Sciatic Nerve Injury Associated with Acetabular Fractures

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Abstract Sciatic nerve injuries associated with acetabular fractures may be a result of the initial trauma or injury at the time of surgical reconstruction. Patients may present with a broad range of symptoms ranging from radiculopathy to foot drop. There are several posttraumatic, perioperative, and postoperative causes for sciatic nerve palsy including fracture–dislocation of the hip joint, excessive tension or inappropriate placement of retractors, instrument- or implant-related complications, heterotopic ossification, hematoma, and scarring. Natural history studies suggest that nerve recovery depends on several factors. Prevention requires attention to intraoperative limb positioning, retractor placement, and instrumentation. Somatosensory evoked potentials and spontaneous electromyography may help minimize iatrogenic nerve injury. Heterotopic ossification prophylaxis can help reduce delayed sciatic nerve entrapment. Reports on sciatic nerve decompression are not uniformly consistent but appear to have better outcomes for sensory than motor neuropathy.

Keywords sciatic nerve palsy · acetabular fracture · hip dislocation · heterotopic ossification

Sciatic nerve palsy associated with acetabular fractures may result from (1) damage at the time of trauma, (2) iatrogenic injury during reconstructive surgery, or (3) a late complication of surgery. Posttraumatic causes of sciatic nerve palsy include fracture–dislocation of the hip joint. Iatrogenic causes include intraoperative positioning and placement of retractors, instruments, or implants. Late complications resulting in sciatic nerve injury include wear debris, implant

migration, hematoma, capsular or muscular scarring, and heterotopic ossification resulting from the use of extensile approaches. The natural history of sciatic nerve injuries is likely dependent on several factors including the anatomic location of the injury, severity of the injury, chronicity of the injury, patient comorbidities, and age. A clear history should be obtained identifying the temporal relationship between onset of symptoms and acetabular trauma or reconstructive surgery and possible preexisting lumbosacral degenerative disease. Patients may present with a broad range of sensory and motor symptoms. Specific imaging studies, including plain radiographs, computed tomography (CT), and magnetic resonance imaging (MRI), and electromyographic studies can help localize the anatomic location of nerve injury.

Preventive measures include avoiding iatrogenic stretch injury to the nerve by keeping the hip extended and knee flexed during surgery. Retractors should be carefully positioned and extensive posterior retraction with the hip in flexion should be avoided. Instrument or implant placement should be performed safely with the assistance of intraoperative fluoroscopy if necessary. Somatosensory evoked potentials (SSEP) and spontaneous electromyography (EMG) can alert the surgeon to potential nerve injury. Heterotopic ossification prophylaxis is useful for preventing progressive sciatic nerve entrapment. Like the natural history of sciatic nerve injuries, results of surgical decompression appear to depend on several preoperative factors and appear to have better outcomes for sensory than motor palsy.

Anatomy

The sciatic nerve is the largest branch of the sacral plexus, formed by the union of the ventral rami of the fourth and fifth lumbar and first, second, and third sacral roots. There are also contributions from the fourth sacral nerve root. In 79% of specimens studied, the sciatic nerve exits the pelvis from below the piriformis muscle [1, 2]. In 14.3% of the specimens studied, the nerve separates into two divisions with one division passing through the muscle and the other passing below the muscle. In 4.4% of the specimens

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studied, the nerve separates into two divisions with one division passing above the piriformis and the other passing below the piriformis. In 2.2% of the specimens studied, an unsplit nerve passes through the piriformis muscle [2]. Once distal to the piriformis muscle, the sciatic nerve courses down the posterior aspect of the thigh supplying the hamstrings. It then divides into the common peroneal nerve and the tibial nerve usually just proximal to the knee joint. The common peroneal nerve courses around the fibular neck and divides into superficial and deep branches to supply the musculature of the lateral and anterior compartments of the leg, respectively. The tibial nerve runs down the posterior aspect of the leg and supplies the musculature of the superficial and deep posterior compartments of the leg and the plantar region of the foot [1].

