

CLINICALLY SPEAKING

Acute-Onset Footdrop Caused by Intraneural Ganglion Cyst of the Common Peroneal Nerve

The Effects of Extraneural Pressure Gradients on Cyst Propagation

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Ganglion cysts are relatively common entities, but intraneural ganglia within peripheral nerves are rare and poorly understood. We present a case of a 51-year-old man who presented with acute left dropfoot. Initial magnetic resonance imaging (MRI) was misinterpreted as common peroneal neuritis consistent with a traction injury corroborated by the patient's history. However, after surgical decompression and external neurolysis were performed, the patient's symptoms worsened. Repeated MRI revealed an intraneural ganglion cyst of the common peroneal nerve with connection to the superior tibiofibular joint by means of its anterior recurrent branch that was evident retrospectively on preoperative MRI. It is crucial to carefully inspect atypical cases to further recognize and appreciate the dynamic aspect of this disease or "roller-coaster" phenomenon. Intraneural ganglion cysts rely heavily on intraneural and extraneural pressure gradients for propagation, which can be drawn from the expanded work of the unifying articular theory. This report emphasizes the importance of understanding the pathoanatomical and hydraulic factors to appropriately identify and treat intraneural ganglion cysts. Increased recognition of this pathologic entity as a differential diagnosis for acute onset dropfoot is also highlighted. (J Am Podiatr Med Assoc 112(2), 2022)

The common peroneal nerve originates as a branch of the sciatic nerve within the popliteal fossa. It courses distally inferolaterally medial to the biceps femoris tendon, to which it then courses laterally around the fibular head. It then passes through the peroneal tunnel between the insertion of the peroneus longus muscle and the fibula. After its exit, before dividing into its terminal superficial and deep branches, the common peroneal nerve supplies a small articular branch to the superior tibiofibular joint.

Ganglion cysts are relatively common entities, but intraneural ganglion cysts within peripheral nerves are rare and poorly understood. Since its first description by Beauchêne¹ in 1810, more than 120 small case series and reports have been described in literature.^{1,2} The pathogenesis of such lesions, however, has remained highly controversial. It has been historically divided into three

theories: degenerative, synovial, and tumoral.³⁻⁵ However, recent work by Spinner and colleagues⁶⁻⁹ proposed a unified theory that encompasses features of all three previously proposed hypotheses to serve as an explanation for this clinical entity.

With postoperative recurrence rates as high as 10% to 20%,^{6,10,11} an appreciation for the proposed pattern of fluid dynamics and propagation is crucial to early recognition and operative treatment. Although these cystic lesions are classically described as static in nature, the origin and extension of intraneural ganglion cysts are dynamic. Their formation and extension rely heavily on intraarticular and extraneural pressure gradients.⁸ Factors such as exercise, postures, and other variables have been documented to increase joint pressure and accentuate the visualization of intraneural ganglion cysts on magnetic resonance imaging (MRI).¹² In addition, these cysts demonstrate size changes, either shrinking or expanding over time in concordance with the above factors.¹³ We present a unique case in which an occult common peroneal intraneural ganglion cyst was not initially recognized on preoperative MRI. It was not until surgical decompression and external neurolysis were performed that repeated postoperative MRI revealed a frank

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intraneural ganglion cyst, demonstrating the effect of extraneural pressure gradients on the accentuation of cyst propagation.

Case Report

A 51-year-old man with a medical history significant for depression and anxiety presented in June of 2019 with a 2-month history of left dropfoot. The patient recalled that the onset of symptoms followed stretching of the posterior leg muscles, where he then experienced sharp pain and paresthesias to the lateral knee and subsequent gait abnormality. On physical examination, there was complete paralysis of the dorsiflexors; however, the evertors were preserved. There was appreciable hypotrophy of lateral compartment leg muscles. Tinel sign was elicited 2 cm distal to the fibular head. There was no sensory deficit in the distribution of the superficial or deep peroneal nerve. No palpable mass or tenderness was appreciated along the fibular head.

