Piriformis Syndrome: A Case Study

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Abstract

Although controversial in its diagnosis, piriformis syndrome is often overlooked as a cause of sciatica. A 51-year-old female patient with chronic low back pain presented to a teaching clinic in the Pacific Northwest 2 weeks after the onset of pain in her left buttock and left leg. Osteopathic manipulative treatment (OMT) provided some relief, but it did not completely resolve her symptoms. This case study examines the implication of OMT in acute cases of piriformis-induced sciatica.

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History and Presentation

A 51-year-old Caucasian female patient with medical history significant for chronic low back pain (LBP) and left leg length discrepancy was seen at a teaching clinic in the Pacific Northwest with complaints of left leg pain and exacerbation of LBP for the previous 2 weeks. The patient was routinely treated with osteopathic manipulative treatment (OMT) to address LBP and was last seen 2 weeks prior to this visit. Since her previous office visit, the patient stated her "back seized up," and she had experienced subsequent left leg pain. The patient denied having recent trauma or illness. She described the pain as varying from "sharp and shooting" to "throbbing and aching," with the latter being a greater concern to the patient.

The patient described the pain as originating in her low back and left buttock and radiating down her posterior thigh, wrapping around to the anterior surface of the lower limb. She rated her pain as being 6 on a 10-point scale at its worst, and she noted that the pain was intermittent. She was careful to avoid provoking factors, which included sitting, extension of the left lower leg, and the supine position (making it difficult to sleep). Palliating factors included standing and stretching and internal and external rotation of the left hip. Over-the-counter ibuprofen and acetaminophen seemed to improve symptoms temporarily. The patient denied having feelings of numbness or tingling, loss of strength, or loss of sensation to touch. She reported no decrease in performance of activities of daily living, but she avoided sitting at work in order to quell the pain. The patient also noted that she had been placing more weight on her right leg since her symptoms began. Although she did not report difficulties with ambulation, she felt that she needed to be overly cautious. She sought relief via adjustment or stretching and the use of a 4 mm lift for her short left leg. Although the patient feared that her symptoms were worsening, she expressed wishes to avoid prescription pain medication and imaging, and she had an immediate reluctance for surgical intervention.

The patient had no surgical history. Her family history was significant for controlled hypertension in her mother and substance abuse in her sister. The patient was unmarried and lived alone with six pets in a home she owned. The patient was a teacher at a special needs school. She denied any history of tobacco, alcohol, or illicit drug use. She routinely walked 2 miles a day for exercise, but she noted that she had decreased exercising due to pain.

Physical Examination

On physical exam, the patient had a blood pressure of 120/83 mmHg, a heart rate of 86, and a respiratory rate of 14. Her height

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was 5'4" (163 cm) and her weight 123 lbs (56 kg). The patient was well groomed, well developed and well nourished. She was alert and oriented to person, place, and time. She was not in acute distress. However, the patient paced the exam room because sitting exacerbated pain in her left buttock and leg. The cardiac exam revealed a regular rate and rhythm, normal S1/S2, without appreciation of murmurs, rubs, or gallops. Lungs were clear to auscultation bilaterally, without adventitious sound. A neurological examination revealed intact sensation to light touch and +5/5 muscular strength of the bilateral upper extremities and lower extremities. Deep tendon reflexes were +2/4 in the bilateral biceps, patellar, and Achilles tendons.

The osteopathic structural exam revealed widespread somatic dysfunction. In her cervical spine, the atlanto-occipital joint was extended, sidebent left, and rotated right. The atlanto-axial joint was rotated right, C3 was flexed with sidebending and rotation to the left, and C7 was found to be flexed with sidebending and rotation to the right. Counterstrain points were tender at LC1 and AC1-5 on the right. Thoracic dysfunction was significant for T5-T9 neutral, sidebent left, and rotated right. In the lumbar region, L3-5 were neutral, sidebent right, and rotated left with a tender counterstrain point of AL1 on the left. The patient had a positive seated and standing flexion test on the left, an anteriorly rotated innominate bone on the left, and a tender inguinal counterstrain point on the left. The straight leg raise test was positive on the left. The flexion, abduction, and external rotation (FABER) exam was negative bilaterally, but the patient was noted to be guarding on the left during the FABER examination. The patient had pain with flexion, abduction, and internal rotation of the left hip. The patient had an R/R forward sacral torsion and tender counterstrain points of the left piriformis and lower pole fifth level (LP5L). Examination of leg length discrepancies revealed a short leg on the right, though the patient had a previous diagnosis of short left leg.

Treatment

Osteopathic manipulative treatment (OMT) was performed to correct the somatic dysfunctions found on physical examination. Counterstain was used initially to address all tender points in the cervical, lumbar, pelvic, and sacral regions. Soft tissue and muscle energy techniques were then applied to the areas of dysfunction in hope of improving range of motion. After treatment, the patient reported a notable decrease in pain, but her symptoms persisted.

