the Journal of the International Academy of Neuromusculoskeletal Medicine. I am honored to serve as the new Editor-in-Chief and excited to share with you the many updates to the journal and its processes.

Over the past several months, we've created new submission guidelines as well as copyright and consent forms, restructured the editorial board, and revamped the journal section of the Academy website including functionality for authors to make submissions directly through the site. You can check it out at ianmmedicine.org/journals/. Also be sure to visit the Academy Facebook page. Every Like and Follow helps expand our exposure and reach.

The journal will publish new editions biannually every June and December. I encourage you to submit your research, case studies, and literature reviews. I promise clear communication and a rapid response time to your submissions. And remember, publication of an article in a peer-reviewed journal is one way to satisfy annual maintenance of credential requirements for board certification.

Lastly, I need to ask for your help in rebuilding the archives. Unfortunately, during the transition to the new Academy website last year, many past issues of the journal were lost. If you personally possess a digital copy of any edition of JIANM or JACO from 2015 to the present, I would truly appreciate if you could send it to me at drlucente@ianmmedicine.org.

Thank you, and I hope you enjoy the issue!

Resistance Training for Fall Prevention: A Narrative Review of the Literature

Trevor Shaw, DC, DACRB, CSCS¹

Adam Sergeant, DC, DACBSP²

Donald Lorentz, DC³

¹ Associate Professor, Palmer College of Chiropractic, FL

² Professor, Palmer College of Chiropractic, FL

³ Faculty, Palmer College of Chiropractic, FL

trevor.shaw1@palmer.edu

Published: June 2022

Journal of the International Academy of Neuromusculoskeletal Medicine

Volume 19, Issue 1

This is an Open Access article which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. The article copyright belongs to the author and the International Academy of Neuromusculoskeletal Medicine and is available at: <https://ianmmmedicine.org/> © 2022

ABSTRACT

Background: Falls are the leading cause of fatal and nonfatal injuries for adults 65 years of age and older. Decreased lower extremity strength, lack of proprioceptive input (coordination), and instability are the main causes of falls in older adults.

Methods: Peer-reviewed articles were accessed from PubMed using search terms Fall Prevention Exercise AND Resistance Training. Articles featuring interventions other than resistance training were excluded.

Discussion: Identifying physical performance deficits is the first step in constructing a quality program to reduce the rate of falls in older adults. Lower body progressive resistance training has demonstrated an ability to reduce fall risk and should be a central component of any fall prevention exercise intervention.

Conclusion: This literature review demonstrates the current effectiveness of strength training for fall prevention. The American College of Sports Medicine guidelines are an effective starting point for preventing falls due to poor leg strength and physical capability.

INTRODUCTION

According to the Centers for Disease Control (CDC), falls are the leading cause of fatal and nonfatal injuries for adults 65 years of age and older every year.¹ Every fourteen seconds, an older adult is seen in an emergency department for a fall-related injury.² In 2017, 2.8 million older adults were treated in emergency departments for injuries from falls, and more than 800,000 of these patients were hospitalized as a result of their injuries. The price of fall-related injuries totaled nearly 31 billion dollars in medical costs and lost wages.¹ The most common forms of injury in older adults are fractures, sprains, contusions, and head trauma.³ Additionally, falls in older adults are the third leading cause of chronic disability, making fall prevention an important public health concern.⁴ While not entirely avoidable, fall risk can be vastly decreased with proper education and training.

Many factors contribute to the likelihood of sustaining a fall-related injury. Decreased lower extremity strength, lack of proprioceptive input (coordination), and instability are the main causes of falls in older adults.¹

According to the CDC, leg strength is the most important factor related to falls.¹ Leg strength often decreases significantly with age, but it does not have to. The current American College of Sports Medicine (ACSM) guideline suggests any person over the age of 65 should be able to leg press between 1.04 (females) and 1.49 (males) percent of their body weight to meet the "average" criteria. These numbers rise to 1.32 and 1.73 percent to reach the "well above average" standing.⁵ Increasing leg strength is the foundation of fall prevention; it builds both bone and muscle density while improving all aspects of balance and stability.¹

Proprioception is the body's ability to sense where it is in space; to sense movement, action, and location.⁶ Proprioceptors in and around the joint send information to the brain about the joint's position. Dysfunctional ankle and foot mechanics can result in altered proprioception which can lead to balance and stability deficits.

Stability of the lower extremity is achieved through strength and proprioception resulting in effective and efficient movement. If the legs are strong and the joints move well, the final step is targeting specific areas of weakness for rehabilitation. Gait training and specific muscle activation exercises like short foot exercises have been shown to decrease the risk of falls and improve balance and coordination and should be considered when designing a fall prevention program.⁷

METHODS

Peer-reviewed articles were accessed from PubMed from the years 2015 through 2020 using the search terms “Fall Prevention Exercise AND Resistance Training”. A total of 61 articles were returned with these search terms. Following our initial results, exclusion criteria eliminated 49 articles that featured interventions other than resistance training, and allowed for a review of 12 articles.

DISCUSSION

As the number of age-related falls increases, so does the need for alternative approaches for fall prevention therapy. It has been predicted that nearly one-third of the elderly population will sustain a fall in their lifetime, with many resulting in injury or death.⁸ Additionally, the likelihood of a second fall doubles after the first.¹ However, the most recent literature from the CDC suggests that many of these falls may be avoidable with preventative measures such as balance and leg strength training.¹ Attributes such as strength, power, endurance, or functional mobility have all been proposed as factors when considering fall prevention.⁹

Fall-related injuries can often be traced back to physical performance deficits.¹⁰ Identifying such deficits is the first step in constructing a quality program to reduce the rate of falls in older adults. Balance (both static and dynamic) and functional mobility, gait speed, and leg strength have consistently demonstrated themselves as factors in elderly falls.¹¹ However, leg strength continuously appears as the apex of priorities.¹ One study notes that muscular strength decreased by as much as 5% per year with advancing age, increasing the importance of developing and maintaining essential strength.⁵

In addition to strength loss, it is also common to experience decreased rate of force development (RFD), the efficiency of movement and gate due to age-related loss of Type II skeletal muscle fiber.¹² Maximal strength training at 85% or more of an individual's 1 rep max has been shown to directly increase these attributes as it increases both the size and quantity of Type II muscle fibers.^{12, 13}

Adults who used aerobic activity as their sole means of exercise demonstrated decreased isometric muscle strength with an even higher rate of concentric strength loss.⁵ This information provides us an avenue for developing an intelligent, evidence-based program with which the elderly population can thrive.

These factors can be used as a checklist or starting point from which to develop a quality fall-prevention program. However, where does one begin when starting a fall prevention program? While the literature shows that progressive exercise is a critical element of program design, the non-exercise professional may still find it challenging to navigate these patients since many falls can be considered multifactorial.¹ Another challenge often posed with progressive training is the lack of ease with which it can be applied to older frail patients.¹⁴

Lower body progressive resistance training (PRT) has demonstrated its ability to reduce fall risk.¹⁵ This approach should be considered a central component of any fall prevention exercise intervention.¹⁴

Reduced activity and fear of falling have also been shown to lead to deconditioning, muscle atrophy, and weakness.¹⁶ Such fear-avoidance behaviors can result in a vicious cycle of movement avoidance due to fear of falling which can be prevented via proper patient education. Fortunately, many of the current guidelines support the idea that progressive resistance training, even when performed in a minimalist setting, can provide lasting results.¹

Progressive exercise training programs do not need to be complex. These programs can be used as a stand-alone exercise in settings such as in-home or community dwellings and have considerable benefit in fall prevention.¹⁵ In fact, individuals with a greater number of strength training sessions per week had 75% reduced odds of falling as opposed to those with fewer sessions, and progressive resistance exercise three days a week for ten weeks significantly improved the results of all muscle strength tests.¹⁵

Balance training can also be considered a significant factor in fall prevention; however, the exact mechanism of balance training resulting in fewer falls remains unclear. Firstly, balance capability can be considered multifactorial. Many elderly individuals suffer cognitive decline during the aging process, which has been acknowledged as a component in loss of balance.¹⁷ Secondly, it becomes difficult to delineate the exact role of balance training, as quality balance training also results in improved leg strength and motor control.¹¹ This does not diminish its importance, though, because the most common reported reason for falls was the loss of balance.¹⁵ Many falls in older people result from the inability to generate lower limb power to produce rapid, explosive movement to step quickly when the balance is lost. The risk of falling is also increased when attempting to “dual-task,” talking while walking, negotiating traffic or obstacles.¹⁸ A study demonstrated that balance, when coupled with free weight-based training, could effectively improve balance, strength, and power at lower loads than machine-based training.¹⁹

Intelligent balance training depends dramatically on the efficient muscle functioning of the foot. Specifically, the ability to functionally integrate the dynamics of the medial longitudinal arch and supportive intrinsic foot muscles is essential as they have demonstrated crucial roles in many lower extremity injuries. Recently the literature has shown the short foot exercises to be more effective than traditional toe-curling exercises for targeting the intrinsic foot muscles.⁷ This allows the clinician to strengthen the foot while supporting the medial arch actively.

Fall Prevention Exercise Program

Using the information synthesized from this review, the authors propose a 4-phase fall prevention program. This program, presented in **Table 1**, begins with unloaded prone, supine, and side-lying exercises targeted at the feet, leg, and hip muscles.⁴ The exercises then progress through varying neural developmental positions such as quadruped, kneeling, and standing over the course of four phases.

The reader will notice the timeline of each phase is unspecified. As a beginning intervention, many individuals will likely progress at different rates. The structure of this program will allow everyone to progress at their own rate.

Finally, the number of sets and reps are a suggestion. Some individuals will see better results in progressing the exercise volume before trying new positions, while others will benefit from new exercises more frequently.

