



A Case Report on Deep Peroneal Nerve Palsy Following Patella Dislocation

Anthony Kouri, Ryan Hamilton, Martin Skie

Department of Orthopaedic Surgery, University of Toledo Medical Center, Toledo, USA

Email: anthony.kouri@utoledo.edu

How to cite this paper: Kouri, A., Hamilton, R. and Skie, M. (2017) A Case Report on Deep Peroneal Nerve Palsy Following Patella Dislocation. *Open Access Library Journal*, **4**: e3563.

<https://doi.org/10.4236/oalib.1103563>

Received: March 27, 2017

Accepted: April 25, 2017

Published: April 28, 2017

Copyright © 2017 by authors and Open Access Library Inc.

This work is licensed under the Creative Commons Attribution International License (CC BY 4.0).

<http://creativecommons.org/licenses/by/4.0/>



Abstract

Background: Peroneal nerve injury following knee dislocation is a known complication described in numerous case reports. However, this complication has very rarely been described following patellar dislocation. We focus on the presentation and subsequent treatment of this complication. **Case:** We describe a thirteen-year-old patient who developed a deep peroneal nerve palsy following patellar dislocation. After her initial visit for patellar dislocation, our patient developed foot drop over the course of one week. Six months following her injury, she had fully functional recovery of the nerve with conservative treatment. **Conclusion:** Residual deep peroneal neuropathy following patellar dislocation is an exceedingly rare complication. Treatment should consist of conservative measures initially; however, surgical exploration may become necessary.

Subject Areas

Orthopedics

Keywords

Patella Dislocation, Peroneal Palsy, Pediatric

1. Introduction

Peroneal nerve injuries are common following trauma, and often present as foot drop in the clinical setting. In fact, this is the most common isolated mononeuropathy of the lower extremity. It is caused by damage to either the deep peroneal nerve (DPN) or more proximally at the common peroneal nerve (CPN). The DPN provides sensation to the first dorsal web space of the foot and an articular branch to the ankle joint. It also provides motor innervation to the tibialis anterior, extensor digitorum longus, peroneus tertius, and extensor hallucis

longus [1]. Often damage to the nerve occurs through transection, stretching, or contusion secondary to trauma.

Traumatic knee dislocation injuries are often reported to cause damage to the peroneal nerve. The current literature provides many examples of knee dislocations and associated peroneal nerve injuries. Some report the incidence to be as high as 50%. Though it is not uncommon to see a peroneal palsy following knee dislocation, there is only one known report in which an isolated patella dislocation has resulted in a peroneal nerve palsy [2]. In that case, the patient ultimately recovered peroneal nerve function within ten days of injury using conservative treatment.

Treatment for peroneal nerve palsy is largely conservative with the use of an ankle foot orthotic (AFO), physical therapy, and observation. Nonetheless, some patients will ultimately require exploration and possible nerve repair after a period of conservative treatment.

We report on a patient with deep peroneal palsy and residual foot drop for six months following patella dislocation. Guardian consent was obtained prior to the publishing of this report.

2. Case Report

A thirteen-year-old girl presented to our clinic with left foot drop following patellar dislocation. She denied any previous history of such incidents, injuries, or surgeries. The initial injury occurred six weeks prior while playing soccer. In an attempt to kick the ball, she planted and hyperextended her knee, which caused a lateral patellar dislocation. The patella was reduced at the school, and she did not seek help that day.

One day after injury, she was taken to an outside orthopaedic surgeon for further evaluation. Radiographs taken at that visit demonstrated no fractures or dislocation. There was minimal swelling on exam. She was placed in a well-padded cast and instructed to follow-up in two weeks.

One week later, she noticed increased numbness and tingling along the dorsum of the left foot. Her symptoms progressed to weakness and foot drop. At that time, she was re-evaluated by the treating orthopaedic surgeon. On exam, there was no significant swelling, vascular compromise, or deformity present. The patient denied any significant pain. All inflammatory labs were within normal limits. The cast was bi-valved and she was placed in an AFO. An electromyogram (EMG) of her left lower extremity was ordered and she followed-up one week later. In that time, she had no resolution of symptoms. The EMG demonstrated findings consistent with deep peroneal nerve (DPN) palsy, and she was referred to our clinic for further evaluation.

This patient presented to us six weeks after the patellar dislocation occurred, and five weeks following onset of DPN palsy symptoms. She had continued complaints of numbness along the dorsum of the left foot and persistent foot drop. Her extensor digitorum strength was resistant only to gravity (grade 2); tibialis anterior and extensor hallucis longus had no motor strength (grade 0).

Two-point discrimination was significantly diminished in the left DPN distribution (>15 mm vs. 10 mm). Left superficial peroneal nerve (SPN) distribution was minimally diminished (8 mm vs. 7 mm). She was instructed to continue the AFO, perform passive range of motion at the ankle, and to follow-up in one month.