The patient was informed of potential pathology, including piriformis syndrome, sciatica, disk herniation, spondylolisthesis, rheumatoid arthritis, osteoarthritis, and compression fracture. The patient was instructed to continue with ibuprofen treatment, 400 mg to 800 mg up to 3 times daily as needed, for analgesia and anti-inflammation. The patient was given information on piriformis syndrome and sciatica to take home, and she was shown proper stretching techniques.

Further discussions were held regarding ongoing treatment. The patient was open to the idea of imaging her lower spine. Because the patient remarked that she would not elect surgical intervention, caudal epidural corticosteroid injections were discussed as a possible nonsurgical treatment for LBP and radiculopathy. The patient requested more information and indicated that she would consider beginning corticosteroid treatment on follow-up in addition to manipulation.

Discussion

First described by Freiburg and Vinke in 1934 and later confirmed by researchers such as Robinson and Te Poorten, piriformis syndrome is a neuromuscular condition characterized by low back, hip, buttock, and leg pain.[1] It often results in compressing the sciatic nerve, leading to peripheral radiculopathy of the ipsilateral lower limb. Common features include tenderness over the greater sciatic notch and aggravation of symptoms with sitting and stretching of the piriformis muscle.[1] Piriformis syndrome is thought to be more prevalent in women than men, often occurring in the fourth to fifth decades of life. Although much research has been conducted on the discogenic causes of sciatica, piriformis syndrome

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For more information on terminology used in *The AAO Journal*, see the

Glossary of Osteopathic Terminology

developed by the American Association of Colleges of Osteopathic Medicine's Educational Council on Osteopathic Principles.

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as a nondiscogenic cause is often overlooked as a diagnosis. As a result, its true prevalence is unknown.[2]

Derived from L4-S3 nerve roots, the sciatic nerve is the largest nerve in the body. Containing both preaxial and postaxial branches, the sciatic nerve exits the greater sciatic foramen anterior (deep) to the inside of the piriformis muscle, extends posterior (superficial) to the short external rotators, and bifurcates at the apex of the popliteal fossa into the common peroneal and tibial nerves. It has mixed functions, providing both motor and sensory innervation.[3] The sciatic nerve's function is primarily external rotation of the hip and, to a lesser extent, abduction and flexion.[1]

The piriformis muscle originates on the anterior surface of S2-4, the sacroiliac joint, the anterior sacrospinous ligament, and the sacrotuberous ligaments. It inserts into the superior medial aspect of the greater trochanter of the femur. Innervation of the piriformis is supplied by (L5)S1-2, and the piriformis provides postural stability during ambulation and standing.

Congenital anatomical variation in both the sciatic nerve and piriformis is found in 15% to 30% of the population. Common variations include proximal bifurcation of the sciatic nerve, resulting in the tibial branch passing either superiorly or inferiorly to the piriformis; a bifid piriformis; and the sciatic nerve passing posterior (or superficial) to the inside of the piriformis muscle.[2]

Sciatica is a relatively common condition with a lifetime prevalence of 13% to 40%.[4] Stafford et al[4] found positive factors for developing sciatica to include increasing height (older age groups only), increasing age, genetic predisposition, a history of walking and jogging, seated occupations, and smoking. Gender, body mass, and parity typically have no influence on the development of sciatica. Prior to the work of Freiburg and Vinke, a herniated intervertebral disk was thought to be the dominant cause of sciatica. In addition to being caused by piriformis compression, nondiscogenic-induced sciatica can result from spinal stenosis in the lumbar canal, from a pelvis without piriformis involvement, and from injury to any point along the normal course of the nerve.[1] Pain can also be referred from the abdominal and pelvic viscera.

Boyajian-O'Neill et al[2] described piriformis syndrome as either primary or secondary in etiology. Primary piriformis syndrome is due to an anatomic variant as described above, and secondary piriformis results from trauma, overuse, and ischemic effects. According to this research, fewer than 15% of patients with piriformis syndrome have primary causes. The most common symptoms include the following:[2]

- pain with sitting, standing, and lying longer than 20 minutes.
- pain or paresthesia radiating from the sacrum through the gluteal region and down the posterior thigh.
- pain decrease with ambulation.
- pain when rising from a seated position.

It is important for physicians to elicit a complete history and to inquire about recent trauma and repetitive exercise, such as walking and jogging. During physical examinations, clinicians should assess patients for common clinical signs, paying extra attention to the lumbar spine, pelvis, sacrum, and leg length discrepancies. A neurologic exam including deep tendon reflexes, muscle testing, and sensation testing also will provide insightful information. Common clinical signs include tenderness in the sacroiliac joint, greater sciatic notch, and piriformis muscle; asymmetrical weakness in the affected limb; and restriction in range of motion. Specific tests for sciatica include the Freiburg, Pace, FAIR (flexion, abduction, internal rotation of hip), Beatty, and Lasegue tests.