Phase 1	Sets / Reps	Phase 2	Sets / Reps
Short Foot	1 set 10 Sec Holds	Standing Short Foot	2 sets 10 Sec Holds
Sit to Stands	1 set 8 -15 reps	BW squats	2 sets 8 -15 reps
Leg curls	1 set 8-15 reps	Glute Bridge w/ ABductor stim	2 sets 8-15 reps
Hip Extensions	1 set 8 -15 reps	Glute Bridge w/ ADductor stim	2 sets 8 -15 reps
Clam Shells	1 set 8-15 reps	Single Leg Stance / March	2 sets 8-15 reps
Adductor Ball Squeezes	1 set 20 second holds	Farmers Carry	2 sets 20 second holds
Phase 3	Sets / Reps	Phase 4	Sets / Reps
Cork Skrew to Agility step	3 sets 10 Sec Holds	Speed Ladder	4 sets 10 Sec Holds
Goblet Squat	3 sets 8 -15 reps	Reverse Sled Drags	4 sets 8 -15 reps
Deadlift	3 sets 8-15 reps	SLDL	4 sets 8-15 reps
Unilateral Carries	3 sets 8 -15 reps	Sled Push	4 sets 8 -15 reps
Bird Dog	3 sets 8-15 reps	Unilateral Carries	4 sets 8-15 reps
Tall Kneeling Heartbeats /	3 sets 20 second holds	Half Kneeling Heartbeats / Halos	4 sets 20 second holds

Table 1

CONCLUSION

Falls are a significant healthcare issue in all populations because of the associated risk of injury. With proper training, falls can be reduced by taking a multimodal approach. Rehabilitation professionals including physical therapists, chiropractors, and physiatrists can assist with evaluation and management. This literature review demonstrates the current effectiveness of strength training for fall prevention. Numerous papers suggest the benefit of multimodal exercise. The ACSM guidelines and the exercise program described in this paper are an effective starting point for preventing falls due to poor leg strength and physical capability.

COMPETING INTERESTS

The authors declare that they have no competing interests.

REFERENCES

1. Facts About Falls. Centers for Disease Control and Prevention. Accessed 09/06/2021, 2021. <https://www.cdc.gov/falls/facts.html>
2. Falls Prevention. Administration for Community Living. Accessed 6/7/2022, 2021.
3. Beedham W, Peck G, Richardson SE, Tsang K, Fertleman M, Shipway DJ. Head injury in the elderly—an overview for the physician. *Clinical Medicine*. 2019;19(2):177.
4. Liu-Ambrose T, Davis JC, Best JR, et al. Effect of a home-based exercise program on subsequent falls among community-dwelling high-risk older adults after a fall: a randomized clinical trial. *Jama*. 2019;321(21):2092-2100.
5. Marcell TJ, Hawkins SA, Wiswell RA. Leg strength declines with advancing age despite habitual endurance exercise in active older adults. *The Journal of Strength & Conditioning Research*. 2014;28(2):504-513.
6. Squire LR, Dronkers N, Baldo J. Encyclopedia of neuroscience. vol 2. Elsevier Amsterdam, The Netherlands; 2009.
7. Lynn SK, Padilla RA, Tsang KK. Differences in static-and dynamic-balance task performance after 4 weeks of intrinsic-foot-muscle training: the short-foot exercise versus the towel-curl exercise. *Journal of sport rehabilitation*. 2012;21(4):327-333.
8. Berry SD, Miller RR. Falls: epidemiology, pathophysiology, and relationship to fracture. *Current osteoporosis reports*. 2008;6(4):149-154.
9. Sciamanna C, Ballentine NH, Bopp M, et al. Working to Increase Stability through Exercise (WISE): Study protocol for a pragmatic randomized controlled trial of a coached exercise program to reduce serious fall-related injuries. *Contemporary clinical trials*. 2018;74:1-10.
10. Papa EV, Dong X, Hassan M. Resistance training for activity limitations in older adults with skeletal muscle function deficits: a systematic review. *Clinical interventions in aging*. 2017;12:955.
11. Motalebi SA, Cheong LS, Iranagh JA, Mohammadi F. Effect of low-cost resistance training on lower-limb strength and balance in institutionalized seniors. *Experimental aging research*. 2018;44(1):48-61.

12. Wang E, Nyberg SK, Hoff J, et al. Impact of maximal strength training on work efficiency and muscle fiber type in the elderly: Implications for physical function and fall prevention. *Experimental gerontology*. 2017;91:64-71.
13. Hewitt J, Goodall S, Clemson L, Henwood T, Refshauge K. Progressive resistance and balance training for falls prevention in long-term residential aged care: a cluster randomized trial of the sunbeam program. *Journal of the American Medical Directors Association*. 2018;19(4):361-369.
14. Benichou O, Lord SR. Rationale for strengthening muscle to prevent falls and fractures: a review of the evidence. *Calcified tissue international*. 2016;98(6):531-545.
15. Alvarez KJ, Kirchner S, Chu S, Smith S, Winnick-Baskin W, Mielenz TJ. Falls reduction and exercise training in an assisted living population. *Journal of aging research*. 2015;2015
16. Choi K, Jeon GS, Cho SI. Prospective study on the impact of fear of falling on functional decline among community dwelling elderly women. *International journal of environmental research and public health*. 2017;14(5):469.
17. Thomas E, Battaglia G, Patti A, et al. Physical activity programs for balance and fall prevention in elderly: A systematic review. *Medicine*. 2019;98(27)
18. Daly RM, Duckham RL, Tait JL, et al. Effectiveness of dual-task functional power training for preventing falls in older people: study protocol for a cluster randomised controlled trial. *Trials*. 2015;16(1):1-15.
19. Eckardt N. Lower-extremity resistance training on unstable surfaces improves proxies of muscle strength, power and balance in healthy older adults: a randomised control trial. *BMC geriatrics*. 2016;16(1):1-15.

Lower Extremity Multiple Thromboses Presenting as Sciatic Leg Pain: A Case Report

Tara R. Cheuvront, DC¹

Adam Sergent, DC, DACBSP²

¹ Assistant Professor, Palmer College of Chiropractic, FL

² Professor, Palmer College of Chiropractic, FL

tara.cheuvront1@palmer.edu

Published: June 2022

Journal of the International Academy of Neuromusculoskeletal Medicine

Volume 19, Issue 1

This is an Open Access article which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. The article copyright belongs to the author and the International Academy of Neuromusculoskeletal Medicine and is available at: <https://ianmedicine.org/> © 2022

ABSTRACT

Objective: The purpose of this report is to describe the management of a patient with lower back and right-sided lower extremity pain originating from multiple deep vein thromboses.

Clinical Features: A 24-year-old female collegiate runner with a history of right gluteal and thigh pain lasting for 2 weeks sought chiropractic care for her ongoing low back pain and right sided lower extremity pain. A physical exam identified a concerning “red flag” for serious pathology contributing to this young woman’s acute lower back pain and associated right lower extremity pain.

Intervention and Outcome: This patient was evaluated, and the clinical exam raised suspicion of a possible deep vein thrombosis as a source of her pain. An immediate referral was placed for further diagnostic work-up at a nearby emergency room where 3 thromboses were identified in the patient’s right lower extremity. The patient was treated with intravenous anticoagulant therapy for 3 days in the hospital setting, then underwent 6 months of oral anti-coagulant medication, along with activity modifications. The patient was diagnosed with Factor V Leiden Thrombophilia, after being referred to a hematologist for further evaluation.

Conclusion: We recommend clinicians stay mindful of all factors contributing to low back pain combined with lower extremity radicular symptoms. In this case, consideration of additional pathologies resulted in the identification of multiple deep vein thromboses as a source of right lower extremity pain imitating sciatic symptoms.

INTRODUCTION

Blood clots originating within the vascular drainage systems of the lower extremities, including deep vein thrombosis (DVT) and pulmonary emboli (PE), are estimated to effect 1 in every 1,000 individuals and contribute to an estimated 60,000-100,000 deaths per year in the United States.¹ Approximately, 1/3 of all diagnosed venous thromboses will embolize to the lungs as a pulmonary embolism, which may lead to respiratory symptoms or even death.² In young and otherwise healthy individuals between the ages of 25-35, the prevalence of deep vein thrombosis (DVT) is only 3 cases per 10,000 people.³ There is sparse evidence on the prevalence in those individuals under the age of 25.

The formation of thrombi in the deep veins occurs most commonly in the large veins of the legs or pelvis. Pulmonary embolism develops when a thrombus dislodges from the walls of a vein, traveling through the heart, and becoming lodged in the pulmonary arterial system as these vessels progressively narrow. There is a 50% chance that patients with untreated proximal deep vein thrombosis (DVT) will develop symptomatic PE within 3 months.⁴ A proximal DVT is one that is in the popliteal, femoral, or iliac veins. For 25% of patients, the initial manifestation of PE is sudden death, via obstruction of the cardio-pulmonary circulation.⁴ Venous thromboembolism (VTE) may be categorized as provoked or unprovoked. This categorization influences the risk of recurrent VTE and duration of anticoagulation therapy. It is important for primary care providers to clearly understand the pathogenesis and causes of thrombosis, and to create evidence-based therapeutic and prophylactic patient care plans that prevent recurrent VTE.⁴ An unprovoked VTE refers to a thrombotic event that is not associated with an environmental risk factor. Examples of nonenvironmental risk factors are hereditary thrombophilia, male sex, and advanced age. A provoked VTE refers to a thrombotic event that has been caused by environmental or acquired risk factors for VTE. Additionally, provoked events may be divided into transient or persistent causes. A transient variable is expected to resolve after the VTE event. Active cancer, congestive heart failure, obesity, and varicose veins are examples of persistent provoked risk factors, while examples of transient provoked risk factors include immobility or prolonged bed rest (> 3 days), estrogen therapy, trauma or surgery, pregnancy, or the presence of a foreign object or implanted medical device.⁴

Clinical signs and symptoms of a VTE include asymmetrical tissue swelling, warmth, redness, or pain in the affected extremity.¹ Pulmonary embolism may present with dyspnea, chest pain, hemoptysis, syncope, tachycardia, or hypotension.⁵ However, these signs and symptoms have been shown to be unreliable at predicting a DVT.⁶

The Wells Criteria is a common scoring system intended to help clinicians estimate the probability that an individual has a DVT. The Wells Criteria involves creating a score based on the presence or absence of 9 items and is shown in **Table 1**.⁷

