

Piriformis Syndrome Mimics Sural Nerve Entrapment*

To the Editor:

Piriformis syndrome refers to both an acute and chronic condition wherein pain is elicited because of compression and irritation of the sciatic nerve by the piriformis muscle.¹⁻⁵ In most patients, the sciatic nerve runs beneath the piriformis, but in as much as 12% of the population, the sciatic nerve bifurcates the piriformis.^{3, 6, 7} In the second population, the likelihood for developing symptoms is believed to be increased.^{3, 4, 7}

The patient with a piriformis syndrome usually presents with a primary complaint of pain in the buttocks which may or may not radiate down the lateral aspect of the leg and into the foot.^{2, 4, 8} The pattern of referral may cover one or more dermatomes and be suggestive of a peripheral neuropathy.^{1-3, 8} This pain is usually aggravated with activity and positions that place the affected leg in a flexed or internally rotated position.^{2, 4, 7, 9} Although this is the typical presentation, occasionally patients will present with symptoms that are purely distal in nature.¹⁰ With careful questioning, they may report symptoms of a more proximal nature that have since resolved leaving purely distal symptoms remaining. The patient with piriformis-related symptoms is usually treated with a combination of stretching, exercise, and modalities to reduce pain and improve blood flow to the region such as ultrasound and electrical stimulation. Steroid injections may also prove effective.^{5, 9, 11}

This pattern of pure distal referral is important to the clinician since the patient's symptoms may mimic a peripheral nerve entrapment.^{1-3, 8} A careful history and physical examination may avoid this misdiagnosis and save both cost and pain for the patient.

Case Report

A 64-year-old male presented to the physical therapy department after being referred from the podiatry service with a diagnosis of sural nerve entrapment. The patient's primary complaint was of pain in his right lateral ankle and foot that had been present for almost 2 years. The pain was intermittent and would

appear without any apparent cause. The patient described his symptoms as "shooting" and "burning" over the lateral surface of the foot and ankle, although currently the symptoms were not severe.

The patient's medical history included Type II diabetes, hypertension, low-back pain, coronary artery disease, and a coronary artery angioplasty 10 months prior to presenting to the clinic. The patient reported that his ankle and foot pain had been treated by the podiatry service with injections and oral analgesics without success.

Examination of the lower leg was completely unremarkable, and the patient's symptoms were not reproducible with range of motion or palpation. A lower quarter screen was performed that included the lumbar spine and hip. It was unremarkable except for pain with palpation over the right piriformis muscle. Firm pressure on the piriformis did reproduce the patient's symptoms exactly as described, suggesting a diagnosis of piriformis syndrome.

The patient was treated with a combination of ultrasound and electrical stimulation to the right piriformis. This was performed with the patient side-lying on the unaffected side with the affected hip flexed and adducted (Fig. 1). He also received deep transverse friction massage and passive stretching to the piriformis, and was instructed in how to perform the stretch at home (Fig. 2). The patient reported complete relief of symptoms following treatment. The patient returned to clinic 1 week later and reported no recurrence of symptoms and compliance with his stretching program.

Discussion

The piriformis syndrome is a relatively common diagnosis in the clinical practice of physical therapy and as such is actively looked for when patients present with complaints of lower extremity pain without any history of trauma or overuse. Patients with this diagnosis are probably not so frequently seen in a podiatric medical setting and may be misdiagnosed in the search for a causal factor for the patient's complaints that is confined to the lower extremity. Although the podiatric physician may not see many patients with a piriformis syndrome, the underlying cause of this disorder, in many cases, is a lower extremity strength imbalance or biomechanical abnormality that results in overworking of the piriformis.²⁻⁵ The author has treated many piriformis syndrome patients successfully using the techniques described above. However,

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Figure 1. Treatment position for combination ultrasound and electrical stimulation. The patient is side-lying on the unaffected side with the affected hip flexed and adducted.



Figure 2. Self-stretching position for piriformis syndrome. The patient is instructed to lie supine, flex the hip, and draw the knee across the body until a stretch is felt in the hip.

in several cases, there was an underlying lower extremity abnormality that required referral to a podiatric physician for orthotic devices or other intervention to normalize the biomechanics of the lower extremity. After the underlying mechanical cause of the patient's complaints was addressed, the author was able to effect a rapid resolution of symptoms.

