Isolated Sciatic Nerve Entrapment by Ectopic Bone After Femoral Head Fracture-Dislocation

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Abstract

Although posttraumatic pelvic heterotopic ossification (HO) after hip fracture dislocation is well established, and nerve encasement by HO may occur, the development of neurologic deficit is rare. A thorough history and adequate clinical suspicion are imperative in the workup of affected patients. Computed tomography and magnetic resonance imaging provide good visualization and assist in surgical planning. If symptoms persist and are recalcitrant to conservative management, surgical intervention with HO excision and nerve neurolysis can be performed with success.

Traumatic hip dislocation and fracture-dislocation are not uncommon, and their incidence is increasing.[1] Despite the relative strength and stability of the hip joints, dislocations occur in 62% to 93% of motor vehicular collisions and constitute 5% of all traumatic joint dislocations.[2] These dislocations are more often posterior than anterior. In the setting of a native hip dislocation, urgent reduction by a skilled surgeon is critical.[1] Failure to perform urgent reduction can result in posttraumatic avascular necrosis (up to 33%), as the femoral head is depleted of its blood supply, and nerve palsy (10%-15%), caused by prolonged nerve traction.[1] Other complications are posttraumatic coxarthrosis (24%-88%), instability, heterotopic ossification (HO), and decreased range of motion.[1,2] Each of these can lead to a significant decrease in function and quality of life.

HO is the formation of mature lamellar bone in soft-tissue sites outside the skeleton, and is a known complication of hip dislocation with or without associated fracture.[1-5] It is thought to originate from osteoprogenitor stem cells lying dormant within the affected soft tissues.[6] With the proper stimulus, the stem cells differentiate into osteoblasts and begin the process of osteoid formation, eventually leading to mature heterotopic bone.[6,7] Although HO can occur in the absence of trauma,[8-12] it often follows traumatic events such as burns, arthroplasties, complex fractures, and spinal cord and brain injuries.[12] Other risk factors are male sex, high Injury Severity Score, delay in fracture fixation, ankylosing spondylitis, persistence of bone debris, postoperative hematoma, and surgical approach.[3]

In hip dislocation, the sciatic nerve is often injured (10%-15% of cases).[3] The nerve can be injured either at time of dislocation or after reduction.[4] In hip fracture-dislocation, the incidence of posttraumatic injury is as high as 30%, and that of perioperative or postoperative injury is 5% to 15%.[4] The sciatic nerve can become entrapped by tendon, muscle, hemorrhagic tissue, scar tissue, and heterotopic bone.[13]

In this article, we report the rare and anomalous case of a patient who developed sciatic nerve entrapment, from the sciatic notch to the midfemur, by HO in isolation from surrounding tissue after posterior femoral head fracture-dislocation.

The patient provided written informed consent for print and electronic publication of this case report.

Case Report

An unrestrained passenger was injured in a motor vehicle collision. After presenting to a hospital, the 29-year-old man was taken to a level II trauma center. On arrival, he had a Glasgow Coma Scale score of 15. Radiographs showed a posterior hip dislocation and a Pipkin Type I femoral head fracture. Initial closed reduction was unsuccessful, so the patient was taken to the operating room for another attempt. However, the hip joint proved irreducible by closed means, even under fluoroscopic guidance.

Open reduction was performed. The patient was placed in the lateral decubitus position and a posterior approach to the hip was used. Disruption of the superior and posterior capsule was noted. A large, minimally articulating piece of the femoral head, found loose in the joint, impeded reduction. The sciatic nerve was grossly contused and indented where the femoral head was exerting pressure. The sheath, however, appeared grossly intact. The loose body was removed and the hip reduced. After surgery, the patient was placed in an abduction pillow but did not receive HO prophylaxis. There was weakness 4/5 on the American Spinal Injury Association scale in the extensor hallucis longus and tibialis anterior muscles. On discharge, the patient