Table 1: Wells criteria for the prediction of deep vein thrombosis (DVT)

Clinical Features	Points
Active cancer (receiving treatment or treated in past 6 months)	1
Paralysis, or recent cast immobilization in lower extremities	1
Bedridden for ≥ 3 days or having major surgery within the past 12 weeks	1
Localized tenderness along the distribution of deep venous system	1
Swelling of the entire leg	1
Calf swelling of ≥ 3 cm (compared to asymptomatic leg)	1
Pitting edema on affected leg	1
Collateral superficial veins (non-varicose)	1
Previous DVT	1
Alternate diagnosis is at least as likely as DVT	-2
DVT Likely 2 points or more	
DVT Unlikely 1 point or less	

Diagnosis of VTE is best done with compression ultrasonography or computed tomography pulmonary angiography (CTPA). However, only 20% of all suspected cases have a VTE confirmed.⁵ D-Dimer testing may also be considered when evaluating a suspected thrombus, although this laboratory test is less accurate when used in the elderly. D-Dimer is a sensitive

marker for VTE and can exclude those VTE that are non-specific and avoid the need for further imaging.⁵

Management of suspected VTE in private practice necessitates an immediate referral for medical management, preferably to the emergency department. Once diagnosed, treatment consists of 3 phases: initial, long term, and extended.⁵ Anticoagulation medications are the treatment of choice for most patients prescribed by their primary care physician. The choice of medication should be carefully weighed with patient characteristics and preference.

The purpose of this case study is to demonstrate the management of a suspected DVT in a college athlete who was seeking chiropractic care for low back and lower extremity pain.

CASE PRESENTATION

A 22-year-old female college athlete presented with pain in her right gluteal region and right anterior thigh. This pain was well-localized and did not extend past her knee. The athlete stated she participates in track and field at a local university and explained she regularly runs about 5 miles per day. She explained she was experiencing low back pain and “sciatica” in her right gluteal region that began about one week prior while playing basketball. She denied experiencing episodes of similar pain in the past. She chose to take a break and rest from her usual daily runs for a few days, which led to a mild reduction in her pain levels. However, on the morning she presented for care, she attempted to return to running just prior to her appointment. The athlete stated she ran one tenth of a mile before having to stop due to pain in her right gluteal area and right anterior thigh region. She also noted that after her run she felt weakness and was unable to bear weight on her right leg. She noticed mild swelling in her anterior thigh with an associated sensation of “tightness” shortly after her morning run. At her initial consultation, she rated her pain as a 6/10 on the numerical pain rating scale (NRS) while resting and a 9/10 while walking or performing any weight-bearing activity. She reported her symptoms were constantly present, and had gradually worsened since running that morning. The athlete described her pain as “deep” and “radiating,” extending from her right gluteal area to her anterior thigh. Bending, standing, walking, sitting, and general lower extremity movements provoked her symptoms. She could not recall anything that provided relief of her pain. On initial presentation, the athlete presented with her mother and utilized a wheelchair to assist with mobility, since walking or bearing weight on her right leg was too painful.

Upon the initial evaluation, a thorough history and review of systems were performed; no findings from her past health history appeared to contribute to her conditions. She also denied any allergies, surgeries, hospitalizations, significant past traumas, or illnesses. The only prescription medication she reported taking was an oral contraceptive. She also denied having sought any prior evaluation for treatment for her current low back and leg pain complaint. Her vital signs were within normal limits. Lower extremity neurological testing demonstrated normal sensory findings. She exhibited weakness with right hip flexion graded

at 3/5 with corresponding pain. Her other lower extremity motor functions were graded within normal limits at 5/5 which included: left hip flexion, bilateral knee extension, bilateral knee flexion, bilateral ankle dorsiflexion/inversion, bilateral ankle plantarflexion/eversion, and bilateral big toe extension. While she retained muscle strength during testing, she labored to move the right leg and took a bit of time for full muscle contraction. Right patellar reflex was graded 1+ and all other lower extremity reflexes were 2+. Lumbosacral orthopedic tests revealed negative straight leg raise test, negative crossed straight leg raise test, negative slump test, negative Braggard sign, negative Bechterew test, negative sacral thrust, and negative Valsalva test. Visual inspection of the athlete's right quadriceps muscles demonstrated moderate edema, causing difficulty in movement of the right lower extremity. There was mild erythema and mild warmth in the anterior thigh, suggesting a possible inflammatory cause of this patient's complaint. Lumbosacral range of motion testing revealed a mild reduction of lumbar extension with antalgia towards her right side. All other lumbosacral ranges of motion were within normal limits and pain-free. Right hip range of motion demonstrated a mild reduction in extension, due to discomfort and "tightness"; all other ranges of motion were pain-free and within normal limits. During segmental and muscle palpation, pain was unable to be reproduced in the lower back. The athlete did exhibit localized pain and tenderness at the right gluteal muscles, specifically inferior to the piriformis muscle and in the region of the right ischial tuberosity. While she was lying prone, mild swelling was also noted in the right calf region, when compared to the left side. Her dorsal pedal and posterior tibial pulses were within normal limits and equal bilaterally. Due to the athlete's painful presentation and difficulty performing transitional movements, the chiropractic physician did not perform additional exams or orthopedic testing in the regions of complaint.

Considering inconclusive musculoskeletal findings upon examination including lack of true nerve root lesion signs and difficulty reproducing the patient's pain in the lumbosacral region, the clinical decision was made to refer the patient to a local emergency room (ER) for further diagnostic work-up. A recommendation with a medical provider was given for a lower extremity doppler ultrasound test to exclude potentially serious pathology, such as thrombosis or compartment syndrome.

The chiropractic physician followed-up with the athlete and her mother later that evening. Her mother stated she took her daughter to the ER after the initial encounter. Three blood clots were identified in the patient's right lower extremity; locations included one within the posterior medial thigh, one within the anterior medial thigh, and the third in the medial calf region. The athlete was immediately admitted to the hospital and underwent intravenous anti-coagulant therapy. She was closely monitored for the effectiveness of the therapy and was hospitalized for a total of 3 days. Had anti-coagulant therapy been ineffective, surgical breakdown of the clot formations would have been performed based on the attending physician's recommendations. The attending physician reportedly claimed if the patient

would have continued to ambulate that day, the risk for pulmonary embolism would have substantially increased. Clinically relevant reasoning associated to patient diagnosis was correlated with the use of birth control, considering she had no other co-morbidities inherent to this diagnosis.

Combined estrogen-progestin oral contraceptives (COC) are widely used. Various contraceptive medications are available and studies have shown these medications increase the risk of thromboembolism. This risk varies according to the type of progestin in the medication, and the risk decreases with shorter duration of use and less estrogen contents.⁸

Following the patient's hospital stay, she was referred to a hematologist and underwent further testing to investigate additional contributors involved with her tendency to form clots. She was diagnosed with a clotting disorder known as Factor V Leiden thrombophilia. The athlete will continue to undergo treatment with anticoagulant medication until otherwise noted. Swelling of the lower extremity reduced within a few weeks after the initial onset and she gradually returned to short distance light jogging as well as other non-compressive activities and movements including swimming and biking.

Venous thromboembolisms are uncommon among young, and otherwise healthy, athletes presenting to a chiropractic clinic. Characteristic risk factors for thromboembolism include obesity, sedentary lifestyle, and older age (>60); these risk factors do not apply to young adult athletes, which makes this a novel case.⁹ The National Blood Clot Alliance has provided guidelines when evaluating suspected venous thromboembolisms in athletes.¹⁰ These guidelines are presented in **Table 2**.

Table 2: National Blood Clot Alliance Guidelines for Suspected Venous Thromboembolisms in Athletes

Traveling long distances to and from a sports competition (by plane, bus, or car)
Dehydration (during and after a strenuous sporting event)
Significant trauma
Immobilization (brace or cast)
Bone fracture or major surgery
Birth control pills and patch, pregnancy, hormone replacement therapy
Family history of DVT or pulmonary embolism
Presence of an inherited or acquired clotting disorder (Factor V Leiden, prothrombin, 20210 mutation, antiphospholipid antibodies, and others)
Presence of a congenital abnormality of the anatomy of the veins
May-Thurner Syndrome (narrowing of the major left pelvic vein)
Narrowing or absence of the inferior vena cava (the main vein in the abdomen)
Cervical rib causing thoracic outlet obstruction.

The presence of venous thromboembolisms requires medical management and cannot be managed by a chiropractor. Clinicians should consider the risk factors shown in **Table 2** when evaluating athletes who may have suspicious findings detected on physical examination. In cases such as this, the appropriate referral will result in further testing. The athlete, prior to this incident, was unaware that she had Factor V Leiden thrombophilia (FVL), which can lead to other clot-related pathologies. Patients may present with either venous or arterial thrombotic events, both of which are associated with a high morbidity and mortality. Co-morbidities of FVL include recurrent venous thromboembolisms as well as obstetric complications due to the risk of anticoagulation, which may adversely affect the fetus in utero or during birth.¹¹ Cerebral vein thrombosis can occur in FVL individuals, especially in patients using oral contraceptive pills.¹¹ Studies have shown FVL to be associated with an increased risk of Budd-Chiari syndrome and a modest increase in the risk of coronary artery disease. The FVL mutation is associated with an increased risk of stroke especially in women, smokers, and younger individuals.¹¹

Factor V Leiden thrombophilia is a genetic disorder characterized by a deficient anticoagulant response to activated Protein C (APC) and an increased risk for venous thromboembolism. The current evidence suggests that the mutation has a modest effect on recurrence risk after initial treatment of a first venous thromboembolism. Factor V Leiden is also associated with up to a 3-fold increased risk for spontaneous abortion or other obstetric complications, although the probability of a successful pregnancy remains high among women with this condition.¹² The clinical expression of Factor V Leiden is influenced by the number of mutated Factor V Leiden alleles, coexisting genetic and acquired thrombophilia disorders, and circumstantial risk factors. Diagnosis requires the activated Protein C resistance assay (a coagulation screening test) or DNA analysis of the *F5* gene, which encodes the Factor V protein. The duration of oral anticoagulation therapy should be based on an assessment of the risks for venous thromboembolism recurrence and anticoagulant-related bleeding. In the absence of evidence that early diagnosis reduces morbidity or mortality, decisions regarding testing at-risk family members should be made on an individual basis.¹² Lifestyle modifications for those diagnosed with FVL are described in **Table 3**.