The case discussed shows the importance of taking a thorough history and physical examination that references not just the lower leg but the entire lower quarter. This may improve the likelihood of not just treating the patient's symptoms but locating the underlying cause. In today's challenging health care market, the ability to quickly diagnose and treat the underlying cause of a patient's complaints is important in keeping costs down. The patient required several clinic visits, two injections, and six plain film x-rays before he was successfully diagnosed and treated. Successful early diagnosis of this patient would

have saved considerable time and money for both the patient and the medical center. This case also shows the potential benefits of using other allied health services, such as physical therapy, in the treatment of the podiatric patient.

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Periosteal Proliferation of the Fibula in Charcot Foot*

To the Editor:

Charcot foot in the diabetic patient is a progressive, chronic, destructive form of joint degeneration seen in patients with underlying neurologic deficiency. Clinically, Charcot foot presents with flatfoot deformity, forefoot valgus, swelling, and erythema of the foot. Radiographically, neuroarthropathic

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changes are seen most commonly in the midfoot (tarsal and tarsometatarsal joints); however, the forefoot (metatarsophalangeal joints) and rearfoot (tibiotalar and subtalar joints) can also be involved in Charcot foot. Repetitive stress applied to the neuro-pathic bones and joints is believed to be the mechanism involved in the destruction, which results in fractures, edema, and ligament laxity, followed by fragmentation, luxation, and collapse of the foot.¹

The three stages of Charcot neuroarthropathy as described by Eichenholtz consist of development, coalescence, and reconstruction.^{1,2} The development stage begins as joint effusion, soft tissue edema, subluxation, and debris formation of bone and cartilage. The stage of coalescence is the initiation of the repair, which presents as a reduction of edema, absorption of the fine debris, and healing of fractures. The stage of reconstruction is described as the final-bone-healing phase that includes continuation of repair and remodeling of bone.

Radiographic presentation of Charcot foot can be described as either atrophic or hypertrophic. The atrophic type consists of predominant osteolysis resulting in a pencil-like appearance of the distal aspects of the metatarsals and phalanges.^{1,2} Conversely, the hypertrophic type is manifested by periosteal new bone formation, sclerosis, and osteophytosis.^{2,4}

In reviewing the literature, the authors did not find a case of Charcot foot associated with periosteal proliferation of the fibula. They describe such a presentation.

Case Report

A 54-year-old male was referred to the podiatric medical clinic from the primary care clinic complaining of a swollen left leg and ankle for more than 1 year (Fig. 1). Previous treatment included a pressure wrap applied three times a week for 2 months, support stocking, and leg elevation.

The patient's medical history was significant for noninsulin-dependent diabetes mellitus of approximately 17 years, chronic renal insufficiency, hypertension, gout, anemia, diabetic retinopathy, and neuropathic osteoarthropathy. His medications included 10 ml Pred-Forte^{®1} ophthalmic suspension, 5 ml of gentamicin ophthalmic solution, OsCal-500^{®2} tablet, 8 mg of doxazosin, 80 mg of furosemide, and Aquaphor^{®3} healing ointment. The diabetes is currently diet-controlled. He has no known drug allergies.

^{®1} Alleagau, Irvine, CA.

^{®2} Smith Kline Beecham, Pittsburgh, PA.

^{®3} Beirschomb, Norwalk, CT.

During the physical examination of the left lower extremity, an increase in temperature around the midfoot area was noted. Pedal pulses were nonpalpable secondary to the presence of nonpitting edema. His skin was intact but shiny and atrophic. Neurologic sensation was diminished and the patient did not complain of any pain. A pes planus deformity was present. Radiographs of the left foot and ankle were ordered and a vascular consultation was requested. Findings from the vascular studies were within normal limits, which concluded that the etiology of the patient's enlarged ankle did not appear to be of vascular origin.