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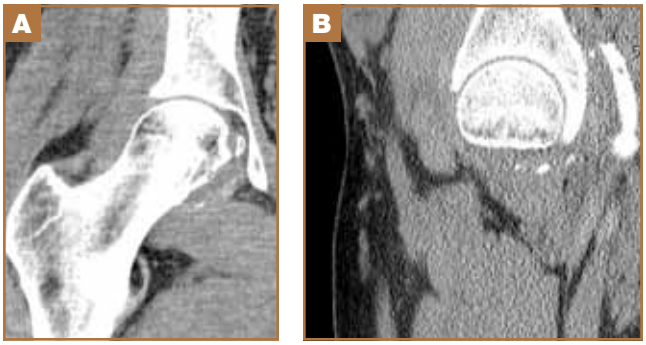

Figure 1. (A) Coronal computed tomography shows Pipkin Type I femoral head fracture. (B) Sagittal computed tomography shows sciatic nerve entrapped in heterotopic ossification along its course.

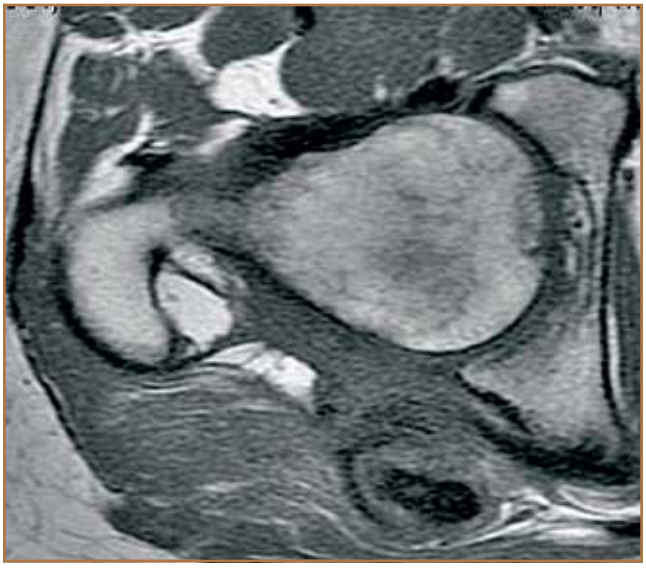

Figure 2. Axial magnetic resonance imaging shows sciatic nerve encased in heterotopic ossification at level of femoral head. There is no evidence of avascular necrosis in femoral head.

Figure 3. Sciatic nerve entrapped in ectopic bone observed during surgery.

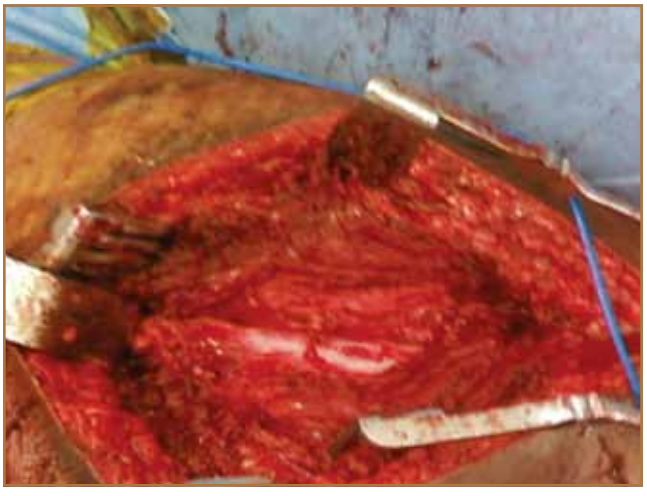

Figure 4. Released sciatic nerve after excision of heterotopic ossification observed during surgery.

was allowed weight-bearing as tolerated.

