RESEARCH ARTICLE

Meralgia Paresthetica: A 5 Model Osteopathic Approach

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KEYWORDS:

Meralgia Paresthetic

Osteopathic Medicine

Osteopathic Manipulative Treatment

Meralgia Paresthetica is the diagnostic term used to describe a neuropathy of the lateral femoral cutaneous nerve and typically presents with numbness, tingling, paresthesias or any other sign of nerve impingement along the anterior and lateral thigh. This condition is quite debilitating and bothersome for patients and typically is underdiagnosed in the outpatient setting, partially due to symptoms being attributed to other hip or lumbar spine causes. This article will provide an osteopathic perspective on this condition with all of its probable causes as well as a reference for a structured approach to managing the patient with osteopathic manipulative medicine. In addition, home exercises, stretches, and behavioral adaptations will be mentioned in order to maintain the results of the osteopathic manipulative treatment. A case report will be presented and specific findings related to this condition will be explained. Osteopathic manipulative treatment, along with behavioral retraining, should be considered as treatment options and offered to patients prior to pursuing more invasive therapeutic measures.

INTRODUCTION

Meralgia paresthetica (MP) is the diagnostic term given to a patient presenting with a mononeuropathy of the lateral femoral cutaneous nerve (LFCN) and typically presents with numbness, tingling, paresthesias, or any other sign of nerve impingement along the anterior and lateral thigh. It is specifically due to compression of this nerve as it passes over the anterior superior iliac spine and then under the inguinal canal at the lateral end as it progresses. It may be more commonly seen in individuals who are obese, have diabetes, or wear their pants too tight at the waist. Presenting clinical signs and symptoms may be sufficient for obtaining the diagnosis, however, electrodiagnostic studies may be useful in confirming the diagnosis and quantifying the degree of nerve involvement.[1] Treatment can vary from activity modification and a holistic approach to TENS unit applications, analgesics and local and/or systemic anti-inflammatories. Surgical interventions with neurolysis or neurectomies for those with significant weakness and atrophy or focal conduction blocks on electrodiagnostic examinations have also been used as treatment modalities.[2] Given that this condition can be considered an inflammatory mononeuropathy based on its clinical presentation, relieving compression of the nerve along its course with osteopathic manipulation and behavioral modifications can provide symptomatic relief and restore normal functioning of the lateral femoral cutaneous nerve.

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 Print ISSN: 1877-573X

MP typically occurs in patients ranging from 30 to 60 years of age and has an approximate incidence of 4-10/10,000 people.[3] The age group of 55-64 years old had the highest incidence of MP in both men and women.[1] It is estimated that the actual incidence may be a little higher due to under-reporting of symptoms and physicians attributing MP to other causes of hip and/or thigh pain. The incidence rates are similar between men and women, ruling out gender as a determinant.

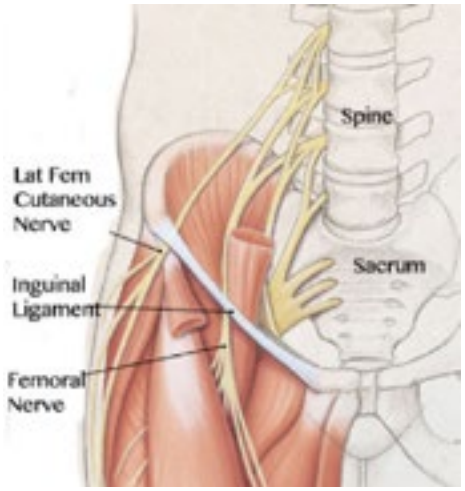
In addition to these factors, there are several behavioral and genetic causes that can contribute to the symptoms of MP. Consistent use of tight belts, tight sweat pants, corsets, or body armor have been associated with the development of symptoms of MP.[4,5] It is suspected that any extra added weight applied to the waist belt can also contribute (i.e. keys, cell phone/pager holders, gun holsters, etc.). Chronic repetitive positioning of the lower extremity in an extended or externally rotated position may also impinge on the nerve. MP as a post-op complication after orthopedic hip surgery, hernia repairs, or being in a prone position during surgery has also been noted.[6,7] In addition, pelvic crush fractures and pelvic osteotomies have also been linked with the prevalence of MP.[7] Among common genetic causes, patients with diabetes mellitus type 2 (DMT2) are seven times more likely to develop MP and there is a strong association of MP as a precursor to DMT2.[1] Obesity, pregnancy, and increased BMI have been associated with MP due to increased abdominal girth.[1,8] Other causes of MP include benign pelvic masses, tumors along the iliac crests, and osteoid sarcomas.[7]