Comparison of radiographs obtained on October 4, 1994, with radiographs from September 11, 1995, showed noticeable progressive changes consisting of extensive periosteal new bone formation involving the posterior distal fibula shaft (Figs. 2 and 3). The musculoskeletal radiologist's impression of this radiographic feature was consistent with a post-traumatic etiology secondary to chronic neurotrophic arthropathy. The radiographs also revealed complete loss of the normal talus architecture with flattening and collapsing into the calcaneus, leading to distortion and flattening of the calcaneus as well. Complete loss of the talocalcaneal joint was associated with marked sclerosis and numerous bone fragments located anterior to the distal tibia.

To rule out the possibility of an underlying neoplasm or infection, laboratory tests, chest x-ray, and a technetium-99m HMPAO were also performed, as well as consultation by the oncology service. The laboratory results revealed an increase in uric acid, chloride, blood glucose, blood urea nitrogen, and creatinine levels, and a decrease in red blood cells,



Figure 1. Clinical presentation during the initial visit.

hemoglobin, and hematocrit. The chest x-ray was normal. The radiologist believed that the technetium-99m-HMPAO findings were also consistent with post-traumatic change. Although there is an increase in uptake noted in the bone on the 24-hr delay images, the relative uptake is not increased to an extent that would imply osteomyelitis. The physician from oncology service concurred with the radiologist's impression of exuberant periosteal new bone formation related to chronic repetitive trauma in a patient with known neurotrophic arthropathy.

A walker was dispensed to the patient, which he currently wears along with his support stockings



Figure 2. Lateral radiograph taken approximately 11 months prior to the initial visit. Note the small bone fragments located at the anterior aspect of the ankle joint.



Figure 3. Radiograph obtained during the initial visit. Note the periosteal proliferation of the distal fibula. Extensive deterioration of the talus and calcaneus is also recognized.

(Fig. 4). After approximately 4 weeks, a decrease in swelling of the left foot, ankle, and leg was recognized. The patient was later measured for a pair of custom-molded high-top shoes along with a pair of inserts. When the patient returned for a follow-up visit, further decrease in edema to the leg was observed and new x-rays were taken that revealed that there were no significant interval changes (Fig. 5). The patient also reports an increase in ambulation after using the walker for approximately 3 months.

Discussion

The spontaneous fracture of bones at the attachment sites of the ligaments results in periosteal proliferation of bone than can be seen along the shaft of tubular bones.^{2,3} This periosteal appearance commonly occurs at the midfoot region, especially at the lesser metatarsal bones.² However, this particular case showed periosteal proliferation of the distal fibula near the ankle joint. The periosteal formation may be caused by the pressure wrap applied to the affected limb; however, serial radiographs were not taken during this period to indicate such a relationship.

Some authors have suggested that the primary goal of treating Charcot foot is to prevent additional trauma to the affected limb.^{4,9} This goal can be ideally achieved with complete nonweightbearing to the affected limb. In this case report, the patient used a walker that immobilized and stabilized the involved joints.⁴ This also allowed the patient to walk freely



Figure 4. Anterior view of the left lower limb in the walker.



Figure 5. A lateral view of the left limb after 3 months' use of the walker.

without the use of crutches, which tend to be cumbersome. The long-term management for this patient includes prescription of custom-molded shoes to prevent any development of ulcers and to support the weakened osseous structures. Follow-up radiographs will also be obtained.

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Catfish Spine Injury of the Foot*

To the Editor:

Penetrating wounds from the spines of marine catfish can often result in a retained foreign-body reaction to the surrounding area. This can occur with or without the knowledge of the individual.¹ The resultant soft tissue granuloma can be treated with simple removal of the foreign body and proper treatment of the site. A number of complications have been reported with catfish stings including: severe localized inflammation caused by the toxins released, a foreign-body reaction, infection, tissue necrosis, amputation, and even death.^{2,3}

Catfish contain hazardous dorsal and pectoral fins, each of which contains a cartilaginous barbed spine along with the leading edge that attaches to venom glands.² The venom itself has also been shown to be dermatonecrotic and locally vasoconstrictive.⁴ A second toxin released has also been identified from the epidermal cells on the skin of the fish: the toxin causes intense throbbing pain, possible muscle fasciculations, and tissue necrosis, all of which are somewhat heat labile.³ The overall structure of the spine creates more extensive tissue injury

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than a simple penetration injury when the spines become embedded.