The inconsistent relationship of the nerve to the piriformis muscle has been postulated to be a possible cause of sciatic pain. A tight piriformis muscle may compress the sciatic nerve in this region causing radicular pain along its course [1, 3, 4]. In most cases of sciatic nerve injury, the peroneal division of the sciatic nerve is usually affected with resultant weakness of the tibialis anterior, extensor hallucis longus, peroneus longus, and peroneus brevis [5, 6]. While it is not completely understood why the peroneal division may be more susceptible than the tibial division to damage, it has been postulated that the tethering of the nerve at the fibular neck and the sciatic notch may restrict its excursion compared to the tibial division which is only tethered at the sciatic notch [5, 7]. In addition, the peroneal division has fewer nerve bundles than the tibial division which are larger in diameter and separated by less connective tissue. Thus, compared to the structure of the tibial nerve, the connective tissue surrounding the peroneal nerve may not as effectively cushion the nerve from injury. Furthermore, the nerve bundles of the peroneal division are larger than the tibial division, making them more vulnerable to ischemic injury as the vasa nervorum has a larger diameter of fibers to supply [5, 7].

Etiology

Sciatic nerve injuries may occur as a result of the initial acetabular trauma (posttraumatic), as a result of iatrogenic trauma during surgery (perioperative), or as a later complication of surgery (postoperative). The prevalence of posttraumatic sciatic nerve injury has been reported to be as high as 30% [7–11]. The prevalence of perioperative or postoperative sciatic injuries has been reported to be approximately 5% to 15% [8, 11–14].

Letournel and Judet noted that the highest incidence of sciatic nerve palsy was in association with a posterior fracture–dislocation of the hip joint [11, 13]. In their series, Helfet and Schmeling noted that all patients with a posttraumatic sciatic nerve injury had fracture patterns including the posterior wall or posterior column [11]. The impact of these injuries may result in blunt contusion to the sciatic nerve, laceration, or stretching of the nerve over the dislocated femoral head [6, 7, 11, 13, 15, 16]. Iatrogenic injury can result from several factors. In posterior approaches, excessive retraction of the posterior gluteal muscle mass with the hip in flexion or

continuous extension of the ipsilateral knee can result in a stretch injury to the sciatic nerve [11–13]. Placement of retractors or reduction clamps in the lesser or greater sciatic notch, drilling into the greater sciatic notch, or placing screws in this region can directly injure the sciatic nerve. Special Hohman-type retractors have been developed for the lesser sciatic notch to protect the nerve during posterior exposure. In addition, it is important to maintain the obturator internus muscle and tendons between the nerve and the retractors during this exposure [11–13, 15].

Postoperative injury may result from several factors. A hematoma may develop which may progressively compress the nerve resulting in sciatic neuropathy in the early postoperative period [17, 18]. Capsular and muscular scarring, hardware debris, or implant migration can irritate the sciatic nerve and present as a delayed neuropathy months to years after surgery [4, 19–24].

Heterotopic ossification around the sciatic nerve is another common postoperative cause of sciatic neuropathy [20, 25, 26]. Formation of heterotopic bone can be exacerbated by traumatic brain injury and may result in encasement and tethering of the sciatic nerve (Fig. 1a–g) [10, 20, 25–27]. The neuropathy in these cases often presents in a delayed progressive fashion and coincides with the formation of the ectopic bone (Fig. 1a–g) [10, 20, 25–27]. The progressive neuropathy may be exacerbated by new trauma. Thakkar and Porter reported on a case of a posterior fracture–dislocation of the hip treated with open reduction 3 years prior. The patient presented with medial leg pain, paresthesias, and a foot drop after a minor fall on the buttock. Radiographs revealed mature heterotopic bone around the hip and extending distally. During surgical exploration, the nerve was encased in heterotopic bone and could only be identified distal to the new bone [26].

Extended surgical approaches such as the iliofemoral or triradiate are associated with a higher incidence of heterotopic ossification [28, 29]. Griffin and colleagues observed a heterotopic ossification rate of 30.2% with the use of the extended iliofemoral approach; no formal prophylaxis for heterotopic ossification was used in this series [29]. Letournel and Judet observed that 69% of operations performed through the extended iliofemoral approach, 45% of operations performed through a combined anterior and posterior approach, and 28% of operations performed through the Kocher–Langenbeck approach developed heterotopic ossification in the absence of prophylaxis [14]. Failure to prophylactically treat heterotopic ossification with postoperative radiation and/or indomethacin can result in nerve entrapment [29–32].