Table 3: Agents/Circumstances to Avoid with Factor V Leiden:¹³

Women heterozygous for the Leiden variant and a history of VTE should avoid estrogen-containing contraception and hormone replacement therapy (HRT).
Women homozygous for the Leiden variant with or without prior VTE should avoid estrogen-containing contraception and HRT.
While asymptomatic women heterozygous for the Leiden variant should be counseled to consider alternative forms of contraception and control of menopausal symptoms, those electing use of:
-Oral contraceptives should avoid third generation and other progestins with a higher thrombotic risk.
-Short-term HRT for severe menopausal symptoms should avoid oral formulations.

CONCLUSION

Young athletes have minimal risk of venous thrombosis; however, this population is exposed to many acquired thrombogenic risk factors, including trauma, immobilization, long-distance travel, and the use of oral contraceptives.¹⁴ Since high performance sports are

known to carry an increased risk of thrombogenesis, measures to avoid thrombosis or a thromboembolic event must be initiated in case of known APC resistance. Suitable measures are early anticoagulation during periods of immobilization, leg muscle exercises for long distance flights, and avoidance of hemoconcentration with a sufficient oral fluid intake.¹⁵

Finally, in relation to this presenting case, chiropractic management must include a thorough history and physical exam with critical thinking to include other differential diagnoses not commonly found. Clinical reasoning and consideration of all differential diagnoses is necessary to rule out red flags or serious pathologies that present as lower back and lower extremity pain.

It is essential for clinicians to evaluate and interpret numerous factors that may be contributing to the patient's clinical presentation, to provide the highest quality care possible and make the appropriate referral for further evaluation and management.

REFERENCES

1. Stone J, Hangge P, Albadawi H, et al. Deep vein thrombosis: pathogenesis, diagnosis, and medical management. *Cardiovasc Diagn Ther.* 2017;7(Suppl 3):S276-S284. doi:10.21037/cdt.2017.09.01
2. Kearon C. Natural history of venous thromboembolism. *Circulation.* 2003;107:I22-30. 10.1161/01.CIR.0000078464.82671.78
3. White RH. The epidemiology of venous thromboembolism. *Circulation.* 2003;107(23 suppl 1):I4–I8
4. Phillippe HM. Overview of venous thromboembolism. *Am J Manag Care.* 2017 Dec;23(20 Suppl):S376-S382. PMID: 29297660.
5. Tritschler T, Kraaijpoel N, Le Gal G, Wells PS. Venous Thromboembolism: Advances in Diagnosis and Treatment. *JAMA.* 2018;320(15):1583–1594. doi:10.1001/jama.2018.14346
6. Akman MN, Cetin N, Bayramoglu M, Isiklar I, Kilinc S. Value of the D-dimer test in diagnosing deep vein thrombosis in rehabilitation inpatients. *Arch Phys Med Rehabil.* 2004;85(7):1091-1094. doi:10.1016/j.apmr.2003.10.023
7. Wells PS, Anderson DR, Bormanis J, et al. Value of assessment of pretest probability of deep-vein thrombosis in clinical management. *Lancet.* 1997;350:1795-8. 10.1016/S0140-6736(97)08140-3
8. Sugiura K, Ojima T, Urano T, Kobayashi T. The incidence and prognosis of thromboembolism associated with oral contraceptives: Age-dependent difference in Japanese population. *J Obstet Gynaecol Res.* 2018;44(9):1766-1772.

9. Hummel C, Geisler PR, Reynolds T, Lazenby T. Posttraumatic Deep Vein Thrombosis in Collegiate Athletes: An Exploration Clinical Case Series. *J Athl Train*. 2018;53(5):497-502. doi:10.4085/1062-6050-362-16
10. <https://www.stoptheclot.org/about-clots/athletes-and-blood-clots/>
11. Albagoush SA, Koya S, Chakraborty RK, et al. Factor V Leiden Mutation. [Updated 2021 Aug 15]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK534802/>
12. Kujovich JL. Factor V Leiden thrombophilia. *Genet Med*. 2011 Jan;13(1):1-16. doi: 10.1097/GIM.0b013e3181faa0f2. PMID: 21116184
13. Kujovich JL. Factor V Leiden Thrombophilia. 1999 May 14 [updated 2018 Jan 4]. In: Adam MP, Ardinger HH, Pagon RA, Wallace SE, Bean LJH, Mirzaa G, Amemiya A, editors. GeneReviews® [Internet]. Seattle (WA): University of Washington, Seattle; 1993–2021. PMID: 20301542.
14. Grabowski G, Whiteside WK, Kanwisher M. Venous thrombosis in athletes. *J Am Acad Orthop Surg*. 2013 Feb;21(2):108-17. doi: 10.5435/JAAOS-21-02-108. PMID: 23378374.
15. Hilberg T, Moessmer G, Hartard M, Jeschke D. APC resistance in an elite female athlete. *Med Sci Sports Exerc*. 1998 Feb;30(2):183-4. doi: 10.1097/00005768-199802000-00001. PMID: 9502342.

Ozone Injection Treatment for Unremitting Head and Neck Pain Post Motor Vehicle Accident: A Case Report

Robert S McKnight, DC, DIANM¹

¹ Advanced Spine & Rehabilitation, St. George, UT

drrobmcknight@backpainsstgeorge.com

Published: June 2022

Journal of the International Academy of Neuromusculoskeletal Medicine

Volume 19, Issue 1

This is an Open Access article which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. The article copyright belongs to the author and the International Academy of Neuromusculoskeletal Medicine and is available at: <https://ianmmmedicine.org/> © 2022

ABSTRACT

Background: An 18-year-old Caucasian female presented for treatment 8 months after a rear impact car crash. She complained of cervical spine pain, pain at the base of the skull, and headaches which began at the base of the skull and radiated bilaterally to the back of the skull and above the ears.

Methods: The patient was treated with ozone injections to the suboccipital and upper cervical paraspinal musculature.

Results: The patient reported a 90% decrease in headache frequency and intensity over a 90-day period following the injections.

Conclusion: Properly diagnosed, unremitting head and neck pain can respond favorably to ozone injection therapy when other standard conservative treatment options have failed.

INTRODUCTION

An understanding of the anatomy and physiology of the suboccipital and upper cervical spine musculature (SUCM) (Semispinalis Capitus, Inferior Oblique, Splenius Capitus, Trapezius, Sternocleidomastoid) with relation to the greater and lesser occipital nerves is required to understand the pathogenesis of headaches secondary to compression and

irritation of these nerves in the suboccipital region. The SUCM can lead to compression of the Greater and Lesser Occipital Nerves (GLON) as they course through the juncture of the skull and upper cervical area as seen in **Figure 1**.¹

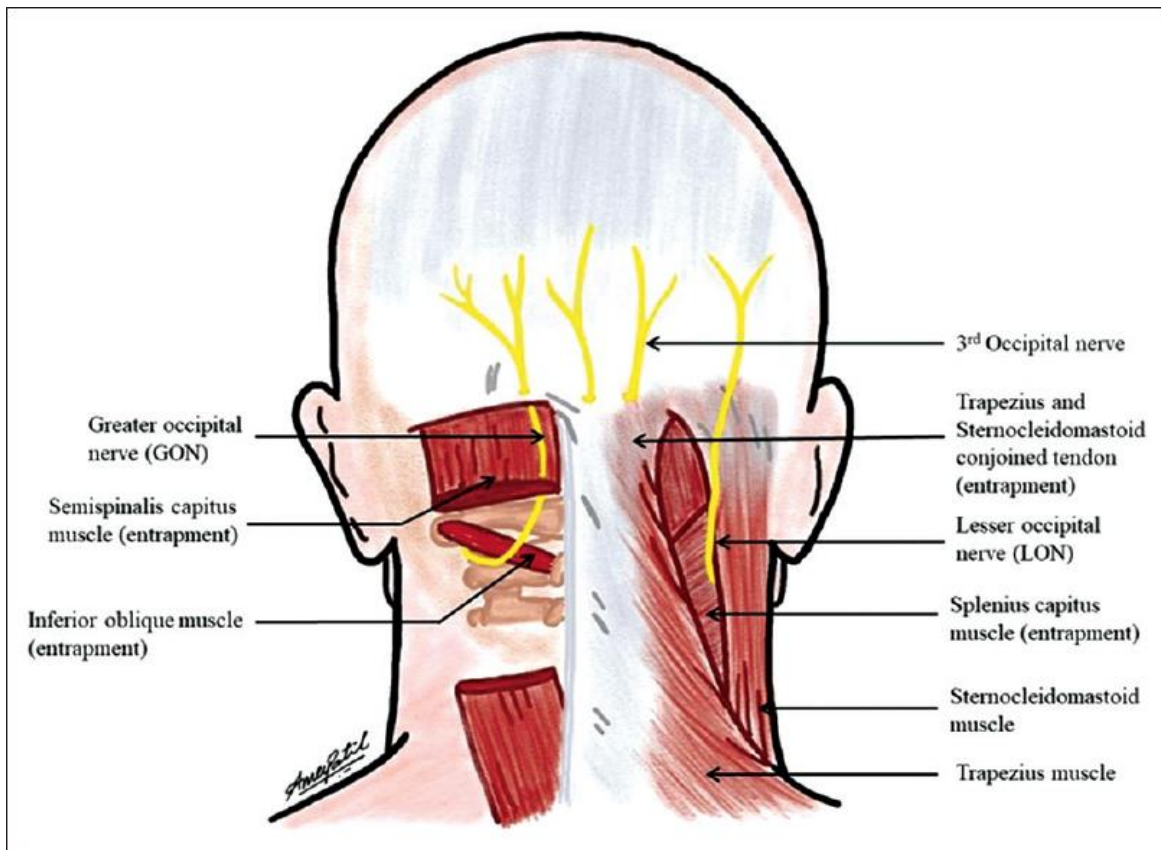


Figure 1

Detailed cadaver studies have shown several areas for possible compression of the occipital nerves along their paths through the posterior cervical muscles and fascia. The GLON may be compressed at one or more of many locations: between the semispinalis and the inferior oblique muscle; at the entry to the semispinalis capitis muscle; at the exit from the semispinalis capitis muscle; at the entrance to the trapezius muscle; the exit from the trapezius; at the fascial insertion of the trapezius at the occipital ridge; and by the occipital artery at the distal location of the nerve above the skull base. The lesser occipital nerve may be compressed by the fascial attachment of the sternocleidomastoid muscle, by branches of the occipital artery, and by fascial bands.²

