Surgical Decompression Improves Symptoms of Late Peroneal Nerve Dysfunction After TKA

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Abstract

Acute peroneal nerve palsy is a well-known complication of total knee arthroplasty (TKA) that causes a neurological deficit typically seen within hours or days postoperatively. Peroneal nerve dysfunction presents more subtly than peroneal nerve palsy, with decreased knee range of motion, lateral knee pain, or both following TKA. The diagnosis of peroneal nerve dysfunction may not be suspected for weeks, months, or even years after TKA. Electromyography and nerve conduction studies can support the diagnosis. Historically, peroneal nerve palsy following TKA has been treated nonoperatively but has had an unsatisfactory rate of complete recovery. Recently, a few reports have demonstrated that patients with either peroneal nerve palsy or dysfunction after TKA have had excellent results with surgical decompression of the peroneal nerve.

The authors describe a 63-year-old woman who reported transient episodes of lateral knee and leg pain for years after undergoing TKA. She eventually underwent electromyography and nerve conduction studies that indicated a diagnosis of peroneal nerve dysfunction. Approximately 10 years after the TKA, she underwent surgical decompression of the peroneal nerve and has done well since, with significant pain relief and an increased activity level.

This case supports the recent literature describing peroneal nerve dysfunction as an uncommon but surgically treatable cause of lateral knee pain following TKA. Increased awareness of the condition and its facile treatment via surgical decompression may result in improved outcomes years after TKA.

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Peroneal nerve palsy is a rare complication following total knee arthroplasty (TKA), with an incidence of 0.3% to 1.3% in studies including at least 1,000 primary TKAs. Risk factors for developing peroneal nerve palsy include preoperative valgus alignment of the knee, preoperative flexion contracture of the knee, epidural anesthesia, rheumatoid arthritis, prolonged tourniquet time, constrictive dressings, hematoma formation, and preexisting neuropathy. Typical symptoms manifest as sensory deficits along the dorsum of the foot and weakness in ankle dorsiflexion or great toe extension. Peroneal nerve palsy usually occurs within the first 2 postoperative days or immediately after surgery while in the recovery room; however, recognition may be delayed if the patient received epidural analgesia. The diagnosis can be supported by electrodiagnostic findings.

Historically, peroneal nerve palsy has been treated with loosening of the surgical dressings in the recovery room and flexion of the knee. Patients undergo standard postoperative physical therapy regimens and are observed for improvements in ankle or great toe strength. For chronic motor weakness causing a “steppage” or “foot-slapping” gait, the patients are prescribed ankle-foot orthoses. Many authors have reported relatively poor recovery from peroneal nerve palsy when it is treated nonoperatively; at reported follow-ups of 6 months to 11 years, the rate of complete recovery from peroneal nerve palsy after nonoperative management ranged from 9% to 68%.

In 2 studies, Krackow et al and Mont et al described surgical decompression of the peroneal nerve for peroneal nerve palsy following TKA in the same 6 patients at 5 to 50 months after TKA. All 6 patients had improvement in symptoms to the extent that they discontinued use of their ankle-foot orthoses. Five patients had complete resolution of symptoms. The 1 patient with incomplete resolution of symptoms was decompressed 11 months after TKA and had complete resolution of motor symptoms with residual sensory paresthesias.

Peroneal nerve dysfunction presents in a less obvious fashion than peroneal nerve palsy after TKA. Zywiel et al outlined the specific symptoms of peroneal dysfunction as pain or dysesthesias in the leg on flexion or extension of the knee affecting rehabilitation, lateral distribution of leg pain, transient presentation of symptoms in the peroneal nerve distribution, and a positive Tinel’s sign of the peroneal nerve at the level of the fibula. In addition, unlike patients with peroneal nerve palsy, patients with peroneal nerve dysfunction do not have pronounced motor palsy (ie, foot drop) or persistent sensory defect of the foot. Patients with peroneal nerve dysfunction can present weeks to months (or even years) after TKA. If a patient has symptoms of peroneal nerve dysfunction, electrodiagnostic studies may be ordered to confirm the diagnosis.

Zywiel et al reported 11 patients treated with surgical decompression of the peroneal nerve for peroneal nerve dysfunction following TKA. All 11 patients had their diagnoses supported by electrodiagnostic studies. The other 5 patients had neuropathic sensory symptoms that interfered with their activities of daily living. Ten patients underwent surgical peroneal nerve decompression within 17 months of their TKA, with 1 patient undergoing decompression 127 months after the TKA. All 11 patients had some level of improvement in their symptoms postoperatively. The 6 patients with decreased knee ROM prior to decompression had a mean ROM improvement of 40° (range, 20°–70°). The 5 patients with neuropathic sensory symptoms prior to decompression had 1 or more of the following: reduction in pain, improvement in sensation in the lateral leg or dorsum of the foot, or higher activity tolerance.