Over the next 5 months, the patient noted increasing right hip pain. The neurologic status of the right lower extremity subsequently declined to the point that a footdrop developed and an ankle-foot orthosis was required. Computed tomography (CT) showed evidence of prior femoral head fracture and fragmentation. In addition, there was significant HO coursing along the sciatic nerve (**Figures 1A, 1B**). Magnetic resonance image (MRI) showed significant HO about the entire course of the sciatic nerve as it exits the foramen. There was no evidence of avascular necrosis (**Figure 2**). Both CT and MRI showed that the surrounding joint region, outside the sciatic nerve, was devoid of HO.

The patient presented to our clinic 10 months after injury. He reported radicular and lancinating pain down the leg. The pain caused an antalgic gait and the footdrop a steppage gait. The patient denied any groin pain with internal rotation of the hip. There was no extensor hallucis longus or tibialis anterior muscle function. The gastroc-soleus complex had 3/5 strength with numbness below the level of the knee in the tibial and peroneal nerve distributions.

Given the progression of neurologic decline and the radiographic evidence of ossific entrapment of the sciatic nerve, we performed HO excision and sciatic nerve decompression. No electromyogram (EMG) was performed, as the clinical examination findings matched the radiographic abnormality, and EMG findings were unlikely to alter management.

A posterior approach was used to expose the hip joint. Minimal HO was encountered during the exposure. On deeper dissection, the sciatic nerve was found encased in HO from the sciatic notch to midfemur (**Figure 3**) in isolation from surrounding structures. The nerve was meticulously decompressed with use of kerrisons and burring techniques (**Figure 4**).

After surgery, a 600-cGy dose of localized radiation was administered to the hip for HO prophylaxis. The patient con-

tinued wearing the ankle-foot orthosis and was discharged 3 days after surgery. A 6-week course of indomethacin was prescribed for additional HO prophylaxis.

At latest follow-up, 4 months after surgery, the patient reported improvement in motor function. Specifically, gastrocsoleus complex strength had improved to 5/5, and he was able to move the extensor hallucis longus and tibialis anterior muscles (2/5). Sensory examination findings also showed improvement, most notably in the tibial nerve division.

Discussion

The hip joint is the site that most commonly develops HO after trauma or neurologic injury.[14] Rarely, the heterotopic bone becomes a compressive lesion causing nerve deficits. Our patient's HO involved only the sciatic nerve; the surrounding tissue was spared. Although the etiology of ectopic bone is poorly understood, the complication may result from surgical trauma.[4] Other factors that may exacerbate HO are traumatic brain injury, soft-tissue damage after initial trauma, extended surgical approaches,[15] and failure to administer postoperative radiation and/or medications such as indomethacin as prophylaxis against HO.[4]

Sciatic nerve injuries after hip dislocation have numerous etiologies and occur in about 10% to 15% of cases. In sciatic nerve injury after hip trauma, the peroneal division is usually involved, with resultant weakness in the extensor hallucis longus, tibialis anterior, peroneus longus, and peroneus brevis muscles.[16] It has been suggested that the tethering of the nerve at the sciatic notch and the fibular neck may restrict its excursion, compared with the tibial division, which is tethered only at the notch.[4,16] Our patient presented with nerve palsy in the tibial and peroneal divisions, indicating presence of significant chronic nerve compression. We theorize that his nerve palsy resulted from very significant nerve compression and contusion, possibly exacerbated by attempted forceful reduction, as evidenced by the gross damage found in the sciatic nerve during surgery. We speculate that additional microscopic endoneurial and fascicular damage may have occurred during and after a forced reduction.

There are few reports of peripheral nerve entrapment by HO.[17-19] We identified 11 cases of sciatic nerve entrapment by HO after management of hip fracture-dislocation.[2,13,20-27] Of the 11 patients, 4 recovered all their sensory and motor function and the other 7 had partial recovery. In only 6 cases was sciatic nerve isolation reported.[3,21-24,27] In these 6 of 11 cases, however, the amount of soft-tissue ectopic bone and the distal extent of the nerve entrapment were unclear. In our patient's case, HO involved the sciatic nerve, from the notch to the midfemur, in isolation from surrounding structures.