At this time, there are not adequate imaging techniques available to image the full scope of the compression sites of the lesser and greater occipital nerves; while segments of the occipital nerves may be visualized with ultrasound, as well as the morphologic changes caused by compression within the semispinalis capitis muscle, it is not possible to visualize the nerve in its entirety to allow determination of all the sites of compression.³

The mechanism by which occipital nerve compression may cause headaches may lie in the resulting inflammation that has been observed in both animal and human studies of nerve compression. Experiments in rat models have demonstrated inflammation both local to and remote from the site of peripheral nerve compression.⁴ Similar findings of local inflammation due to nerve compression in human subjects were demonstrated in a 2014 study. The authors reviewed the pathology of resected sections of the lateral femoral cutaneous nerve in seven subjects with meralgia paresthetica who underwent neurectomy for chronic, disabling pain. Histology of the resected nerves showed perineural thickening, demyelination, axonal degeneration, and varying degrees of endoneurial and epineurial inflammation. Collections of inflammatory cells of mild to moderate size were observed in five of the seven subjects, and the degree of inflammation correlated with the degree of axonal injury. Review of the same nerve from autopsy controls did not reveal perineural thickening, axonal loss, or inflammation.⁵

It is important and helpful to distinguish the symptoms of unremitting head and neck pain associated with occipital nerve compression as defined by the International Headache Society.⁶ Occipital Neuralgia (Greater and Lesser) is defined as pain that has two of the following three characteristics: 1) recurring in paroxysmal attacks lasting from seconds to minutes; severe in intensity; shooting, stabbing or sharp in quality, 2) The pain is associated with dysesthesia and/or allodynia, and 3) either tenderness to palpation of the nerve or its branches, or the presence of tender trigger points at the emergence of the occipital nerves. The pain must also be temporarily eased by an anesthetic block of the occipital nerves.

Proper diagnosis of occipital nerve compression as the cause of the patient's unremitting head and neck pain is paramount. The Headache Classification Committee of the International Headache Society (IHS) in the International Classification of Headache Disorders, 3rd edition clarifies some of the differential diagnoses with regards to unremitting head and neck pain and occipital neuralgia. The presence of unremitting head and neck pain in no way excludes the presence of clearly central and brainstem processes being instrumental in the pathophysiology of other headache types, such as low-frequency migraine with aura. One may, in fact, conceptualize headache disorders existing along a continuum of pathophysiology, reflecting the anatomic continuum of nerves from intra- to extracranial. Strong central processes such as low-frequency episodic migraine with aura lie at one end, the "central end," of the spectrum, while unremitting, solely sub-occipital and occipital pain lies at the other, "peripheral end," of the spectrum. Most patients will reside at a point between the ends of the spectrum, and some patients may have two completely different headache types, and thus reside at two places on the spectrum. The clinical characteristics of the separate headaches will differ, as will effective treatments for each headache type. The concept of a spectrum for the pathophysiology of headache disorders is, in fact, illustrated by the range of responses following nerve decompression. Some patients with the purely peripheral factor of nerve compression may have a significant reduction in

pain, similar to the reduction of lumbar radicular pain seen after a lumbar microdiscectomy, while those individuals with additional centrally mediated headaches such as menstrual migraine, chemically induced headaches, dehydration headaches, or migraine with aura will likely continue to experience these headaches. Correct identification of patients who have predominant peripheral factors such as occipital nerve compression as the cause of the unremitting head and neck pain is the most important step in determining if occipital nerve decompression is an appropriate intervention. It is important to note, however, that elimination of the posterior peripheral trigger for unremitting head and neck pain *may* additionally reduce frontal headaches that are consistent with migraine, again, likely reflecting the anatomic spectrum of the neural networks that span both the extracranial and intracranial spaces, and the elimination of activation of extracranial trigeminal nociceptors by the removal of the inflammation-causing compression. It is also of note that indirect results of nerve decompression may result in additional improvement that extends beyond the elimination of unremitting head and neck pain, such as improved sleep from the reduction in occipital allodynia, proper hydration, or dependency on caffeine.⁶

Ozone aids in the inhibition of pain receptors as well as a decrease in myofascial hypertonia. Several authors have described the anti-inflammatory, analgesic, and anti-edema properties of injected medical ozone, and propose that the oxidation of the algogenic receptors would inhibit the pain signal and activate the antinociceptive system.⁷ This is supported in a preclinical study in which the authors induced sciatic damage in mice and confirmed the corticofrontal activation of genes of caspase 1, 8, and 12 (pro-inflammatory, pro-apoptotic, and responsible for allodynia) caused by the injury; this expression was normalized with a single peripheral injection of O₂/O₃ around the damaged area, which also reduced mechanical allodynia.⁸

These properties would favor a muscle relaxant effect, as well as improved mobility of the treated area that can be observed clinically. This effect is very important in muscle recovery with O₂/O₃ injections.⁹ The utility of ozone therapy in the treatment of painful muscle hypertonia highlights the tremendous muscle relaxant effect that is produced.¹⁰

Headaches after a car crash are common and are recognized as one of the bigger pain complaints among patients who have sustained a hyper acceleration/deceleration injury.¹¹ However, ozone injections are not commonly utilized as a successful treatment option. This case indicates that ozone injections should be considered.

CASE PRESENTATION

An 18-year-old Caucasian female presented for care and treatment for her persistent neck and head pain following a rear impact motor vehicle collision 8 months prior.

Following the crash, the patient received multiple conservative treatment options at other facilities. These included chiropractic manipulation, physiotherapy modalities, deep tissue

myofascial release therapy, and cervical spine specific rehabilitation. The patient reported a slight decrease in her pain for a short period of time with each treatment. However, the head and neck pain would return within hours. She also underwent myofascial trigger point injections to the parascapular musculature. She reported only a few hours of pain relief with these injections.

At her initial visit, the patient described the pain as a 3/10 at its best and a 7/10 at its worst. The headaches were described as severe, sharp, shooting, constant, daily, and started at the base of the skull then radiated to the side of her head and over her ears.

Examination of the cervical spine revealed a decrease in active cervical spine range of motion with pain. Significant tenderness in the bilateral cervical paraspinal musculature and bilateral suboccipital musculature was noted. When palpated, the suboccipital myofascial hypertonicity reproduced the patient's sharp and shooting head pain. The location of the head pain was described by the patient as starting at the back of the head/base of the skull with radiating head pain above the ears.

The patient had no history of migraine headaches or migraine with aura. She did describe periodic headaches prior to the crash associated with dehydration, caffeine consumption, and stress. These headaches were infrequent and occurred with physical activity, dietary changes, and stressful situations.

Following the examination and subsequent diagnosis of unremitting head and neck pain associated with occipital nerve compression/irritation, a discussion of mechanism of injury and etiology of the condition were explained to the patient, and intramuscular ozone injections to the upper cervical musculature and suboccipital musculature were recommended.

After obtaining consent for treatment, the patient was taken to a procedure room where she was instructed to lay prone on the procedure table. The upper cervical/suboccipital area was exposed. The most tender musculature in the upper cervical and suboccipital regions were palpated and identified. The patient confirmed the areas to be injected by indicating a reproduction of her head pain upon palpation of the subject musculature. Once the areas for injection were identified and marked (2 in the suboccipital musculature and 2 in the upper cervical spine musculature), a local anesthetic spray was applied to the injection sites. The skin was then prepared with standard sterile preparation techniques. A 30-gauge ½ needle was then utilized to inject 5ml of 19 gamma ozone into each of the target muscles for a total of 20ml of ozone. The injection sites were then covered with a small bandage and the patient underwent 15 minutes of interferential therapy to the region. The patient was given instructions to avoid soaking the affected area in water for 24 hours. She was given a handout with home based cervical spine stretches to perform and was instructed to perform these stretches several times a day for the next week.

Within one week the patient reported a significant decrease in the frequency and intensity of her headaches. She reported experiencing only 2 headaches in the 7-day period. It is important to note that one of those headaches, according to the patient, was due to dehydration as she hiked a lot the day that the headache appeared. Two weeks after the injections, the patient was no longer experiencing headaches.

90 days after the intramuscular upper cervical and suboccipital ozone injections, the patient reported a 90% decrease in the frequency and intensity of her headaches. According to the patient, her post injection headaches now coincided with increased stresses associated with schooling and/or a lack of caffeine consumption. This would indicate a return to her normal degree of headaches in frequency and causative factors.

An updated palpatory examination of the upper cervical and suboccipital musculature was now negative for reproduction of the patient's head pain. This, in conjunction with the patient's description of her changes in symptoms, indicated a successful decompression of the occipital nerves and relief of the unremitting head and neck pain with intramuscular ozone injections.

CONCLUSION

Unremitting head and neck pain is common following an injury to the upper cervical spine and suboccipital musculature. While conservative treatment options such as spinal manipulation, physiotherapy modalities, deep tissue therapy, trigger point injections to the paraspinal musculature, and spine specific rehab are often effective in treating head and neck pain, some patients do not respond to traditional treatment options, as was the case with this patient. After proper diagnosis and treatment with intramuscular ozone injections, decompression of the occipital nerves was achieved thus eliminating the patient's injury related head and neck pain.