The authors describe a woman with chronic transient symptoms of neuropathic pain along the lateral aspect of her right knee and leg following TKA who experienced years of worsening symptoms, underwent imaging studies that did not diagnose her condition, and received injections that failed to provide her with lasting pain relief. The diagnosis of peroneal nerve dysfunction was made after reviewing her history and her physical examination and electrodiagnostic studies.

**Case Report**

A 63-year-old woman reported pain and discomfort in her right lateral knee and leg. She had undergone a right TKA 10 years previously but had no other pertinent medical or surgical history. She had approximately 10° of flexion contracture and varus alignment of the knee prior to TKA (Figure 1). During the TKA, a cementless standard-plus Low Contact Stress femoral component and a cemented standard-plus Low Contact Stress tibial baseplate with a 10-mm polyethylene insert (Depuy Orthopaedics, Inc, Warsaw, Indiana) were implanted. The patella was not resurfaced. The implants were well-sized with no signs of instability, and the patella tracked well centered in the trochlea. After the TKA, the patient described difficulties in gaining adequate knee
ROM immediately postoperatively. At the 2-month postoperative examination, the patient lacked 15° of knee extension and had a maximum knee flexion of 95°. She completed an additional month of physical therapy, and her knee ROM was 10°-115° at the 3-month postoperative examination. She did not undergo manipulation under anesthesia at this time.

Although TKA relieved her preoperative knee pain, she reported a different type of pain postoperatively that consisted of transient episodes of pain along the lateral aspect of the knee and leg. The patient’s orthopedic surgeon treated her for degenerative joint disease of the proximal tibiofibular joint for the next several years, with multiple corticosteroid injections into that joint providing the patient with only a small amount of temporary relief after each injection. At routine 5- and 10-year follow-up clinical examinations, the patient’s knee showed no signs of instability and the patella tracked well. The patient’s follow-up knee radiographs showed no signs of loosening, osteolysis, or other hardware complications (Figures 2A, B).

The patient’s lateral knee and leg pain then worsened progressively, especially when walking, over several years prior to her presentation to the authors’ institution. More recently, the pain limited her ability to walk more than 100 yards. She developed transient symptoms of numbness over the lateral aspect of her right knee and leg, as well as the dorsum of her foot. She denied having any back, buttock, or thigh pain or pain when standing from a seated position. No other neurologic or constitutional symptoms were reported.

Magnetic resonance imaging of the lumbar spine demonstrated a minimal grade I anterolisthesis of the L4 on L5 without significant canal or foraminal stenosis, and the patient’s symptoms were inconsistent with lumbar radiculopathy. The patient was then referred to the authors’ institution for a second opinion regarding lumbar spondylosis as the etiology of her symptoms.

On evaluation at the authors’ institution, the patient had no knee effusion or skin changes overlying the knee or leg with painless knee ROM from 10°-115°. She had full strength throughout the bilateral lower extremities. She had slightly diminished but intact sensation to light touch along the lateral aspect of the dorsum of the right foot, but sensation was otherwise intact fully in the bilateral lower extremities. She had a positive Tinel’s sign elicited over the fibular head. She had a negative straight-leg raise bilaterally.

Electrodiagnostic studies were consistent with severe peroneal nerve mononeuropathy with symptoms of peroneal nerve dysfunction. Because her symptoms were refractory due to years of conservative management, surgical decompression of the peroneal nerve was performed after obtaining informed consent. An incision centered on the fibular head extending distally toward the tibialis anterior and proximally toward the knee crease was made and taken down sharply through the subcutaneous tissues. Dissection revealed a scarred hamstring and peroneus longus fasciae. The peroneal nerve was entrapped by the fascia of the peroneus longus at the fibular neck, and in situ decompression was performed without mobilization of the nerve. Neurolysis of the nerve distally revealed that the deep and superficial branches did not have any obvious abnormalities. Proximally, the nerve was free of epineural scar, although the overlying distal hamstring fascia was fibrous and was released. No neuroma or ganglion was visualized along the peroneal nerve path from the distal thigh to its division into the deep and superficial branches.

Six weeks postoperatively, the patient’s lateral knee and leg pain had completely resolved. She no longer had transient episodes of numbness on the lateral aspect of her knee and leg or on the dorsum of her foot. Her knee ROM was unchanged, but she reported increased activity levels. She was satisfied with the surgical results.