Sciatic nerve decompression may have better outcomes for sensory deficits than for motor deficits.[4] Our patient noted full motor function improvement in the tibial nerve division but only partial improvement in the peroneal nerve division. Compared with the tibial nerve, the peroneal nerve lies more lateral; is more tethered given its dual fixation and thus is under more stretch; has a less robust vascular supply; and has less protective force-absorbing connective tissue.[4,16,28] As a result, it was no surprise that our patient had more significant improvement in tibial nerve function than in peroneal nerve function. At latest follow-up—only 4 months after surgery—motor and sensory function was improved but still diminished.

While neurologic signs and symptoms suggest specific nerve division involvement, radiographic imaging modalities assist in HO staging and surgical planning. Amount of HO can be graded on radiographs using the system of Brooker and colleagues,[29] which relies solely on anteroposterior pelvic views. A modified classification using additional Judet views can provide a more accurate correlation with hip range of motion. CT is also of value in staging and preoperative planning.[3] Other imaging methods (eg, MRI) offer additional information, including amount of inflammation[5] and confounding disease processes, such as avascular necrosis. We used radiography, CT, and MRI in surgical planning and in determining extent of nerve involvement.

HO recurrence after excision and prophylaxis for sciatic nerve palsy in the setting of trauma has not been extensively studied. In other areas of hip surgery, however, indomethacin, bisphosphonates, and irradiation have all been successful in preventing recurrence.[3] Radiation administered 24 hours before surgery or within 72 hours after surgery has been effective in preventing pluripotent mesenchymal stem cell differentiation into osteoprogenitor cells.[7] Caution should be exercised when irradiating patients with femoral head fractures and/or dislocations because of the already increased risks for avascular necrosis and nonunion. In addition, though indomethacin reduces the rate of HO after surgical management of acetabular fractures by 30% to 45%, it can cause bleeding and have gastrointestinal complications.[7] Research is needed to clarify which form of perioperative prophylaxis is best.

We can only hypothesize the cause of our patient's presentation. It might be explained by neuropraxia, which may have occurred secondary to nerve traction during hip dislocation and/or reduction. da Paz and colleagues[30] theorized that sudden shearing can damage sensory and autonomic nerve fibers, leading to changes in humoral, neural, and local factors. Such injuries can alter vascular and metabolic changes, may combine with musculotendinous apparatus microtrauma to induce ossification through an inflammatory response or directly through release of osteoblast-stimulating factors.[30] Our patient's sciatic nerve was contused macroscopically, but there was no apparent injury to the nerve sheath. However, the nerve was not carefully explored under magnification, which may have revealed more significant injury. It is possible that HO prophylaxis can be helpful in mitigating this biochemical cascade.[31,32] Our patient did not receive HO prophylaxis after initial injury.

In light of this complication, we recommend a few precautions for posterior open reductions: careful exploration of the sciatic nerve to identify potentially significant injuries, postoperative use of radiation or indomethacin as prophylaxis, and heightened suspicion in cases involving progressive postoperative neurologic deficit. A workup with EMG is recommended if the clinical appearance is unclear, and further imaging with

CT is useful in identifying the presence and significance of HO if the patient also notes pain and loss of motion.

Conclusion

Our patient's presentation of isolated sciatic nerve encasement by HO and progressive neurologic deficit is rare and highlights the variable manifestations of HO after trauma and the need for further research to understand HO pathogenesis and the natural history of nerve recovery after crush injury. It is important for treating surgeons to note the presence or absence of nerve injury at time of hip dislocation, as it may dictate the patients course in terms of possible subsequent sciatic nerve complications. In addition, surgeons should consider nerve entrapment by HO in a patient who has developed progressive nerve deficits after significant hip trauma. We recommend careful preoperative imaging and planning, and managing refractor y sciatic nerve entrapment by HO with surgical excision and with concomitant use of irradiation and indomethacin, unless contraindicated.

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