A Unique Case of Ulnar Tunnel Syndrome in a Bicyclist Requiring Operative Release

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ABSTRACT

The continued growth of recreational and competitive sports is accompanied by the need for health care providers to recognize and treat conditions in athletes that have been traditionally associated with other occupational injury. This is particularly important when early diagnosis and prompt intervention for prevention and treatment may alter the outcome. We present an interesting case of ulnar tunnel syndrome in a high-performance bicyclist with compressive ulnar neuropathy refractory to nonoperative management but successfully treated with surgical release. We review evaluation, diagnosis, and historical and current treatment algorithms.

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lnar tunnel syndrome (UTS) is an uncommon problem, seen usually in association with occupational injury involving repetitive trauma or pressure. It has been noted in construction workers, carpenters, and pneumatic drill operators.¹ Potential structural etiologies include thrombosis of the ulnar artery, proliferation of synovium, prominent hook of hamate, schwannoma, and ganglion cyst.² The common factor in all of these is sustained pressure over the hypothenar area or a space-occupying mass in Guyon's canal.

Symptoms of ulnar nerve compression in the bicycle rider have been a recognized problem since 1896, when Destot described the ulnar nerve being compressed at the pisiform.¹ Since that time there have been numerous case reports discussing the motor and sensory symptoms produced and whether conservative treatment is sufficient to obtain symptom relief or surgical intervention is necessary. In the vast majority of reports reviewed, conservative therapy resulted in symptom relief. ^{3,4} In fact, some authors have recommended against surgery for this injury when it is associated with bicycling, suggesting that nonoperative management should be the rule.⁵

We report a case of UTS that developed after an isolated bicycling event; the patient had both motor and sensory involvement, associated muscle atrophy, and an ulnar claw deformity that proved unresponsive to nonoperative treatment. Subsequent ulnar tunnel release led to a rapid and complete resolution of symptoms.

CASE REPORT

A 29-year-old woman experienced the acute onset of numbness and tingling in the ring and small fingers of her left hand following an 80-mile bike ride. Clawing of the digits of that hand began immediately. Despite a period of splinting, activity modification, and avoidance of all provocative activities, including biking, her symptoms persisted and even progressed. She was seen in the occupational therapy clinic 5 weeks after her injury, having been referred for weakness and decreased range of motion of the affected hand. At that time she had visible atrophy of her ulnar-innervated intrinsic musculature, and she was subsequently referred to the orthopedic hand surgery service.

The patient denied pain at rest but reported exquisite tenderness with pressure over Guyon's canal in the left hand as well as decreased sensation in the small finger and the ulnar aspect of the ring finger. Active range of motion of her ring finger proximal interphalangeal joint (PIP) was found to be 35° to 115° , with the distal interphalangeal joint (DIP) measured at 5°-55°. Active range of motion of her small finger PIP was found to be 20°-94°, with the DIP of this finger measured at $15^{\circ}-65^{\circ}$. The patient had full passive motion of all her digits without contractures. Measurement of her grip strength was clinically significant, in that the right hand averaged 40 pounds (using Jamar testing; Jamar Dynamometer, Model 1, Clifton, NJ), whereas the left hand averaged only 7 pounds (17.5% of the grip strength

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Table. Shea and McClain's Classification of Ulnar Nerve Compression Lesions⁶

Туре	Sensory Deficit	Motor Deficit	Location
I	Yes	Yes	Ulnar nerve proximal to the canal or within Guyon's canal before the bifurcation of the deep and superficial branches
II	No	Yes	Deep motor branch compression distal to Guyon's canal or within the canal distal to the bifurcation
111	Yes	No, except palmaris brevis	Superficial sensory branch compression distal to Guyon's canal or within the canal distal to the bifurcation

Modified from Shea J, McClain E. Ulnar-Nerve Compression Syndromes at and below the Wrist. J Bone and Joint Surg. 1969;51-A(6): 1095-1103. Used with permission.

in the right hand). Her lateral pinch was found to be 12 pounds on the right hand and 4 pounds on the left, and tip pinch was 11 pounds on the right hand and 2 pounds on the left.