The diversity of the habitat of the catfish also contributes to the possible complications of the injury. Catfish are native to fresh and salt water environments, which increases the amount of water-borne bacterial organisms such as *Aeromonas* or *Vibrio*. Other possible sources of infection include *Erysipelothrix*, *Norcardia*, *Chromobacterium*, *Sporothrix*, or *Actinomyces*.²

There are numerous treatment options and recommendations cited in the literature relating to fish-spine injuries. Many state that established principles of local wound care management should be followed. These include irrigation, debridement, topical antibiotics, and systemic antibiotics if required. Relief of pain is accomplished by immediate immersion of the involved extremity in water that is as hot as tolerated (up to 45° C or 120° F) for 30 min to several hours as needed.⁵ Hot water appears to denature the toxins within the wound.⁶ Local anesthetics (narcotic or otherwise) have been less effective for pain management.^{5,6} The wound should be explored surgically and radiographically for any foreign material that should be removed. Prophylactic antibiotic therapy and tetanus immunization status should be ascertained. The site should then be allowed to heal by secondary intention.^{5,6}

Case Report

A 30-year-old male presented to the podiatry clinic with a chief complaint of pain in his right foot. Three months prior, the patient caught a large catfish in the Gulf of Mexico. After removing the hook, the catfish fell on his right foot. The spine from the pectoral fin apparently penetrated his shoe and was retained in the soft tissue around the first metatarsophalangeal joint unknowingly for 3 months. Two days prior to his visit, the patient stated that he was soaking his right foot and noticed a hard, white object jutting out from his skin. With a pair of tweezers, the patient removed a foreign body from his right foot. The patient's medical history was unremarkable.

On clinical examination, the foreign body measured approximately 1.4 cm and was consistent with a catfish spine. The spine was hard, opaque, and presented with a very sharp serrated edge (Fig. 1). The dorsomedial aspect of the first metatarsophalangeal joint also presented with an elevated area of granulation tissue with a discharge of serosanguineous fluid (Fig. 2). A small sinus tract was noted that probed into the soft tissue adjacent to the joint. There was little erythema and no signs of infection. The radio-

graphs were within normal limits. Culture and sensitivity were also taken and revealed no growth after 48 hr. A tetanus booster was given.

The wound was then copiously lavaged and irrigated with a mixture of povidone iodine, hydrogen peroxide, and normal saline. Antibiotic ointment was applied and the site was dressed with a sterile dressing. A postoperative shoe was also dispensed.

The patient returned in 1 week and the wound was probed as before, but only to approximately 1 cm. The focal area of reactive tissue had markedly reduced. The patient had no complaints and at 3 weeks, the wound was completely healed.

Conclusion

Marine envenomation injuries can be devastating and at times lethal. This case presentation, however, was uneventful and demonstrated complications associated with aquatic life. The treatment regimen was consistent with the literature and 1 year following the injury, the patient presented without complications or recurrences.



Figure 1. A catfish spine measuring approximately 1.4 cm removed from the right foot.



Figure 2. An elevated, reactive area of granulation tissue secondary from the catfish spine. Note the serosanguineous fluid exuding from the penetrating sinus tract.

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Bilateral Interosseous Lipoma of the Calcaneus*

To the Editor:

Intraosseous lipoma is a benign tumor of fatty tissue contained in bone. The first report of such a tumor was made by Cornill and Ranvier in 1880.² In 1955, Child³ was the first to describe this lesion in the os calcis.⁴⁻¹² Only one reported case of bilateral calcaneal intraosseous lipoma has been described in previous literature.

The incidence of bone lipoma is less than 1:1,000 as described by Dahlin¹³ at the Mayo Clinic. The incidence may be higher, since many of these lesions are asymptomatic. There is no prevalence between genders, and the age range varies from 5½ to 85 years old. The tumor appears in axial and appendicular skeleton with more than 50% of reported tumors involving long bones.^{14, 15} The lower extremity is more commonly involved than the upper extremity, with the metaphyseal region being most frequently affected.