At her follow-up visit, she had persistent numbness in the first toe web space. Her strength in extensor hallucis longus (grade 2), extensor digitorum longus (grade 4), and tibialis anterior (grade 3) improved. She was told to wean out of the AFO at home as her dorsiflexion strength began to increase. When she returned six weeks later her strength advanced in extensor hallucis longus (grade 3), tibialis anterior (grade 4), and digitorum longus (grade 5). Sensation in the first web space began to return at this visit. We discontinued the AFO. At her six-month follow-up visit, she had complete return of motor and sensory function. Her muscle strength in regard to extensor hallucis longus, extensor digitorum longus, and tibialis anterior was grade 5 at that time. Additionally, 2-point discrimination was 9 mm and 8 mm in the DPN and SPN distributions, respectively.

3. Discussion

Foot drop is the most common isolated mononeuropathy of the lower extremity. It is caused by damage to the DPN or the CPN. The DPN provides sensation to the first dorsal web space of the foot and an articular branch to the ankle joint. It also provides motor innervation to the tibialis anterior, extensor digitorum longus, peroneus tertius, and extensor hallucis longus [2].

The CPN is fixed near the neck of the fibula. Proximal to the fibula the nerve is susceptible to a traction injury when the knee is subjected to varus and hyperextension forces. Distal to the fibula, the SPN runs vertically downwards in the substance of the peroneus longus muscle while the DPN continues closely applied to the interosseous membrane between tibialis anterior and extensor digitorum longus. Because of this latter relationship, the DPN is more vulnerable to traction injury during dislocation [3].

In addition to knee dislocation, other knee injuries have been associated with peroneal nerve palsy. Specifically, ligamentous knee injuries have been reported to have a high occurrence of peroneal nerve injury. In a series of 31 lower extremity sports injuries, 17 were peroneal nerve injuries. Eight of these traumatic injuries were associated with a ligamentous injury of the knee. Most of these involved anterior cruciate ligament rupture. This often occurred in conjunction with injury to the lateral, collateral, or posterior cruciate ligament [4].

It is reasonable to suggest that a ligamentous knee injury may have occurred at the time of our patient's patella dislocation, thus making peroneal nerve injury more likely. However, on exam our patient had no findings consistent with ligamentous injury. Furthermore, throughout her care, she had no subjective feelings of instability or pain with activity.

Our reported case is the second known instance of an adolescent with patellar dislocation and subsequent DPN palsy. In comparison to the case presented by

Shaikh *et al.*, the patient we present had a significantly longer recovery time for her peroneal palsy. The palsy previously reported resolved within 10 days, while our patient's palsy required six months to fully resolve [2].

Numerous factors had to be considered and eliminated before we were able to conclude that the patella dislocation had caused the DPN palsy. Though it is possible that a tightly applied cast caused this injury, we strongly believe it was not the source. When the patient originally presented to us, she had very minimal swelling. The cast was made intentionally loose with a significant amount of padding. On inspection at follow-up, there appeared to be no abnormal force contribution from the cast. Additionally, our patient had no swelling or pain throughout the week that she developed the DPN palsy. It is also possible that she sustained a more severe injury than initially recognized. However, her physical exam was not consistent with a ligamentous injury or a knee dislocation. In our opinion, it is more likely that the palsy began to develop immediately following her injury, and was missed on initial exam.

No current studies exist evaluating prognosis for return of nerve function following patellar dislocation. Niall *et al.* studied CPN palsy after knee dislocation, which demonstrated a poor long-term prognosis. They found that the SPN innervated muscles had a better prognosis for recovery than the DPN innervated anterior compartment muscles [3]. Bonneville *et al.* also reviewed CPN palsy after knee dislocation. In that study, they found that the anatomic status of the CPN, subjected to violent traction by dislocation, was the most significant prognostic factor for neurologic recovery. In about 25% of dislocations, contusion-elongation over several centimeters was associated with as poor a prognosis as total rupture [5]. In our case, the CPN was functioning appropriately. The DPN was most affected by the injury. Based on the work of Bonneville *et al.*, this may have favorably contributed to our patient's full recovery.

For recognized DPN palsy, it is best to initially treat with conservative management before considering surgical options. Treatment is often multimodal depending on patient symptoms, and the severity of their neuropathy. Pain is typically the first presenting symptom and is often neuropathic in nature. Pain can be managed using topical lidocaine, capsaicin, selective serotonin reuptake inhibitors, antiepileptics, opioids, and μ -receptor agonists. Modalities such as heat and ice can be used as well, but need to be applied carefully [4]. Many of these patients have sensory loss, and may cause further injury if they are not careful. If the patient has complete loss of strength, passive range of motion may be all that is possible. It is imperative to maintain proper ankle range of motion so that residual heel cord contracture will not prevent them from being able to walk. Equinovarus foot deformity is a common complication of ankle dorsiflexion weakness. It is essential that the patient maintain his range of motion to have the ability to ambulate. Patients with sensory loss should check their feet daily to prevent ulcer formation. A simple lace-up ankle sleeve with medial and lateral support may assist a patient with proprioceptive loss if present. Ankle dorsiflexion is necessary to clear the toes while ambulating. An AFO maintains the foot