With a good history, examination, and proper diagnosis, injury induced unremitting head and neck pain can be treated successfully with intramuscular ozone injections. If a patient does not respond to physical medicine modalities, traditional chiropractic care, conservative treatment options, or ozone injections, additional investigation or referrals may need to be considered.

CONSENT

Written informed consent was obtained from the patient for publication of this case report.

COMPETING INTERESTS

The author declares no competing interests.

REFERENCES

1. Blake P, Burstein R. Emerging evidence of occipital nerve compression in unremitting head and neck pain. *J Headache Pain*. 2019;20(1):76. Published 2019 Jul 2. doi:10.1186/s10194-019-1023-y
2. Lee M, Brown M, Chepla K, Okada H, Gatherwright J, Totonchi A, et al. An anatomical study of the lesser occipital nerve and its potential compression points: implications for surgical treatment of migraine headaches. *Plast Reconstr Surg*. 2013;132(6):1551–1556.
3. Janis JE, Hatef DA, Ducic I, Reece EM, Hamawy AH, Becker S, et al. The anatomy of the greater occipital nerve: part II. Compression point topography. *Plast Reconstr Surg*. 2010;126(5):1563–1572. doi: 10.1097/PRS.0b013e3181ef7f0c
4. Schmid AB, Coppieters MW, Ruitenberg MJ, McLachlan EM. Local and remote immune-mediated inflammation after mild peripheral nerve compression in rats. *J Neuropathol Exp Neurol*. 2013;72(7):662-680. doi:10.1097/NEN.0b013e318298de5b
5. Berini SE, Spinner RJ, Jentoft ME, et al. Chronic meralgia paresthetica and neurectomy: a clinical pathologic study. *Neurology*. 2014;82(17):1551-1555. doi:10.1212/WNL.0000000000000367
6. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition. *Cephalalgia*. 2018;38(1):1-211. doi:10.1177/0333102417738202
7. Re L, Sanchez GM, Mawsouf N. Clinical evidence of ozone interaction with pain mediators. *Saudi Med J*. 2010;31(12):1363-1367.
8. Fuccio C, Luongo C, Capodanno P, et al. A single subcutaneous injection of ozone prevents allodynia and decreases the over-expression of pro-inflammatory caspases in the orbito-frontal cortex of neuropathic mice. *Eur J Pharmacol*. 2009;603(1-3):42-49. doi:10.1016/j.ejphar.2008.11.060
9. Balkanyi A. The interaction between ozone therapy and oxygen radicals and their importance in practice; Proceedings of the Ninth Ozone World Congress; New York, NY. June 3–9, 1989; International Ozone Association Pan American Committee; pp. 22–27.
10. Hidalgo-Tallón FJ, Torres-Morera LM, Baeza-Noci J, Carrillo-Izquierdo MD, Pinto-Bonilla R. Updated Review on Ozone Therapy in Pain Medicine. *Front Physiol*. 2022;13:840623. Published 2022 Feb 23. doi:10.3389/fphys.2022.840623
11. Yadla S, Ratliff JK, Harrop JS. Whiplash: diagnosis, treatment, and associated injuries. *Curr Rev Musculoskelet Med*. 2008;1(1):65-68. doi:10.1007/s12178-007-9008-x

Regenerative/Injection Management of a Patient with Achilles Tendinopathy: A Case Report

Bruce V. Gundersen, DC, DIANM¹

¹ Holladay Physical Medicine, Salt Lake City, UT

brucegundersen@gmail.com

Published: June 2022

Journal of the International Academy of Neuromusculoskeletal Medicine

Volume 19, Issue 1

This is an Open Access article which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. The article copyright belongs to the author and the International Academy of Neuromusculoskeletal Medicine and is available at: <https://ianmmmedicine.org/> © 2022

ABSTRACT

Background: This paper presents a case report of physical medicine management of a patient with Achilles tendinopathy.

Case Presentation: A 42 year old female presented with a chief complaint of acute Achilles tendon and gastrocnemius pain on the left side. Her history included successful completion of over 100 half marathon races in the past without intense pain. Current pain prohibits running or non-antalgic walking. The history of intervention included self-care and advice from physical therapists, other runners, interested bloggers, and well-meaning relatives.

Diagnosis and Disposition: Achilles tendinopathy is a common cause of disability. Despite the economic and social relevance of the problem, the causes and mechanisms of Achilles tendinopathy remain unclear. Tendon vascularity, gastrocnemius-soleus dysfunction, age, sex, body weight and height, pes cavus, transverse and longitudinal arch dysfunction, and lateral ankle instability are considered common intrinsic factors. The essence of Achilles tendinopathy is a failed healing response, with haphazard proliferation of tenocytes, some evidence of degeneration in tendon cells and disruption of collagen fibers, and subsequent increase in non-collagenous matrix. Tendinopathic tendons have an increased rate of matrix remodeling, leading to a mechanically less stable tendon which is more susceptible to

damage. The diagnosis of Achilles tendinopathy is mainly based on a careful history and detailed clinical examination. The latter remains the best diagnostic tool. Over the past few years, various new therapeutic options have been proposed for the management of Achilles tendinopathy. Despite the morbidity associated with Achilles tendinopathy, many of the therapeutic options described and in common use are far from scientifically based. New minimally invasive techniques of subdermal or intramuscular injection of ozone at various concentrations seem to allow faster recovery and accelerated return to sports, rather than open surgery. A genetic component has been implicated in tendinopathies of the Achilles tendon, but these studies are still at their infancy.

Keywords: Achilles, Tendinopathy, Tendonitis, Ozone, Injection, Neuromusculoskeletal Medicine, Fascia, Therapy, Soft Tissue, Chiropractic, Runner, Chronic Pain

INTRODUCTION

About 52% of runners experience Achilles tendinopathy (AT) in their lifetime. In the United States military, the rate of clinically diagnosed AT cases was 5/1000 person-yr in 2015. The pathophysiology can be viewed on a continuum proceeding from reactive tendinopathy where tenocytes proliferate, protein production increases, and the tendon thickens; to tendon disrepair in which tenocytes and protein production increase further and there is focal collagen fiber disruption; to degenerative tendinopathy involving cell death, large areas of collagen disorganization, and areas filled with vessels and nerves. Inflammation may be present, especially in the early phases. Some evidence suggests AT pain may be due to neovascularization and the ingrowth of new nerve fibers in association with this process. Prospective studies indicate that risk factors include female sex, Black race, higher body mass index, prior tendinopathy or fracture, higher alcohol consumption, lower plantar flexion strength, greater weekly volume of running, more years of running, use of spiked or shock absorbing shoes, training in cold weather, use of oral contraceptives and/or hormone replacement therapy, reduced or excessive ankle dorsiflexion range of motion, and consumption of antibiotics in the fluoroquinolone class. At least 10 simple clinical tests are available for the diagnosis of AT, but based on accuracy and reproducibility, patient self-reports of morning stiffness and/or pain in the tendon area, pain on palpation of the tendon, and detection of Achilles tendon thickening appear to be the most useful. Both ultrasound and magnetic resonance imaging (MRI) are useful in assisting in diagnosis with MRI providing slightly better sensitivity and specificity. Conservative treatments that have been researched include: (1) nonsteroidal anti-inflammatory medication, (2) eccentric exercise, (3) stretching, (4) orthotics, (5) bracing, (6) glyceryl trinitrate patches, (7) injection therapies (corticosteroids, hyaluronic acid, platelet-rich plasma injections), (8) shock wave therapy, and (9) low-level laser therapy. Nonsteroidal anti-inflammatory medication and corticosteroid injections may provide short-term relief but do not appear effective in the longer term. Eccentric exercise and shock wave therapies are treatments with the highest evidence-based effectiveness. Prevention strategies have not been well researched, but in

specific populations balance training (soccer players) and shock-absorbing insoles (military recruits) may be effective. Ultrasound scans might be useful in predicting future AT occurrences.



The illustration above shows the irritation and inflammation of the tendon, its return to “normal” and the result after repetitive episodes of inflammation, disability, and symptomatic recovery. Eventual tears on the tendon result.

CASE PRESENTATION

The patient is a 42 year old female with a 16 year history of long distance running who provided the following information:

HISTORY NARRATIVE

“I started running seriously in 2005 after my first baby at age 26, and alternated 1 marathon each year with a baby the following year. My mileage began to increase significantly in May of 2014 as I began to run a half marathon each month for almost 6 years. My weekly mileage fluctuated from 10-30 miles consistently, even with occasional injury. Most of my issues were related to my feet and calves, from what I believed to be a weak core from separated abdominal muscles from 4 pregnancies in 6 years. I also have an injury from a bone chip in my left quad from 34 years ago. I've been able to avoid knee and muscle pain in that leg.

When I had injuries related to running, I used muscle stimulation, massage, and foam rolling to ease pain and get back to running, and lots of deep stretching, including 8 years of weekly Hot Bikram Yoga. Around the end of May 2021, I developed pain in my Achilles tendon while running, mostly in my right leg. I received 1 ozone shot in my right Achilles in June 2021.

In mid-July of 2021, I ran 4 half marathons 4 days in a row to complete a 4 state challenge. The courses were all very flat, but I finally started to feel the effects in my calves, ankles, and feet. My usual routine of therapy did not solve the problem of overuse. By the end of July 2021, both Achilles tendons hurt when running and with regular walking.

I received ozone shots in both right and left Achilles tendons August 5th, 2021. My calves felt better and I started really training for my next marathon in January of 2022. In September 2021, I finished my 100th half marathon at age 42. No pain in my feet or Achilles tendons since those shots. I just completed my 6th marathon and 103rd half marathon.

When I had a gait analysis done (in 2010) it revealed that I had weak abdominal muscles that had been stretched and separated during pregnancies. I had a tummy tuck (2019) to re-attach my separated abdominal muscles to help my balance and strength during running. I had 4 pregnancies in 6 years, and I am currently still working on proper alignment and core strength during running. All of this has affected my calves and feet in marathon training. My Achilles pain in both legs occurred 1 year post surgery as the alignment of my body was different and I began to ramp up weekly mileage.”