Natural history

Reports on the natural history of sciatic nerve injury have provided somewhat conflicting results. Epstein noted that 60% of 38 posttraumatic sciatic nerve injuries resulting from posterior fracture–dislocation of the hip fully recovered at 30 months after injury [33]. Letournel and Judet reported that 60% of 34 sciatic nerve injuries in 569 surgically treated acetabular fractures had significant to complete recovery [13]. Eighteen of these cases involved

the peroneal division and one was a complete lesion secondary to intraoperative nerve transection, the remainder were patchy or pure sensory lesions. Nine cases recovered fully and 12 cases had significant recovery over a 3-year period. One patient presented with severe sciatic pain without motor deficit 8 weeks after open reduction and internal fixation. Surgical exploration revealed nerve entrapment by heterotopic bone and fibrous tissue. Surgical release with removal of scar and heterotopic bone improved neurologic symptoms in this patient [13]. Higher neurologic recovery rates after sciatic nerve injury have been reported by others. Tile noted that 75% of posttraumatic injuries and all iatrogenic injuries either completely or partially recovered [11, 16]. Helfet and Schmeling reported that, when intraoperative monitoring was used, 100% of postoperative severe peroneal nerve injuries recovered [11].

In a report on 14 patients with displaced acetabular fractures and sciatic nerve injuries, Fassler and colleagues described, in detail, the severity and nature of the initial nerve deficit and correlated this with nerve recovery. They classified injuries as either mild (motor 3–4 or mainly sensory symptoms) or severe (motor <3 with markedly diminished sensation). They also divided injuries into involvement of the peroneal division (which was considered satisfactory if the individual muscles of the anterior compartment of the leg were graded >3) or involvement of the tibial division (which was considered satisfactory if plantar flexion strength was >3 with intact protective plantar sensation). They observed that patients who had mild peroneal, mild tibial, or severe tibial nerve injury all had significant recovery. However, severe involvement of the peroneal nerve was associated with a poor prognosis. Only three of ten patients with severe peroneal injuries had a satisfactory result, and one of these was 17 years old. Five of these patients had foot drop.

Clinical assessment

Sciatic nerve palsy may present with a wide range of clinical findings. Sensory manifestations include posterior thigh pain at the level of the sciatic notch, radicular pain along the sciatic nerve distribution with hip and knee range of motion, diminished sensation, and paresthesias. Motor manifestations include weakness or paralysis of muscles innervated by the sciatic nerve [3, 7, 15]. The peroneal division of the sciatic nerve is usually affected with resultant weakness of the muscles of the anterior and lateral compartments of the leg [5, 6]. Posterior thigh and radicular pain may be worsened by hip flexion and knee extension and thus can limit motion at these joints. This pain-limited motion compounded with the initial acetabular trauma can result in contractures further restricting hip motion [7, 12]. These symptoms, often mimicking radiculopathy or spinal stenosis, have been mistakenly interpreted as originating from the spine and have resulted in inappropriate lumbar decompression [22, 23]. Obtaining a detailed history of the temporal relationship between neurologic symptoms and acetabular trauma or reconstructive surgery and eliciting a clear past history of lumbosacral pain, radiculopathy, or myelopathy will help to differentiate between the lumbosacral nerve roots, lumbosa-

cral plexus, or sciatic nerve as the location of the pathology. A detailed physical examination, including a thorough sensory and motor examination, including evaluation for reflexes will help to identify the anatomic location of the neurologic deficit.

Plain radiographs of the pelvis may reveal heterotopic bone, hardware migration, wear debris, or fracture fragments in the greater sciatic notch (Fig. 1a–c). Radiographs of the spine may reveal evidence of spinal stenosis, spondylolisthesis, or fracture which may contribute to sciatic nerve-type pain or motor weakness. CT scans can clearly delineate the extent and location of heterotopic bone, acetabular or spinal column fracture patterns, and foraminal or canal stenosis (Figs. 1d–f and 2a). MRI of the pelvis can reveal muscular enlargement, edema, or scarring around the sciatic nerve which may potentially represent injury (Fig. 2b–d). MRI of the spine can identify lumbar disc degeneration or herniation which may contribute to sciatic nerve-type symptoms.

EMG has been shown to be helpful in identifying the anatomical location of a neurological lesion and whether it is a neuropraxia, axonotmesis, or neurotmesis [7]. Yuen and colleagues have shown that a recordable compound muscle action potential of the extensor digitorum brevis and an initial absence of paralysis of muscles controlling ankle plantar flexion and dorsiflexion are good prognostic factors for recovery in patients with sciatic neuropathy [34].