ETIOLOGY

A mononeuropathy of the lateral femoral cutaneous nerve (LCFN) is what brings about the symptoms of MP. Originating from the L2 and L3 spinal nerves, the lateral femoral cutaneous nerve enters the pelvic cavity lateral to the psoas muscle. It continues towards the ASIS over the iliacus muscle. Many variants can be found for its path as it makes a right angle to exit the pelvic cavity above, below, or through the inguinal canal. Whichever course it takes, it is usually within 5cm of the ASIS. Therefore, it passes through the lacuna muscularis, a passageway inferior to the inguinal canal that takes the nerve to the sartorius muscle. In this passageway it may travel along the iliopsoas muscle, which, if in a contracted state, may compress some nerve fibers. After passing this location, the nerve passes superficially over the sartorius muscle and branches into the anterior and posterior branches (Figure 1). There is also a common variant of the nerve piercing though the sartorius muscle. The anterior branch continues deep to the tensor fascia lata for about 5-10 cm down the lateral thigh and then pierces the fascia to become subcutaneous, where it may be subjected to mechanical stress. The lateral femoral cutaneous nerve takes a long and complicated pathway, which presents many opportunities for the nerve to become entrapped. As with any nerve, prolonged chronic repetitive trauma may lead to trophic changes of the skin, neuropathic pain, and underlying tissue changes.

FIGURE 1:

Anatomy of LCFN and Cutaneous Sensory Distribution



There are several pathological changes that may affect the LFCN under prolonged compression or entrapment. In addition to the disordered orientation of nerve fiber bundles intermixed with increased connective tissue components that can be present in compression neuropathies,[9,10] an autopsy study of 12 nerves revealed that the LFCN demonstrated local demyelination and Wallerian degeneration. Greater relevance for this case were the additional findings of endoneurial vascular changes and polarized internodal swellings suggesting that mechanical factors are responsible for the symptoms of MP.[10,11,12]

These findings suggest that an osteopathic structural exam on the patient with MP should be considered an essential component of the physical examination in order to identify areas of mechanical compression on the LFCN by means of somatic dysfunctions of the lumbar spine, pelvis, sacrum, and lower extremities.

CASE PRESENTATION

A 68 year old overweight female with a history of bilateral mastectomy secondary to breast cancer presented with numbness and tingling of the anterior and lateral aspect of the right lower extremity for the past several weeks. Given the presentation and excluding other causes of her symptoms, she had been diagnosed with MP by her PCP. She was then provided with NSAIDs to use as needed and referred to a neurologist for treatment. The neurologist, recognizing the neuropathic component of her symptoms, prescribed Gabapentin three times daily for symptoms. This medication did not provide the sufficient relief that the patient was expecting and produced significant side effects (i.e. weight gain). She was also referred to a general surgeon but had no interest in pursuing surgical intervention at this point. The patient had heard of osteopathic manipulation through a family member and decided to see if it would be particularly helpful. She had stopped taking Gabapentin by the time of this initial visit, and denied any recent trauma, surgery or prior injury to the region.

Upon eliciting a detailed history and performing an osteopathic structural exam, patient stated that she forgot to mention that she spends hours a day knitting on the couch in which she tucks her right foot under her buttocks and keeps her lower extremity flexed and externally rotated. Physical exam findings demonstrated normal stance, gait, muscle strength testing and reflexes of lower extremities. She did have diminished sensation to touch on the anterior and lateral aspect of the right thigh. Her BMI was found to be 29, otherwise vital signs and other physical exam findings were normal.

Osteopathic exam findings were consistent with the following: bilateral hypertonicity to the psoas muscles right sided more severe than left, L2 flexed, rotated, and sidebent right, positive ASIS compression test on right with innominate findings consistent with a posteriorly rotated and out-flared pelvis. A restriction in excursion of the right hemidiaphragm was noted during the inspiratory phase of thoracic respiration. In addition, a chronically hypertrophied tensor fascia lata muscle with tenderness to palpation and a taut iliotibial band were also noted on her right.