PAST HISTORY NARRATIVE

“I have a 34 year old injury in my left quad. I had a three-wheeling accident that resulted in a bone chip in my left femur, which has calcified. It does not cause any pain, but I do have weakened quad muscles as a result and I take particular time to strengthen my quads and hamstrings to counter the effects of that injury. I’ve noticed more consistent problems in my left leg because of this injury.”

EXAMINATION

This 42 year old female presented with complaints of Achilles tendon and calf pain self-rated on the visual analogue pain scale (VAS) at 8. Inspection of the foot, ankle calf, thigh, and pelvis was unremarkable. Palpation of the bony prominences revealed tenderness of the calcaneus bilaterally. Palpation of the soft tissues showed tenderness on the dorsum of the feet and the Achilles tendons. Mensuration of the thighs and legs were symmetrical respectively. Muscle grading of the hip, knee, and foot were all grade 5 with noted pain on dorsiflexion of the right foot. Compression of the right calf increased the pain response. Heel and toe walking and other muscle testing of the ankle could not be performed due to guarding. Deep tendon reflex of the Achilles was also guarded beyond value. Thompson’s test for tear of the Achilles tendon was negative but performance produced increased pain.

MANAGEMENT AND OUTCOME

The patient was treated on two separate occasions. The first encounter began with the patient rating her Achilles pain at 10 on VAS. Treatment began and included ozone

injection into two subcutaneous regions adjacent to the most tender points of the right Achilles tendon, one medially and one laterally using 10 cc and 8 cc respectively of 19 gamma dosage. The injections sites were prepared using cold pack anesthesia, isopropyl alcohol and silver gel. Injection technique included specific, brisk penetration, blowing the plug, delivering the active ingredient, and retrograding withdrawal at each site. The patient reported extreme pain on full delivery of the ozone which lasted for about 2 minutes. The patient was then instructed to do mild ROM movements of the foot and ankle to pain tolerance. A 15 minute application of electrical stimulation using graduating and recurring frequency of Bi-Polar current from 1 to 150 pps increased to patient tolerance. Specific manual manipulations of the foot and ankle joints were performed and included the general foot mobilization, modified metatarsal shear, foot figure eight, a modified version of the Achilles tendon technique, supine plantar cuneiform, Tarsal – Metatarsal shear, posterior tibia and fibula techniques. Considerable extra-vertebral manipulation is important. This was followed by specific percussive massage using a Theragun with very mild trigger point massage for 10-40 seconds per point. Treatment concluded with mild eccentric exercise of the foot, ankle, and calf muscles using surgical hose as resistance. Treatment time was a total of 31 min. The patient rated her pain on exit as 0 (VAS) and was instructed to return PRN.

The second encounter was almost two months later when complaints of bilateral Achilles tendonitis were reported. Several races had been run during the interim two months. The patient reported gradual increase in pain due to several races. She reported 10 VAS on the right and 8 on the left. The same protocol was delivered only this time on both sides. The patient again experienced extreme pain on full delivery of the ozone which lasted for about 2 minutes. Following the completion of the treatment protocol, pain level was again reported at 0 (VAS) bilaterally. The patient returned 7 months later for an unrelated complaint and coincidentally reported having run 10 half marathons and has had no return of the Achilles complaint on either side.

LIMITATIONS

The author recognizes limitations of this case study. Generalization of the diagnostic findings and outcomes represented in this case may not necessarily apply to other patients. Measurements of the outcomes have not been globally standardized in all respects.

CONSENT

Written consent for publication was obtained from the patient.

COMPETING INTERESTS

The author declares no competing interests.

REFERENCES

1. Smith NL, Wilson AL, Gandhi J, Vatsia S, Khan SA. Ozone therapy: an overview of pharmacodynamics, current research, and clinical utility. *Med Gas Res*. 2017;7(3):212-219. Published 2017 Oct 17. doi:10.4103/2045-9912.215752
2. Shallenberger, Frank. *Bursting with Energy*. Turner Publishing Company. Kindle Edition. pp. 473-474
3. Bonetti M, Fontana A, Cotticelli B, Volta GD, Guindani M, Leonardi M. Intraforaminal O(2)-O(3) versus periradicular steroidal infiltrations in lower back pain: randomized controlled study. *AJNR Am J Neuroradiol*. 2005;26(5):996-1000.
4. Hoppenfeld S. *Physical Examination of the Spine and Extremities* – 1976, Chapter 8.
5. Evans R. *Illustrated Essentials in Orthopedic Assessment* – 1994, Chapter 12.
6. Kirk C, Lawrence D, Valvo N. *States Manual of Spinal, Pelvic and Extravertebral Tehchnics*, Second Edition – 1985, Part 3.
7. Fischer J. *Atlas of Injection Therapy in Pain Management* – 2012, Chapter 7.
8. Malanga G, Ibrahim V. *Regenerative Treatments in Sports and Orthopedic Medicine* – 2018.
9. Longo UG, Ronga M, Maffulli N. Achilles tendinopathy. *Sports Med Arthrosc Rev*. 2009;17(2):112-126. doi:10.1097/JSA.0b013e3181a3d625
10. Rompe JD, Furia JP, Maffulli N. Mid-portion Achilles tendinopathy--current options for treatment. *Disabil Rehabil*. 2008;30(20-22):1666-1676. doi:10.1080/09638280701785825
11. Maffulli N, Longo UG, Kadakia A, Spiezia F. Achilles tendinopathy. *Foot Ankle Surg*. 2020;26(3):240-249. doi:10.1016/j.fas.2019.03.009
12. Bliznakov EG, Watanabe T, Saji S, Folkers K. Coenzyme Q deficiency in aged mice. *J Med*. 1978;9(4):337-346.
13. Blomstrand R, Diczfalusy U, Sisfontes L, Svensson L. Influence of dietary partially hydrogenated vegetable and marine oils on membrane composition and function of liver microsomes and platelets in the rat. *Lipids*. 1985;20(5):283-295. doi:10.1007/BF02534261
14. De Schrijver R, Privett OS. Energetic efficiency and mitochondrial function in rats fed trans fatty acids. *J Nutr*. 1984;114(7):1183-1191. doi:10.1093/jn/114.7.1183
15. Tan SC, Chan O. Achilles and patellar tendinopathy: current understanding of pathophysiology and management. *Disabil Rehabil*. 2008;30(20-22):1608-1615. doi:10.1080/09638280701792268

16. McShane JM, Ostick B, McCabe F. Noninsertional Achilles tendinopathy: pathology and management. *Curr Sports Med Rep*. 2007;6(5):288-292.
17. Etzioni A, Levy J, Nitzan M, Erde P, Benderly A. Systemic carnitine deficiency exacerbated by a strict vegetarian diet. *Arch Dis Child*. 1984;59(2):177-179. doi:10.1136/ad.59.2.177
18. Lombard KA, Olson AL, Nelson SE, Rebouche CJ. Carnitine status of lactoovo vegetarians and strict vegetarian adults and children. *Am J Clin Nutr*. 1989;50(2):301-306. doi:10.1093/ajcn/50.2.301
19. Mozaffarian D, Katan MB, Ascherio A, Stampfer MJ, Willett WC. Trans fatty acids and cardiovascular disease. *N Engl J Med*. 2006;354(15):1601-1613. doi:10.1056/NEJMr054035
20. Ravaglia G, Forti P, Maioli F, et al. Effect of micronutrient status on natural killer cell immune function in healthy free-living subjects aged ≥ 90 y. *Am J Clin Nutr*. 2000;71(2):590-598. doi:10.1093/ajcn/71.2.590
21. Czervionke LF. Lumbar intervertebral disc disease. *Neuroimaging Clinics of North America* 3:465-485, 1993.
22. Davis RA. A long-term outcome analysis of 984 surgically treated herniated lumbar discs. *J Neurosurg*. 1994;80(3):415-421. doi:10.3171/jns.1994.80.3.0415
23. Iliakis E. Ozone treatment in low back pain. *Orthopaedics* 1995; 1: 29–33.
24. Andreula CF, Simonetti L, De Santis F, Agati R, Ricci R, Leonardi M. Minimally invasive oxygen-ozone therapy for lumbar disk herniation. *AJNR Am J Neuroradiol*. 2003;24(5):996-1000.
25. Fabris, G., et al. Percutaneous Treatment of Lumbar Herniated Disk: 10 Years of Experience in Udine. *Rivista di Neuroradiologia* 10.5 (1997): 523-532.
26. Zennaro H, Dousset V, Viaud B, et al. Periganglionic foraminal steroid injections performed under CT control. *AJNR Am J Neuroradiol*. 1998;19(2):349-352.
27. Grant G. Primary afferent projections to the spinal cord. In: *The Rat Nervous System*. 2nd ed. San Diego: Academic Press;1995:61– 66.
28. Siddall PJ, Cousins MJ. Spinal pain mechanisms. *Spine (Phila Pa 1976)*. 1997;22(1):98-104. doi:10.1097/00007632-199701010-00016
29. O'Donnell JL, O'Donnell AL. Prostaglandin E2 content in herniated lumbar disc disease. *Spine (Phila Pa 1976)*. 1996;21(14):1653-1656. doi:10.1097/00007632-199607150-00007