Prevention

Letournel and Judet have advocated taking several simple precautions to prevent iatrogenic sciatic nerve injury. They recommended, when operating in the posterior approach, to keep the hip extended and the knee flexed to minimize traction on the sciatic nerve. They advocated the use of transcondylar traction to maintain this position during the operation which they note reduced their postoperative sciatic nerve palsy rate from 18.4% to 3.3% [13]. Excessive retraction posteriorly with the hip in flexion or levering retractors placed in the lesser sciatic notch can place tension on or directly compress the sciatic nerve, respectively, and should be avoided. Special Hohman-type retractors with curved edges should be used for nerve retraction. The obturator internus tendon and muscle belly should be held between the nerve and the retractor [11–13]. Great care should be taken when drilling or placing screws in the vicinity of the greater sciatic notch; appropriate fluoroscopic views should be obtained immediately to confirm safe instrument positioning and hardware placement [11–13, 15].

SSEP have been shown to be helpful in preventing iatrogenic sciatic nerve injury during pelvic and acetabular surgery [8, 35, 36]. Middlebrooks and colleagues have suggested that intraoperative monitoring is unnecessary, arguing that observing the preventive methods advocated by Letournel and Judet can result to a very low incidence of iatrogenic injury [37]. Nevertheless, monitoring may be helpful in avoiding nerve injury in high-risk groups such as those with posterior wall or column fracture and preoperative nerve injury. In these groups, SSEP changes are seen 60% of the time [8]. Specifically in this group, the incidence of posttraumatic nerve injury was 30% (25/83) and the

Fig. 1. Anteroposterior radiograph (**a**) and axial computed tomography images (**b** and **c**) of a patient 6 weeks after a motor vehicle accident with a displaced transverse-posterior wall right acetabular fracture. The patient sustained multiple additional injuries including a head injury and liver laceration. Because of persistent hemodynamic instability, the head injury, and subsequent persistent fevers, the patient was not cleared for acetabular reconstructive surgery until 8 weeks after the injury. The patient was intubated and had a complete foot drop as well as diminished sensation and paresthesias on the right side at the time of transfer to our facility. Note the extensive heterotopic ossification in the vicinity of the sciatic nerve on the right side. He also had a pubic root fracture on the left side; there is now extensive heterotopic ossification on that side as well. Reprinted with permission from The Journal of Bone and Joint Surgery, Inc. **d** Intraoperative photo demonstrating extensive heterotopic ossification at the sciatic notch. The patient is in the prone position. Proximal is to the right of the image and distal is to the left of the image. Posterior is to the top of the image and anterior is to the bottom of the image. Curved clamp is pointing to heterotopic ossification extending to the sciatic nerve (*black arrow*). To the right of the nerve, residual scar is seen as the nerve enters the greater sciatic notch. Distally, the nerve has been dissected free of scar and bursa.

incidence of intraoperative SEP waveform changes was 29% (24/83) [8]. The use of SSEPs resulted in low iatrogenic sciatic/peroneal neuropraxia rates (2%) [35]. Monitoring the tibial and peroneal divisions separately is recommended as monitoring the tibial division alone resulted in a 5% postoperative injury rate to the peroneal division [8].

There is evidence that several anesthetics may interfere with or suppress intraoperative monitoring [38–40]. In general, narcotic agents are less likely to interfere with monitoring than inhalation agents [40]. Epidural anesthesia blocks SSEP and motor tract monitoring. Therefore, general anesthesia with intravenous anesthesia is used when intraoperative monitoring is required. If a spinal/epidural is placed for managing postoperative pain, anesthesia should not be administered through the spinal column until after intraoperative monitoring is completed.

Unfortunately, SSEP monitoring does not provide information on the motor conduction pathways of the sciatic nerve or allow for instantaneous monitoring. In contrast, spontaneous EMG allows for instantaneous monitoring, allowing the surgical team to respond rapidly to noxious stimuli and avoid an iatrogenic injury. Spontaneous EMG also monitors motor pathways. Helfet and colleagues showed in a prospective study that SSEP and EMG in combination is better than SSEP alone in preventing iatrogenic nerve injury because of the potential for instantaneous monitoring. The surgical team can then respond more rapidly to offending stimuli, such as excessive retraction, and thus prevent permanent nerve injury [41].

Sciatic nerve monitoring has been used with good results during revision hip arthroplasty [42–44]. Motor-evoked potentials when used in combination with EMG monitoring identified significant electrical events during acetabular reconstruction, specifically if there was excessive posterior retraction with the hip in flexion [44]. In addition, excessive lateral or anterior femoral retraction during hip arthroplasty has also demonstrated neurologic activity, suggesting that the sciatic nerve was at risk [43].