Still, the diagnosis of piriformis syndrome remains controversial: Only 21 out of 29 surveyed physical medicine and rehabilitation specialists in the United States believe the condition exists.[1] A 10-year study published in 2002 by Fishman et al [9] aimed to

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definitively define piriformis syndrome by using electromyography (EMG). Boyajian-O'Neill et al[2] stated that EMG may assist in differentiating piriformis syndrome from intervertebral disk herniation. However, studies performed by Fishman et al[9] have been largely criticized by the scientific community for failing to meet standard diagnostic reporting criteria.[1] Hopayian et al[1] do not favor using EMG in clinical practice.

Although a clear diagnostic definition of piriformis syndrome remains to be established, studies published by Boyajian-O'Neill et al[2] and Hopayian et al[1] agree on the clinical presentation of piriformis syndrome as described earlier in this article.

For discogenic sciatic neuralgia, the initial intervention historically has been surgical removal of the offending herniated intervertebral disk. However, this approach often failed to provide symptomatic relief, and pressure on the nerve root has been positively correlated with loss of function rather than with pain.[4] Nerve root impingement is now understood to involve not only compression but also inflammatory and immune responses.

Anti-inflammatory agents, such as nonsteroidal anti-inflammatory drugs (NSAIDs), and myorelaxants can help to control pain and spasm. Ice and heat also help to temporarily relieve symptoms, and progressive stretching like routine OMT and physical therapy improves flexibility and increases strength.[5] Other nonpharmacological treatments include psychotherapy, cognitive behavioral therapy, and acupuncture.[4] Anticonvulsants, tricyclic antidepressants, serotonin-norepinephrine reuptake inhibitors, and opioids are the pharmacological agents used for radiculopathy. Seventy-nine percent of patients experience symptom reduction with initial conservative therapy.[2]

Boyajian-O'Neill et al[2] described an osteopathic approach to diagnosing and managing patients with piriformis syndrome. Osteopathic physicians should use OMT to address somatic dysfunction with indirect, direct, passive, and active modalities. Counterstrain and facilitated positional release are the favored indirect techniques for patients with piriformis-induced sciatica. In deciding which modalities to use, it is important for clinicians to keep in mind the following factors: Does the patient have primary or secondary piriformis involvement, acute inflammatory or chronic fibrosis, or functional or structural implications?[6] According to Fligg,[6] indirect maneuvers are favored over direct techniques. The author goes on to argue that stretching in the acute phase may increase the inflammatory response, thereby further entrapping the sciatic nerve. For chronic cases, either indirect or direct pressure techniques may be applied.

Maxwell[7] describes using trigger point therapy prior to adjustment in the acute phase. This can be accomplished by applying a constant heavy thumb, ultrasound, or muscle stimulator over the affected area for approximately 30 seconds or until the spasm is released. Applying 30 lbs of pressure (eg, a physician's elbow) over the affected area has been found to be the most effective method of release.[7]

If patients fail to respond to manipulation, researchers suggest following up with acupuncture or injection of trigger points with lidocaine hydrochloride, corticosteroids, or botulinum toxin type A.[2] Additionally, patients have been shown to benefit from therapeutic ultrasound. Those refractory to pharmacologic and nonpharmacologic therapy are candidates for surgical decompression.[2]

Lumbar and caudal epidural corticosteroid injections have become a mainstay in the treatment of patients for sciatica, although evidence is lacking in consistency regarding the number of injections, ideal volume, the content of the injectate, the need for fluoroscopy, the most effective route, and long-term benefits.[4] Computed tomography or magnetic resonance imaging can be used to more accurately guide clinicians with injections.[8] Studies regarding the efficacy of prolotherapy for patients with neuropathic pain remain to be published, but prolotherapy is being used experimentally, and there are already some reports of successful treatment.

Studies have shown that lumbar and caudal epidural corticosteroid injections are safe procedures with rare and temporary side effects.[4] Infection at the site of injection is the most common reported complication. Serious complications such as nerve root damage, epidural hematoma, and abscess are rare. Other systemic side effects include cushingoid symptoms such as obesity, hypertension, hirsutism, diabetes, immunosuppression, and changes in menses. Several cases of dural puncture have been reported, resulting in headache. Patients may experience a temporary increase in sciatic neuralgia, as well as syncope, stiff neck, flushing, urinary retention, hypotension, and vomiting. These effects typically do not require treatment.[4]

Conclusion

Primary care physicians and specialists have ample opportunity to change the lives of patients suffering from either acute or chronic sciatic neuralgia. While a concise diagnostic definition of piriformis syndrome remains unclear, osteopathic physicians are uniquely equipped to care for patients with low back, buttock, and leg pain.

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Nascent clinicians should be aware that although it is noted in this case study, sciatic neuralgia is not always a component of piriformis syndrome.

Clinicians can offer patients proven neuromuscular symptomatic reduction with conservative treatment, including OMT and pharmacologic therapy. And with corticosteroid epidural injections, physicians can offer minimally invasive treatment before resorting to surgery. This approach will reduce patients' healthcare costs and decrease their recovery time, complication rate, and time in physical rehabilitation, thereby improving the overall quality of life for those we serve.

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