The authors report a case study of a bilateral intracalcaneus lipoma. Etiology, diagnostic methods,

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pathophysiology, differential diagnosis, and treatment will be discussed.

Case 1

A 31-year-old male presented with a chief complaint of pain and swelling in the right heel after kicking a stair tread while working as a carpenter. His medical history was unremarkable. Physical examination revealed pain on palpation of the lateral aspect of his right calcaneus. Plain radiographs revealed a 2.8-cm lytic lesion in the anterior aspect of the right calcaneus (Fig. 1). The left calcaneus was also studied and revealed a similar lesion that was asymptomatic. A computed tomography scan was performed and a presumptive diagnosis was made based on the attenuation numbers that fell within the range of lipomatous tissue (Fig. 2). A magnetic resonance imaging examination was also performed and revealed a 2.8-cm lesion with a signal intensity analogous to fat on T1 and T2 images (Fig. 3). Laboratory evaluations preoperatively were all within normal limits.

Under general anesthesia and tourniquet hemostasis, an incision was made from the lateral malleolus to the base of the fifth metatarsal. The incision was carried down to the calcaneus. A window was created in the calcaneus, and the cavity was curetted. The tissue from the cavity was cultured and its contents sent for pathologic evaluation. The tissue removed from the cyst appeared to be yellow adipose-like tissue. Iliac crest bone graft was then used to pack the cavity and the window of cortical bone was replaced. The incision was closed in layers, and a dry sterile dressing and a short leg cast were applied. The microscopic appearance on pathologic examination showed adipose tissue interspersed with osseous trabeculation, which is consistent with the diagnosis of intraosseous lipoma (Fig. 4). The culture and Gram's stain revealed no organisms. The patient was followed until 1 year after grafting and remained asymptomatic (Fig. 5).

Discussion

Lipoma is the most common benign tumor of connective tissue.¹⁶ Lipomas within bone are a rare neoplasm. The etiology of this tumor is unknown. The lesion has been described as traumatic in origin with fractures leading to fat degeneration.¹⁷⁻¹⁹ Also, it has been described as secondary to a bone infarct with subsequent fat metaplasia.^{20, 21} The most common belief is that the lesion is a true primary bone tumor^{4, 13-15, 22}

Pain is usually the most common symptom, although swelling associated with pain occurs in some instances. Many patients have asymptomatic lesions



Figure 1. Lateral radiograph shows a cystic lesion in the calcaneus.

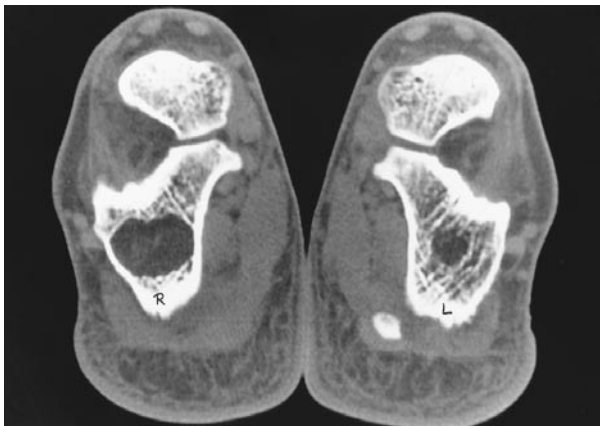


Figure 2. Computed axial tomography shows a lytic lesion in the right calcaneus that is approximately 2 cm. The left calcaneus has a similar lesion that is approximately 4 mm. The lesions are well circumscribed with a partially sclerotic rim.