30. Kawakami M, Tamaki T, Hashizume H, Weinstein JN, Meller ST. The role of phospholipase A2 and nitric oxide in pain-related behavior produced by an allograft of intervertebral disc material to the sciatic nerve of the rat. *Spine* (Phila Pa 1976). 1997;22(10):1074-1079. doi:10.1097/00007632-199705150-00004
31. Saal JS, Franson RC, Dobrow R, Saal JA, White AH, Goldthwaite N. High levels of inflammatory phospholipase A2 activity in lumbar disc herniations. *Spine* (Phila Pa 1976). 1990;15(7):674-678. doi:10.1097/00007632-199007000-00011
32. Group M, Stanton-Hicks M. Neuroanatomy and pathophysiology of pain related to spinal disorders. *Radiol Clin North Am*. 1991;29(4):665-673.
33. Vanderlinden RG. Subarticular entrapment of the dorsal root ganglion as a cause of sciatic pain. *Spine* (Phila Pa 1976). 1984;9(1):19-22. doi:10.1097/00007632-198401000-00006
34. Wall PD, Devor M. Sensory afferent impulses originate from dorsal root ganglia as well as from the periphery in normal and nerve injured rats. *Pain*. 1983;17(4):321-339. doi:10.1016/0304-3959(83)90164-1
35. Weinstein J. Report of the 1985 ISSLS Traveling Fellowship. Mechanisms of spinal pain. The dorsal root ganglion and its role as a mediator of low-back pain. *Spine* (Phila Pa 1976). 1986;11(10):999-1001. doi:10.1097/00007632-198612000-00005
36. McCarron RF, Wimpee MW, Hudkins PG, Laros GS. The inflammatory effect of nucleus pulposus. A possible element in the pathogenesis of low-back pain. *Spine* (Phila Pa 1976). 1987;12(8):760-764. doi:10.1097/00007632-198710000-00009

Chiropractic Care and Rehabilitation Combined with Myofascial Release Technique After Double Mastectomy: A Case Report

Jesse Hodges, DC, MS, CSCS¹

Nicole Beck, CPT²

¹ Associate Professor, Palmer College of Chiropractic, FL

² Student, Palmer College of Chiropractic, FL

Jesse.hodges1@palmer.edu

Published: June 2022

Journal of the International Academy of Neuromusculoskeletal Medicine

Volume 19, Issue 1

This is an Open Access article which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. The article copyright belongs to the author and the International Academy of Neuromusculoskeletal Medicine and is available at: <https://ianmmmedicine.org/> © 2022

ABSTRACT

Objective: The purpose of this report is to examine the use of chiropractic care and rehabilitation combined with myofascial release to treat a patient diagnosed with breast cancer who underwent a double mastectomy to remove all cancerous lymph nodes.

Methods: A 67-year-old female presented with a chief complaint of bilateral radiating numbness and swelling into her hands after a radical double mastectomy to remove all cancerous lymph nodes. Previous chiropractic care and physical therapy had no impact on her symptoms. All shoulder range of motion was decreased at the start of treatment. A wall angel functional assessment induced pain and inability to place her arms into the 90-degree position on the wall indicating external rotator cuff and pec minor involvement bilaterally. All motor and reflex testing was within normal limits, sensation was decreased along the C6 and C7 dermatomes bilaterally.

Results: Passive myofascial release was performed on the infraspinatus and pectoralis minor muscles bilaterally along with a prone diversified chiropractic adjustment to restore motion to the cervicothoracic junction at C7-T1. An immediate increase in shoulder range of

motion was noted as well as an increase in scapulohumeral rhythm. The patient was seen once per week for 6 weeks and given stretches for the infraspinatus and pectoralis minor muscles bilaterally to be performed 3 to 5 times per day with 30 second holds.

Conclusion: The patient's symptoms decreased after one treatment, and no symptoms were reported after 4 treatments. Chiropractic care along with myofascial release can potentially improve the quality of life of a patient even after major surgery.

Key Words: Lymph Nodes, Breast Cancer, Myofascial Release, Chiropractic

BACKGROUND

When cells of the body react abnormally to external and/or internal change, they can reproduce more rapidly than normal leading to cancerous formations. This case study will focus primarily on the formation of cancerous cells within the breast tissue. Breast cancer can be identified may be identified by a small hard and nontender nodule during self-inspection or during a yearly examination given by the patient's primary medical physician. Signs and symptoms of breast cancer can include, but are not limited to, a lump, thickening of the surrounding skin, rash, dimpling of the skin, and redness or swelling of the affected area. Identification of any of these signs or symptoms should be immediately discussed with the patient's health care provider.¹⁻³

It is recommended that women begin regular mammography screening beginning at age 45 unless a patient is high risk.^{4,5} If self-inspection finds something of concern, a mammogram, ultrasound, or possibly magnetic resonance imaging (MRI) of the breast may be ordered.⁴

Mammogram is a low-dose x-ray of the breast used to look for presence of disease. Ultrasound or sonogram uses high-frequency sound waves to create a picture of internal organs. MRI uses magnetic field and radio waves to create a detailed picture of the organs and tissues within the body.^{1,5,6} These tests may indicate something suspicious, but only a biopsy, which is the removal of tissue for examination under a microscope, can definitively diagnose breast cancer.⁷

Breast cancer is categorized into 4 stages based on the severity and progression of the disease.⁵ Stage 1 is clinically non-invasive which means the cancer is isolated to the location in which it is found and is unlikely to spread to other parts of the body. Stage 2 is determined by the size of the cancer cells found; typically, the tumor will be measured between 2-5 centimeters to have this classification. Stage 3 is determined when the tumor is over 5 centimeters in size and more than 4 lymph nodes are involved. Stage 4 is classified when the cancer metastasizes beyond the breast and lymph nodes to other parts of the body.⁵

The literature is limited with information about breast cancer surgery with lymph node involvement and chiropractic care, however other research suggests that decreased range of motion of the scapula and shoulder can directly affect activities of daily living in a normally

healthy individual.⁸

CASE PRESENTATION

A 67-year-old female presented with a chief complaint of bilateral radiating numbness and swelling into her hands. She had been diagnosed with stage 2 breast cancer and underwent a radical double mastectomy to remove all cancerous lymph nodes. Previous chiropractic care and physical therapy had no impact on her symptoms. All shoulder range of motion was decreased at the start of treatment. Wall angel functional assessment showed pain and inability to place her arms into the 90-degree position indicating external rotator cuff and pec minor involvement bilaterally. The patient reported she had been under previous chiropractic care which consisted strictly of spinal adjustments and gave little to no relief causing her to seek other treatment possibilities. Physical therapy required her to wear compressive bandages to help reduce the post-surgical lymphadenopathy. She reported that this caused her a lot of discomfort and limited her already decreased ability to perform the simplest of daily living tasks. She reported taking multiple over the counter non-steroidal anti-inflammatory medications with no improvement. Her condition was hindering her ability to perform daily activities and her job performance.

The initial examination revealed all motor and reflex testing to be within normal limits, and sensation was decreased along the C6 and C7 dermatomes bilaterally. The patient's blood pressure was 135/70 and pulse was 65. Decreased active shoulder range of motion was observed bilaterally. This was confirmed by a positive O'Donoghue test, which reproduced pain during resisted range of motion (ROM). Passive range of motion was within normal limits for all shoulder motion and no pain or discomfort was reported, decreasing the possibility of ligamentous damage. Resisted range of motion causing pain indicated that the causative factor was muscular. Soft tissue palpation revealed hypertonicity in both the infraspinatus and pectoralis minor muscles bilaterally.

Myofascial release technique, a manual therapy used to relax hypertonic musculature by stripping the muscle belly from origin to insertion while passively moving the affected joint through its proper range of motion, was performed on the infraspinatus and pectoralis minor muscles bilaterally. A prone diversified chiropractic adjustment was performed on the cervicothoracic junction at C7-T1 to restore motion to the spine. The patient demonstrated an immediate increase in shoulder range of motion and reported a relief in tension throughout the upper body. She was given home stretches to be performed 5 times per day for 30 second holds to assist with the healing process, with hopes of further increasing shoulder range of motion over time. She was treated once per week for 6 weeks before a reevaluation was performed. Her symptoms diminished from daily to weekly occurrence over the first 2 weeks of treatment, and at the time of reevaluation she reported no symptoms.

Figure 1 shows the bandage treatment prescribed by the physical therapist. **Figures 2 and 3** show the prescribed stretches for the infraspinatus and pectoralis minor, respectively.



Figure 1



Figure 2



Figure 3

CONCLUSION

While breast cancer is prevalent and well researched in many areas, this case offers a unique perspective as a chiropractic physician was an integral part of the rehabilitative efforts. Using myofascial release technique of hypertonic muscles, chiropractic adjustive therapies, and rehabilitative exercises on a patient with a history of double mastectomy and postsurgical lymphedema opens the door for further research into the potential benefits of chiropractic care in relieving symptoms caused by serious illness.

LIMITATIONS

This case report is limited in scope and double mastectomy combined with bilateral numbness in both hands is not a common clinical presentation.

CONSENT

Written consent for publication was obtained from the patient.

COMPETING INTERESTS

The authors declare that they have no competing interests.

REFERENCES

1. Morrell RM, Halyard MY, Schild SE, Ali MS, Gunderson LL, Pockaj BA. Breast cancer-related lymphedema. *Mayo Clin Proc.* 2005;80(11):1480-1484. doi:10.4065/80.11.1480
2. Anderson, L, Hojris, I, Erlandesen, M, Andersen, J. Treatment of Breast-Cancer-related Lymphedema with or without lymphatic drainage: A randomized study. *Acta Oncologica.* 2009;39(3), 399-405.
3. Fourie, WJ, Robb, KA, (2009). Physiotherapy management of axillary web syndrome following breast cancer treatment: Discussing the use of soft tissue techniques. *Physiotherapy.* 2009;(95), 314-320.
4. McKenzie, DC, Kalda, AL. Effect of Upper Extremity Exercise on Secondary Lymphedema in Breast Cancer Patients: A Pilot Study. *Journal of Clinical Oncology.* 2003;(21), 463-466.
5. Stubblefield MD, Custodio CM. Upper-extremity pain disorders in breast cancer. *Arch Phys Med Rehabil.* 2006;87(3 Suppl 1):S96-S101. doi:10.1016/j.apmr.2005.12.017
6. Alcantara J, Alcantara JD, Alcantara J. The chiropractic care of patients with cancer: a systematic review of the literature. *Integr Cancer Ther.* 2012;11(4):304-312. doi:10.1177/1534735411403309

7. https://www.youngsurvival.org/uploads/pdf/educational-materials/BreastHealthAndYou-2016_FINAL.pdf
8. Kanga, I, Steiman, I. Chiropractic management of a patient with breast cancer metastases to the brain and spine: A case report. *JCCA*. 2015;59(3), 269-278.