Radiographic findings for the affected wrist were unremarkable, and electromyography and nerve conduction studies were obtained. The findings of these were abnormal, showing a prolonged latency of the ulnar sensory and motor nerve across the wrist of the left hand. The median, radial, Despite continued occupational therapy, splinting, and cessation of all bicycling, the patient's symptoms worsened over the ensuing 8 weeks. She elected to undergo a left ulnar tunnel release. Under general anesthesia, a Brunner-type incision was made over the interval between the pisiform and the hook of the hamate, and Guyon's canal was released in its entirety. There was no evidence of any mass, aneurysm, or other spaceoccupying lesion, but there were distion had markedly improved. Twopoint discrimination had improved to 5 mm in all fingertips, and she was able to rest her hands in a more normal posture. Ten weeks postoperatively, all symptoms, including incisional tenderness, had completely resolved. The patient had no increased sensitivity and felt that she had regained full strength of her affected hand. Full range of motion was noted, and 2-point discrimination was 5 mm in all fingertips. The grip strength of the left hand has improved to 37 pounds (88.1% of the strength of the unaffected hand, which was measured at 42 pounds on average). At her 2-year follow-up, the patient continued to have normal sensation and had returned to all activities, including biking, without any further incidents.

DISCUSSION

Guyon's canal is formed medially by the pisiform bone, laterally by the hook of hamate, palmarly by the volar carpal ligament, and dorsally by the transverse carpal ligament. The ulnar nerve, artery, and vein pass through the canal. Proximal to the canal, the dorsal cutaneous branch of the ulnar

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and dorsal cutaneous branches of the ulnar nerve had normal conduction and latencies. Electromyography of the left upper extremity was significant for active and chronic denervation changes in the ulnar-innervated intrinsic musculature. The more proximally ulnar innervated muscles, such as the flexor carpi ulnaris (FCU) and the flexor digitorum profundus (FDP), were normal. Specifically, the patient had 1+ fibrillations and 1+ positive sharp waves in the first dorsal interosseous muscle, and her fourth dorsal interosseous muscle showed no fibrillations but 1+ sharp waves and many polyfasciculations.

tinct narrowing and flattening of the ulnar nerve in zone 2 directly beneath the distal edge of the volar carpal ligament. Postoperatively, the patient was placed in a volar splint for 2 weeks and then weaned out of the splint and began early range-of-motion and strengthening exercises. She reported that at 1 week after surgery sensation was dramatically improved, but the hand was still weak and she continued to show a positive Froment's sign.

One month after the procedure, the splint was discontinued, and the patient still demonstrated residual weakness in the ulnar innervated intrinsic musculature but her sensanerve comes off to provide sensation to the dorsal surface of the medial hand. Within the canal, the nerve branches into the superficial branch, providing the hypothenar eminence, the small finger, and the ulnar portion of the ring finger with sensation and the palmaris brevis with motor function. The deep branch of the ulnar nerve provides the hypothenar muscles, interossei, the third and fourth lumbricals, and the adductor pollicis with motor function. Compared with the median nerve in the carpal tunnel, the ulnar nerve is more prone to injury by direct external compression in Guyon's canal, because of its more superficial location.⁶ The area of the transverse carpal ligament covering the canal is also thinner than the portion of the ligament covering the carpal tunnel.¹

Specific patterns of ulnar nerve compression syndromes are classified by the presence or absence of motor and sensory symptoms. A frequently cited classification system is that of Shea and McClain,⁶ who divided the lesions into 3 categories (Table). They found that type II lesions were the most common (52%), while 30% of lesions were type I, and 18% were type III. The patient in the case we report displayed what these investigators classified as a type I lesion. Most of the case reports about cyclists experiencing ulnar nerve compression syndrome concern patients with either a type II or type III lesion. In one case report of a type I lesion in a cyclist, ³ the patient was noted to have required only 4 days of conservative therapy to recover from mixed motor and sensory symptoms.

Woischneck reported a similar case of a patient who developed bilateral ulnar tunnel syndrome after prolonged mountain biking and had all of the symptoms improve over time with nonoperative measures.⁵ Capitani discussed a case of bicycle-induced UTS in which there was motor involvement only.⁷ For treatment of UTS, a recent report recommended that operations not be performed on patients with ulnar neuropathy, as symptoms in the patients all resolved quickly.⁵ A prospective study on cyclists' UTS published in 2003 found that 23 of 25 cyclists tested experienced motor or sensory symptoms or both.⁸ Eight of the 25 had type II lesions (32%), with type II lesions distributed equally betwen mountain bikers and road cyclists. Conservative therapy was recommended.

CONCLUSIONS

Given the increasing interest in cycling and triathlons, orthopedic surgeons should be prepared to see more cases of ulnar nerve compression syndrome. This case report presents evidence that contradicts the contention that the symptoms of UTS will universally resolve with nonoperative treatment, and it demonstrates that good results can be achieved with surgical intervention in selected cases. As with other compressive neuropathies, surgical intervention might be considered for those patients refractory to appropriate splinting and activity modification that remove further insult to Guyon's canal. We feel that although a patient with UTS may very well respond to conservative treatment, surgical intervention is appropriate if symptoms continue or worsen. Furthermore, a greater sense of urgency should attend consideration of surgical decompression when there is unremitting numbness or progressive motor involvement.

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