MANAGEMENT WITH OSTEOPATHIC MANIPULATIVE TREATMENT

Due to the several factors that may contribute to the symptoms of MP, management of the patient must encompass a multifactorial approach. Reports of successful treatment modalities range from surgery to electroacupuncture to off-label use of medications to behavioral modifications.[2,4,8] Given this variety, principles of patient-centered care with emphasis on the five models of osteopathic diagnosis and treatment would be beneficial for MP. The five models are the biomechanical model, the respiratory-circulatory model, the neurological model, the metabolic-energy model, and the behavioral model.[13,14] The patient above was treated with different modalities in order to fully address all components contributing to her symptoms, while maintaining specific emphasis on these models.

MUSCLE ENERGY TECHNIQUE TO INNOMINATE SOMATIC DYSFUNCTION

A few muscle energy techniques were applied to the patient to address structural abnormalities that may have been compromising the LFCN in order to address the biomechanical model. Particular attention was directed at the innominate somatic dysfunctions and to the lumbar spine.

For the innominate posterior rotation dysfunction, the patient laid supine slightly diagonal so that the right sacroiliac joint was off the edge of the table. The physician was standing on the right side of the table while his cephalad hand was placed over the patient's left ASIS to prevent the patient from rolling off the table. The caudad hand was placed on the distal femur, just proximal to the knee. The physician's caudad hand passively extended the patient's right hip, bringing the innominate into anterior rotation, until the edge of the restrictive barrier was reached (Figure 2). Be careful not to pass the barrier or over extend the hip as this may stretch and further irritate the LCFN. The physician instructed the patient to lift the right leg toward the ceiling while the physician applied an equal counterforce. This isometric contraction was maintained for 3-5 seconds, after which the patient was instructed to stop and relax. Once the patient has completely relaxed, the physician extended the patient's right hip to the edge of the new restrictive barrier. Three to five repetitions of these directions were performed after which reassessment of the area was performed.[15]

For the right out-flared innominate dysfunction: The patient laid supine while the physician stood on the left side of the table. The patient's right hip and knee were flexed to approximately 75 degrees and the right foot was positioned just lateral to the left knee. The physician's cephalad hand was placed under the patient's right innominate, grasping the medial aspect of the right PSIS. The physician's caudad hand was placed on the patient's right knee and adducted it until the edge of the restrictive barrier was reached (Figure 3). The physician instructed the patient to abduct the right hip while the physician applied an equal counterforce. This isometric contraction was maintained for 3-5 seconds, then the patient was instructed to stop and relax. Once the patient completely relaxed, the physician further adducted the patient's right knee to the edge of the new restrictive barrier and drew traction laterally on the right PSIS. Three to five repetitions of these directions were performed after which reassessment of the area was indicated.[15]

MUSCLE ENERGY TECHNIQUE TO LUMBAR SD (L2FRSR)

The patient was placed in the right lateral recumbent position with the knees and feet together and shoulders and pelvis perpendicular to the table. The physician stood in front of the patient and with his cephalad hand he contacted the L2-L3 level. With his caudad hand, the physician flexed the patient's hips and knees until motion was localized at the L3 vertebral segment. The patient was instructed to straighten her lower leg and with his caudad hand the physician passively extended it to achieve motion at the L3 vertebral segment. The patient's left top leg was flexed and the foot placed in the popliteal space of the extended, bottom leg. The physician then switched hands so that the caudad hand was monitoring the segment. The now cephalad hand grasped the patient's lower arm and gently pulled it in an anterior and caudad direction until a rotational motion was felt at the L2-L3 level. The lower arm was then moved posterior and superior to induce left side bending. The patient should be grabbing the head of the table now with the upper body in a supine position. The patient was then instructed to take a deep breathe, exhale completely and while doing so reach further inferiorly by grasping the edge of the table with the superior arm and to continue this for a few respiratory cycles. The physician now grasped distal to the knee of the patient's top leg and elevated it until side bending of the lumbar spine was achieved to the motion barrier while monitoring the segment with the cephalad hand (Figure 4). The patient was asked to push her elevated foot down toward the table for 3 to 5 seconds, while the physician provided an isometric resistance. The patient was instructed to relax for 3-5 seconds after which the physician further elevated the foot until motion was noticed at the new barrier. The procedure was repeated at least three times after which the somatic dysfunction improved. Finally, a passive stretch was performed after the last repetition.[16]

FIGURE 2:

Posterior Innominate Muscle Energy

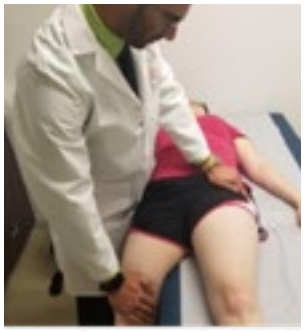


FIGURE 3:

Outflare Innominate Muscle Energy

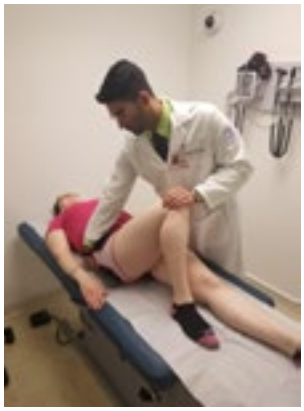


FIGURE 4:

Lumbar Spine Muscle Energy

FIGURE 5:

Diaphragmatic Lift Technique

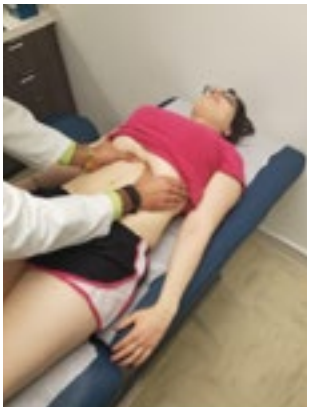
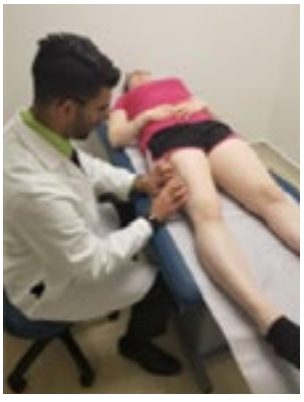


FIGURE 6:

Peripheral Nerve OMT of LFCN

FIGURE 7:

Peripheral Nerve OMT of LCFN Perforating Branches



DIAPHRAGMATIC LIFT TECHNIQUE

Due to the connections of the psoas muscle with the crural muscles at the posterior abdominal wall, it would be common to find diaphragmatic restrictions associated with psoas muscle spasms. Relieving dysfunctions in the respiratory-circulatory realm by treating the diaphragm was considered to be significant to bring about changes in the patient's' condition. Dr. William G. Sutherland, an original student of the founder of osteopathic medicine, Dr. Andrew T. Still, described a diaphragmatic lift technique in which the ultimate goal would be to "draw the diaphragm cranially, elevating the floor of the thorax, drawing upward on the abdominal contents, promoting venous and lymphatic drainage from the lower half of the body."[17] Based on his description of this technique, the physician can stand on the side that is easiest for them facing the patient. Then the physician's thumbs were placed right inferior to the sternum at the costosternal junctions while the rest of the fingers were spread around the lateral edges of the ribs towards the patient's head. A cephalad and lateral lift is then exerted by the physician, lifting the diaphragm. The patient was instructed to exhale while the physician continued to lift the diaphragm in a cephalad and lateral direction. Tension was maintained and held while the patient inspired and was slightly increased during exhalation (Figure 5). Concomitantly, the patient was instructed to exhale immediately after inhaling and to not hold the breath in, while the physician continued to engage new barriers with cephalad and lateral tractions. Finally, when no more advancement of fingers were noted, the patient was told to rapidly exhale, while simultaneously doing the valsalva maneuver (closing throat and increasing intrathoracic pressure) and expanding her chest.

PERIPHERAL NERVE OSTEOPATHIC TREATMENT OF LFCN

A direct treatment to the lateral femoral cutaneous nerve would specifically address the neurological model of osteopathic diagnosis and treatment. The LFCN is usually treated in the region of its anterior branch which runs two fingerbreadths subcutaneously on a horizontal line over the pubic bone. In this technique, and as with perforating skin branches of other nerves, palpation of the nerve was done from distal to proximal, always at the skin surface. The LFCN specifically runs from the middle to the upper third of the leg. In this nerve lesion, at the anterior aspect of the tensor fascia lata, there was a small, hardened, and pressure-sensitive deepening in the skin. For the actual treatment, the patient was in a supine position with the legs extended and the physician's thumb glided from the middle of the leg to the anterior lateral surface while maintaining pressure (Figure 6). The treatment released the tension in the tissue and at the same time released the perforating branch from impingements. The perforating points can also be felt and identified. Applying pressure while gliding from the middle of the leg to the anterior lateral surface, as in the previous technique, can help release tension and further release perforating branches from impingement (Figure 7). Multiple pain pressure points were discovered and even disappeared completely after a few manipulations.[19]

BEHAVIORAL INTERVENTION

The metabolic-energy model attempts to optimize the patient's internal homeostasis by establishing a healthy relationship between energy production and expenditure. In this case, due to body habitus and body mass index, the patient was screened for the presence of DMT2 by obtaining a Hemoglobin A1c measurement, which was 5.4 (within normal limits). The importance of exercise and a well balanced diet were enforced during this encounter and the patient stated interest in following up appropriately. A referral to a nutritionist was then provided.