**DISCUSSION**

This case represents successful treatment of late knee and leg pain after TKA by peroneal nerve decompression in the subset of patients with poor outcomes after TKA with knee and leg pain of an unclear etiology, but with symptoms and signs of peroneal nerve dysfunction. Callahan et al9 performed a meta-analysis of 9879 patients and demonstrated fair or
poor outcomes in 10.7% of patients after TKA, but only 3.8% of the total patients underwent revision TKA. Many patients do not undergo revision TKA because the etiology of their poor result is unknown or is not thought to be amenable to revision TKA. Although the number of patients is small, those diagnosed with peroneal nerve dysfunction have done well with surgical decompression and avoided the morbidity associated with revision TKA. Even after years of activity-limiting lateral knee and leg pain, the current patient benefited from surgical decompression of the peroneal nerve. To the authors’ knowledge, only 1 prior report exists of a patients who underwent peroneal nerve decompression for peroneal nerve dysfunction more than 10 years after TKA, and that patient also benefited from surgical treatment.

Peroneal nerve palsy and dysfunction are rare following TKA. More common pathologies exist in patients, who report lateral knee pain after TKA. The work-up of painful TKA must raise concerns for intra-articular knee pathology such as infection, hardware complication, instability, and malpositioning as dictated by the clinical presentation of the patient. Orthopedic surgeons must also consider underlying neurological disorders, diabetes mellitus, spinal stenosis, herniated nucleus pulposus, other lumbar spondylosis, referred hip pain, degenerative joint disease of the proximal tibiofibular joint, claudication, deep venous thrombosis, complex regional pain syndrome, and psychosomatic illness as causes of lateral knee pain after TKA. Knowledge of peroneal nerve dysfunction is critical for the appropriate evaluation and treatment of patients with pain and discomfort after TKA.

The diagnosis of peroneal nerve dysfunction is challenging, as noted by Zywiel et al. Patients with peroneal nerve dysfunction can have subtle weakness in ankle dorsiflexion or great toe extension, as well as subtle sensory defects that can be found on physical examinations. Patients may also have completely normal motor and sensory examinations, so it is the transient complaints of weakness or diminished sensation in the peroneal nerve distribution that lead the physician to order electrodiagnostic studies to explore the potential diagnosis of peroneal nerve dysfunction.

The current patient had a subtle decreasing sensation on the dorsum of the foot and reported transient pain along the lateral knee and leg. These nerve-related symptoms distinguish the current patient’s presentation from the localized pain of degenerative joint disease of the proximal tibiofibular joint, for which she had received multiple injections. The diagnosis of peroneal nerve dysfunction was difficult to make and took years of evaluation by the patient’s primary care physician and multiple specialists to determine.

It is unknown whether the current patient developed peroneal nerve dysfunction immediately after her TKA or years later due to another cause. She did have a 10° flexion contracture prior to her TKA, which is an identified risk factor for peroneal nerve palsy after TKA. If the patient had been managed by the algorithm suggested by Zywiel et al. (Figure 4), then she possibly would have undergone electrodiagnostic studies after having inadequate knee ROM 4 weeks postoperatively and perhaps an earlier diagnosis of peroneal nerve dysfunction could have been made. Because the patient was not appropriately evaluated with a neurologic examination and did not undergo electrodiagnostic studies until years after her TKA, the exact timing and causality of her peroneal nerve dysfunction cannot be determined. It is possible that peroneal nerve entrapment may have caused the patient’s symptoms irrespective of her TKA. Regardless, the patient’s presentation suggests a natural history of peroneal nerve dysfunction following TKA. The authors believe that it is critical for surgeons to consider peroneal nerve pathology in the setting of the postoperative TKA patient with lateral knee pain or decreased ROM.
CONCLUSION

Peroneal nerve dysfunction is a rare cause of lateral knee pain or decreased ROM after TKA. Symptoms of peroneal nerve dysfunction after TKA include inadequate progress with knee ROM after 4 weeks of physical therapy, transient neuropathic symptoms along the lateral aspect of the knee, subtle sensory and motor deficits in the peroneal nerve distributions, and a positive Tinel’s sign elicited at the fibular head. A complete differential diagnosis associated with these symptoms should be explored. The diagnosis of peroneal nerve dysfunction can be supported by electrodiagnostic studies. Facile treatment with surgical decompression of the peroneal nerve has successfully improved peroneal nerve dysfunction after TKA. Although it is not ideal to decompress the peroneal nerve after years of symptoms, surgical treatment can be successful in a late presentation of symptoms. The authors encourage surgeons to be aware of peroneal nerve dysfunction and to report cases in the literature to improve understanding of this pathology.

REFERENCES