in neutral position so that the patient can achieve a normal gait pattern [4]. Surgical intervention in a closed peroneal nerve injury is typically not considered until the patient is at least 4 months removed from the time of injury, and all conservative options have been attempted. For failed conservative treatment of peroneal nerve traction injuries, patients have 35% to 50% recovery of sensory and motor function with secondary nerve grafting. Transfer of the tibialis posterior tendon to restore active dorsiflexion is the alternative reconstructive procedure [6]. These are surgical options we would have considered [7].

In this case, we obtained an EMG to confirm our diagnosis of DPN palsy, and placed the patient in an AFO until function returned. She was also instructed to do passive range of motion exercises at home. Her progress was monitored in one-month intervals. The patient's strength in the extensor hallucis longus (grade 0 → 5), and tibialis anterior (grade 0 → 5) improved significantly with conservative management. If satisfactory function had not begun to return by 4 - 6 months, we would have obtained another EMG, and considered the aforementioned surgical interventions.

4. Conclusion

In this case report, we presented a thirteen-year-old girl who sustained a patella dislocation that was complicated by the development of a deep peroneal nerve palsy. This was only the second reported case in which an isolated patella dislocation led to a peroneal nerve palsy. In both instances, conservative treatment in the form of AFO use, passive range of motion, and observation was implemented. Our patient began to see some return of sensation and motor function at 10 weeks, but did not fully recover until she was 6 months out from the time of injury. This was a significantly longer recovery period than the prior reported case. We feel that conservative treatment is the best management strategy in these patients, and we would treat any patient with a similar presentation in the same manner.

Disclosures

Dr. Kouri has nothing to disclose. He meets the authorship requirements as stated in the Uniform Requirements for Manuscripts Submitted to Biomedical Journals. Dr. Hamilton has nothing to disclose. He meets the authorship requirements as stated in the Uniform Requirements for Manuscripts Submitted to Biomedical Journals. Dr. Skie has nothing to disclose. He meets the authorship requirements as stated in the Uniform Requirements for Manuscripts Submitted to Biomedical Journals.

Fund

No Funding was received for this work. Each author believes that this manuscript represents honest work.

References

- [1] Lawrence, S.J. and Botte, M.J. (1995) The Deep Peroneal Nerve in the Foot and An-

kle: An Anatomic Study. *Foot & Ankle International*, **16**, 724-728.

- [2] Shaikh, A.H., Dawood, A., Murthy, G.E., et al. (2012) Patella Dislocation Association with Drop Foot. *Journal of the College of Physicians and Surgeons-Pakistan*, **22**, 182-183.
- [3] Niall, D.M., Nutton, R.W. and Keating, J.F. (2005) Palsy of the Common Peroneal Nerve after Traumatic Dislocation of the Knee. *Journal of Bone and Joint Surgery*, **87**, 664-667. <https://doi.org/10.1302/0301-620X.87B5.15607>
- [4] Baima, J. and Krivickas, L. (2008) Evaluation and Treatment of Peroneal Neuropathy. *Current Reviews in Musculoskeletal Medicine*, **1**, 147-153. <https://doi.org/10.1007/s12178-008-9023-6>
- [5] Bonneville, P., Dubrana, F., Galau, B., Barbier, O., Neyret, P., Rosset, P. and Saragaglia, D. (2010) Common Peroneal Nerve Palsy Complicating Knee Dislocation and Bicusiate Ligaments Tears. *Orthopaedics & Traumatology: Surgery & Research*, **96**, 64-69. <https://doi.org/10.1016/j.otsr.2009.12.001>
- [6] Westhout, F.D., Paré, L.S. and Linskey, M.E. (2007) Central Causes of Foot Drop: Rare and Underappreciated Differential Diagnoses. *Journal of Spinal Cord Medicine*, **30**, 62-66. <https://doi.org/10.1080/10790268.2007.11753915>
- [7] Garozzo, D., Ferraresi, S. and Buffatti, P. (2004) Surgical Treatment of Common Peroneal Nerve Injuries: Indications and Results. A Series of 62 Cases. *Journal of Neurosurgical Sciences*, **48**, 105-112.



Open Access Library

Submit or recommend next manuscript to OALib Journal and we will provide best service for you:

- Publication frequency: Monthly
- 9 [subject areas](#) of science, technology and medicine
- Fair and rigorous peer-review system
- Fast publication process
- Article promotion in various social networking sites (LinkedIn, Facebook, Twitter, etc.)
- Maximum dissemination of your research work

Submit Your Paper Online: [Click Here to Submit](#)

Or Contact service@oalib.com