To address aspects of the behavioral model, the patient was instructed to remove aggravating factors that may compress the waist line in any manner: use of tight belts, tight pants, body armor, etc. She was also instructed to be cautious as to how she normally sits on the couch and to avoid prolonged sitting with the right leg tucked under her buttock. To maintain the biomechanical changes achieved with OMT, the patient was also advised to perform a stretching exercise at least twice a day which particularly encourages free motion of the diaphragm and posterior abdominal wall attachments.

EXERCISES – STANDING PSOAS & ACHILLES STRETCH

One's gait is truly important when diagnosing lower extremity somatic dysfunctions. Hypertonic muscles can alter gait and create new and chronic somatic dysfunctions. In this case, it was important to ensure that the psoas, hamstrings, quadriceps, gastrocnemius, and soleus muscles were appropriately stretched for an effective gait with proper swing phase and heel strike. The patient was encouraged to thoroughly stretch the psoas muscle by increasing lumbar lordosis and extending slightly at hip; then to transition into a stretch for the Achilles tendon, which particularly in females, tends to contract due to frequent use of high heels. Effective stretching of this tendon will assist in normal gait mechanics. Standing two feet from the wall and, facing it with feet shoulder-width apart, place palms on the wall at shoulder height. From this position it is important to keep elbows fully extended and attempt to extend the lower back to stretch muscles that support the lumbar spine by gently arching the pelvis towards the wall. The patient was instructed to maintain this position and breathe slowly and deeply for at least ten respiratory cycles. To transition into the Achilles tendon stretch, have the patient return the pelvis to a relatively neutral position and instruct to simply bend the knees as much as possible while keeping the heels on the floor. While maintaining this position breathe slowly and deeply for at least ten respiratory cycles (Figure 8). Repeat both stretches at least twice a day but more if tolerated.[18]

FIGURE 8:

Standing Psoas and Achilles Stretch

FOLLOW-UP

The patient returned for a follow-up encounter one week after the first visit with a renowned sense of empowerment. Her symptoms had not returned with the same severity at all, on the contrary, she stated that if she felt any tingling or numbing of her thigh she would immediately stand up and perform the stretching exercises indicated for her. This would temporarily ease and eventually stop the progression of symptoms altogether. Continuously performing the stretches and being very aware of the resting position of her right hip during her knitting on the couch allowed her to be free of symptoms of MP. She did not feel the need to take an NSAID for her MP but was content on the fact that if she had to resort to it she would without issues.

Osteopathic structural evaluation at follow-up was only significant for a restricted right pelvic diaphragm which was not evident at the initial visit. This could be a new somatic dysfunction or a compensatory mechanism after other somatic dysfunctions were treated. A simple pelvic diaphragm release was performed in a supine position, which the patient tolerated very well. The patient was instructed to continue with the exercises and stretches and to return to the office if symptoms returned, persisted, or worsened.

CONCLUSION

The etiology of MP can be one that is multifactorial, ranging from musculoskeletal dysfunctions to genetic causes. Given this wide array of causes, the treatment of MP should encompass a multifactorial approach. Utilizing the tenets of osteopathic medicine which state that "structure and function are intimately related" as well as "the body is capable of self-healing, self-regulating mechanisms" can provide the physician with an in-depth manipulative treatment rationale for MP and other mononeuropathies. In addition, once this approach is combined with a motivated patient's willingness to partake in a home exercise program and suggested behavioral modifications for relieving compression of the nerve, a successful outcome can be obtained. Empowering the patient with responsibility for their health by use of exercises and stretches also allows application of another tenet which is "the human is a combination of mind, body and spirit" at a deeper level of self-consciousness and self-realization. An osteopathic approach can be key in treating musculoskeletal causes and modifying behavioral contributions that has been refractory to other treatment options. Although both surgical and non-surgical approaches have been shown to be effective in treating the symptoms of MP, osteopathic manipulation should also be considered before more invasive treatment modalities.

AUTHOR DISCLOSURES:
No relevant financial affiliations.

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