Fig. 2. CT and MRI in a 32-year-old man 9 weeks after sustaining a mild closed head injury and buttock contusion from a fall down a flight of stairs. Symptoms included radicular pain, paresthesias, and ankle weakness. **a** Axial CT cut of the pelvis showing enlargement and ossification of the piriformis muscle (arrow). **b** T1-weighted magnetic resonance image showing low-signal intensity in the area of ossification (arrows). **c** T2-weighted magnetic resonance image demonstrating heterogeneous signal intensity in the area of ossification (arrow). **d** Gadolinium-enhanced image showing enlargement and ossification of piriformis muscle (arrow). Reprinted with permission from The Journal of Bone and Joint Surgery, Inc.

Prophylaxis for heterotopic ossification can be helpful in preventing progressive nerve encasement, restriction of hip range of motion, and delayed progressive neuropathy [14]. Prophylaxis with perioperative irradiation and indomethacin have both been used with good results [30–32]. Burd and colleagues reported on 166 acetabular fractures reconstructed through a posterior, extensile, or combination approach. Seventy-eight patients were randomized to 800 cGy of local radiation therapy within 72 h after surgery, and 72 patients received a 6-week course of indomethacin (25 mg, three times a day) beginning within 24 h after surgery. Moderate to severe heterotopic ossification (Brooker grade III or IV) was observed in 11% of the patients in the indomethacin group and in 4% of the patients in the radiation therapy group. This difference was not statistically significant [32]. Thus, the two prophylactic regimens appear equally effective. Heterotopic ossification developed in 16 out of 16 patients who did not receive prophylaxis with six demonstrating moderate to severe heterotopic ossification [32].

Combination therapy with perioperative irradiation and indomethacin may be more effective than either alone. Moed and Letournel reported on 54 acetabular fractures reconstructed through a posterior or an extended iliofemoral surgical approach treated with indomethacin and irradiation for heterotopic ossification prophylaxis. Indomethacin was administered as daily doses of 25 mg started within 24 h of operation and continued for 4 weeks. Irradiation was by either 1,200 cGy in three daily doses or by a single 700-cGy dose on the first postoperative day. Combination therapy

was very effective with 44 fractures showing no ectopic bone and ten fractures showing minimal heterotopic ossification (Brooker grade I) [45, 46]. Irradiation with 700 cGy dose was as effective as irradiation with 1,200 cGy in three daily doses [46].

Surgical decompression and results

There are few reports in the literature describing surgical decompression of the sciatic nerve following acetabular fractures or reconstructive surgery [3, 10, 26, 27, 47]. Kleiman and colleagues reported on a patient with a posterior wall acetabular fracture–dislocation who was treated with open reduction and internal fixation of the posterior wall fragment [10]. The patient was neurologically intact in the immediate postoperative period, but developed progressive sciatica and weakness in ankle dorsiflexion postoperatively. Radiographs and surgical exploration revealed heterotopic bone entrapping the sciatic nerve. Sciatic nerve release at 4.5 months after surgery with excision of heterotopic ossification resulted in a complete return of sensation, but no improvement in motor function [10].

Hirasawa and colleagues reported on a similar case of a posterior hip dislocation treated with open reduction where sciatic neuropathy developed over 4 months [27]. The sciatic nerve was explored and was observed to be encased in heterotopic bone. Sciatic nerve release with excision of ectopic bone almost completely improved sensory and motor symptoms [27]. Thakkar and Porter reported on a

patient who sustained a posterior fracture–dislocation of the hip with sciatic nerve laceration who was treated with open reduction and nerve repair [26]. Three years after injury, he developed pain and paresthesias along the medial side of his leg and a foot drop after a fall on the buttock. Radiographs and surgical exploration revealed encasement of the sciatic nerve in ectopic bone. Sciatic nerve release with excision of ectopic bone resolved sensory symptoms, but the foot drop remained at 2 years follow-up [26].