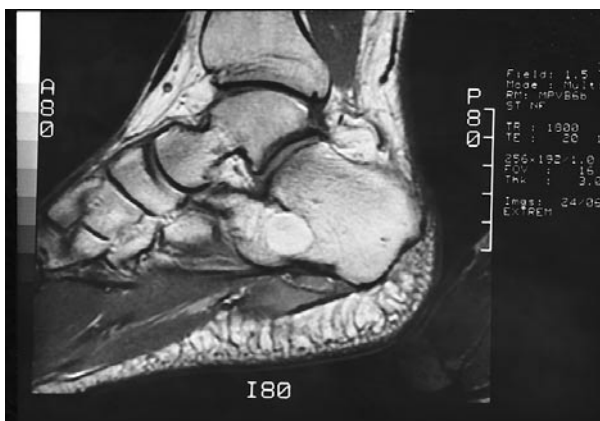


Figure 3. Magnetic resonance imaging scan of the right rearfoot in the sagittal plane. This lesion is a well circumscribed osteolytic lesion in the anterior aspect of the calcaneus with signal intensity identical to fat, 2.8 cm in diameter.

that are usually incidental radiographic findings.

Radiographically, bone lipomas are seen as radiolucent lesions with well defined edges, occasionally having central calcification.^{9, 21} Infrequently, a sclerotic border can be noted, but cortical erosions or periosteal reactions are usually not found.

Computed tomography has been used for definitive diagnosis of intraosseous lipomas.^{10, 11} Computed tomography attenuation measurements corresponding to fat are used to make the diagnosis.

Magnetic resonance imaging was also used in the authors' case reports, which showed a T1 signal intensity identical to fat and compatible with lipoma.

Microscopically, intraosseous lipoma specimens consist of lobules of mature adipose tissue separated by thin septa containing capillary channels interspaced by osseous trabeculae. Occasionally, central necrosis within adipose tissue is noted along with reactive bone formation or fat calcification.

Different diagnosis for a lytic bone lesion can be

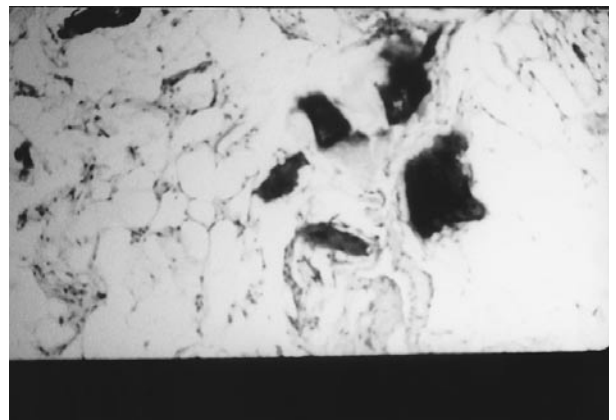


Figure 4. Photomicrograph of intraosseous lipoma showing adipose tissue lobules separated by trabeculae of bone (H & E $\times 40$).



Figure 5. Postoperative radiograph.

Table 1. Differential Diagnosis of Intraosseous Lipoma^a

Collagen disorders
Pseudocyst of rheumatoid arthritis
Infectious conditions
Chronic tuberculosis
Metabolic conditions
Localized osteoporosis
Hyperlipoproteinemia
Malignant tumors
Chondrosarcoma
Liposarcoma
Osteosarcoma
Metastatic disease
Benign tumors
Simple bone cyst
Aneurysmal bone cyst
Chondromyxoid fibroma
Benign giant cell tumor
Fibrous dysplasia
Osteoblastoma
Chondroblastoma
Pigmented synovitis
Intraosseous lipoma

^aFrom Leeson MC, Kay D, Smith BS: Intraosseous lipoma. Clin Orthop 181: 186, 1983. Reprinted with permission.

extensive (Table 1).²³ Physical, radiographic, and other special studies should narrow the differential diagnosis.

The most common treatment for this lesion is curettage-graft surgery. Treatment is rendered for symptomatic lesions and for lesions in weightbearing bones. After surgery, no recurrences have been reported and prognosis is good.

Conclusion

Although intraosseous lipomas have been reported in the literature, only once has it been reported bilaterally. Often, these lesions will remain asymptomatic unless some insult leads to them become painful or they are an incidental finding. Plain radiographs, computed tomography, and magnetic resonance imaging scans are used for diagnosis. Surgical curettage with packing of either allogenic or autogenous bone grafts proved to have excellent results.

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