Surgical releases of the sciatic nerve have also been performed for complications related to soft tissue hypertrophy and scarring [3, 4, 20]. Benson and Schutzer have proposed that hematoma formation and scarring between the external rotators and sciatic nerve following blunt trauma to the buttock may precipitate pain in the region of the sacroiliac joint, greater sciatic notch, and piriformis muscle. The pain noted in this “piriformis syndrome” often mimic radiculopathy and can be exacerbated by hip flexion [3]. Benson and Schutzer reported on 14 patients who had a history of a blow to the buttock with pain in the area of the greater sciatic notch which was exacerbated by hip flexion, adduction, and internal rotation; 11 patients had severe radicular lower extremity pain. Surgical exploration revealed adhesions between the piriformis muscle, the sciatic nerve, and the roof of the greater sciatic notch. Surgical release of the piriformis tendon with sciatic neurolysis resulted in good to excellent results in all patients at an average 2.3 months follow-up [3]. Uchio and colleagues reported on a case of bilateral sciatic neuropathy with radicular pain occurring after falls 4 and 6 years after bilateral cementless total hip arthroplasty [4]. Surgical exploration revealed a hypertrophic hip capsule and a tense piriformis muscle. Releasing each piriformis muscle and both sciatic nerves resolved the symptoms [4]. Beauchesne and Schutzer reported on a case of myositis ossificans of the piriformis muscle in a patient who fell down a flight of stairs sustaining contusions to the head, buttock, and thigh [20]. Symptoms included radicular pain, paresthesias, and ankle weakness. At 6 weeks post-trauma, radiographs revealed periosteal bone formation in the anterior and lateral aspects of the middle third of the right femur. CT and MRI revealed enlargement and ossification of the piriformis muscle (Fig. 2a–d). Surgical exploration revealed an osseous mass within the piriformis muscle; the sciatic nerve was intimately associated with this mass and was compressed between it and the roof of the sciatic notch. Excision of ectopic bone with sciatic nerve decompression resulted in complete relief of sensory and motor symptoms at 4 weeks [20].

Sciatic nerve release has also been performed for complications related to wear debris, hardware loosening, or migration [21–24]. Crawford reported on three patients with long-standing total hip replacements who developed progressive sciatic neuropathy with radicular pain following compression of the nerve by wear debris. At surgical exploration, in all three cases, a granulomatous mass consisting of histiocytes and multinucleated giant cells was noted to be compressing the sciatic nerve. Excision of the lesion and nerve decompression resolved the symptoms in all patients in the immediate postoperative period [21].

Stiehl and Stewart reported on a case of sciatic neuropathy following pelvic plate loosening 6 months following pelvic reconstruction. Surgical exploration at 1 year revealed a screw impinging on the nerve. Removal of hardware and sciatic nerve release improved the patient’s symptoms [24]. Intrapelvic migration of an acetabular cup has also been reported as a cause of delayed sciatic neuropathy [23]. Acetabular revision surgery with removal of the protruded cup and cement completely resolved sciatic neuropathy in this patient. Unfortunately, this patient had undergone a laminectomy without relief of her symptoms before it was determined that cup migration was the problem [23].

Issack and colleagues have retrospectively reviewed the effect of sciatic nerve release on preoperative sciatic neuropathy associated with acetabular fractures and reconstructive acetabular surgery. Ten patients with sciatic neuropathy associated with an acetabular fracture were treated with sciatic nerve release from scarring and heterotopic ossification in addition to other procedures including open reduction and internal fixation of the acetabulum and total hip arthroplasty. At 26 months follow-up, the authors noted that radicular pain, diminished sensation, and paresthesias improved after nerve release with all patients obtaining partial to complete relief. Fifty-seven percent of patients with motor symptoms demonstrated improvement in function after nerve release. Forty percent of patients with a foot drop demonstrated functional improvement after nerve release. No patient was made worse neurologically by sciatic nerve release. They concluded that, while surgical decompression of the sciatic nerve can resolve sensory neuropathy, motor symptoms are more resistant to improvement [47].

Conclusions

Sciatic nerve injuries associated with acetabular fractures may be posttraumatic, perioperative, or postoperative and can present with a wide range of sensory and motor symptoms. Effective management of these injuries requires a clear history of the temporal relationship between onset of symptoms and acetabular trauma or reconstructive surgery and possible preexisting spinal disorders. Appropriate imaging studies, including plain radiographs, CT, and MRI, and electromyographic studies can help localize the site of nerve injury. Iatrogenic injury can be prevented by keeping the hip extended and knee flexed during surgery. Excessive posterior retraction with the hip in flexion or levering of retractors in the lesser sciatic notch should be avoided. Safe internal fixation can be aided with the use of intraoperative fluoroscopy, SSEP, and spontaneous EMG. Heterotopic ossification prophylaxis in high-risk cases, such as after extensile approaches, should be instituted to preventing late sciatic nerve entrapment. Surgical decompression appears to depend on several preoperative factors and appear to have better outcomes for sensory than motor palsy; however, it is unclear if surgical decompression improves the outcomes of sciatic nerve palsy over the natural history as there are no prospective randomized studies on the subject.

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