Plain radiographic films of the left knee were unremarkable. Muscle paralysis and paresthesias prompted MRI, which revealed soft-tissue swelling surrounding the common peroneal nerve from the level of the distal femoral diaphysis and superior aspect of the popliteal fossa distally to the level of the fibular neck (Fig. 1). At the time, it was unclear whether the nerve was thickened because of intrasubstance fluid or whether this represented slow flow or thrombosis of the adjacent vein (Fig. 1). At that time, the patient was diagnosed with common peroneal neuritis consistent with a traction injury corroborated by the patient's history. He was previously treated with two rounds of corticosteroid injections without relief of symptoms and elected to proceed with surgical intervention.

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The patient was surgically decompressed with external neurolysis of the common peroneal nerve. One and 3 weeks postoperatively, the patient was seen in the office for evaluation and found to have no improvement in muscle strength to the dorsiflexor compartment. Given inadequate clinical progression at this time, electromyography and nerve conduction velocity testing were performed, revealing the absence of motor units that could be activated at the level of the tibialis anterior and extensor digitorum brevis muscles, and normal motor units with normal recruitment of the medial head of the gastrocnemius, vastus medialis, and biceps femoris on the same side.

Magnetic resonance imaging was repeated, which clearly demonstrated a homogenous, multilobulated

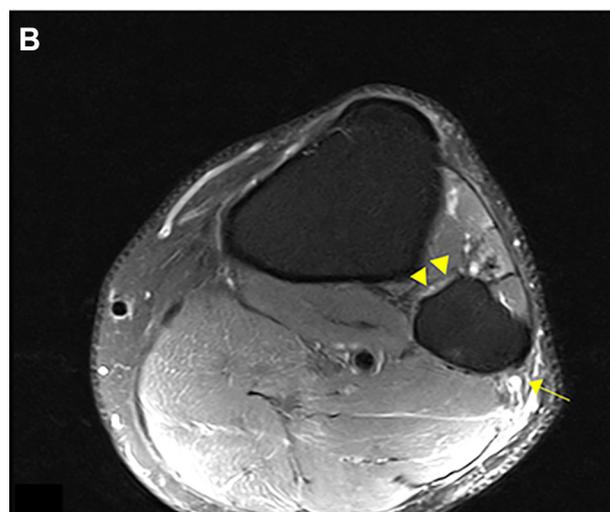
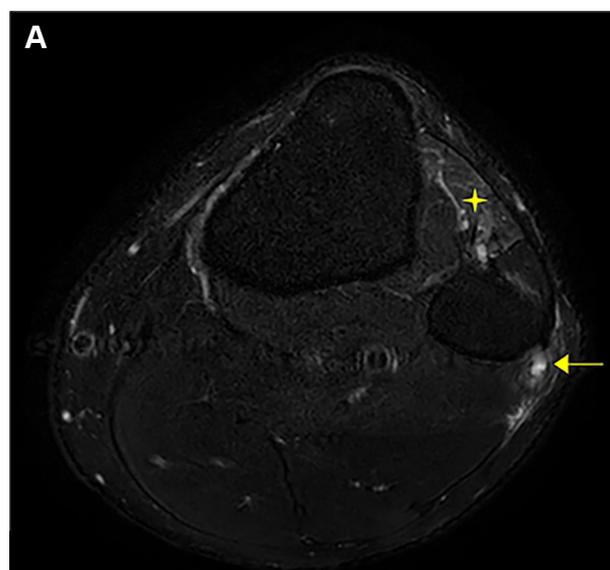


Figure 1. Initial magnetic resonance images obtained at presentation. (A) A T2-weighted fast spin echo Dixon axial image demonstrating cystic changes surrounding the common peroneal nerve (arrow) at the level of the peroneal neck with denervation changes to the tibialis anterior muscle belly (asterisk). (B) A T2-weighted turbo spin echo axial image at the level of the superior tibiofibular joint showing subtle contrast within the common peroneal nerve (arrow) and the joint (arrowheads).

cystic lesion, measuring 4.8 cm from proximal to distal and 1.5×1.9 cm transverse at the most proximal aspect within the common peroneal nerve (Fig. 2A). There was associated edema without significant fatty replacement of the anterior compartment musculature (Fig. 2B). Of note, the distal

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portion of the cyst narrowed at the site of origin, which appeared to be the anterior recurrent branch of the common peroneal nerve to the ventral synovial lining of the superior tibiofibular articulation (positive “tail sign”) (Fig. 2C). Diagnosis was made of interval marked progression and frank development of an intraneural ganglion cyst of the common peroneal nerve.

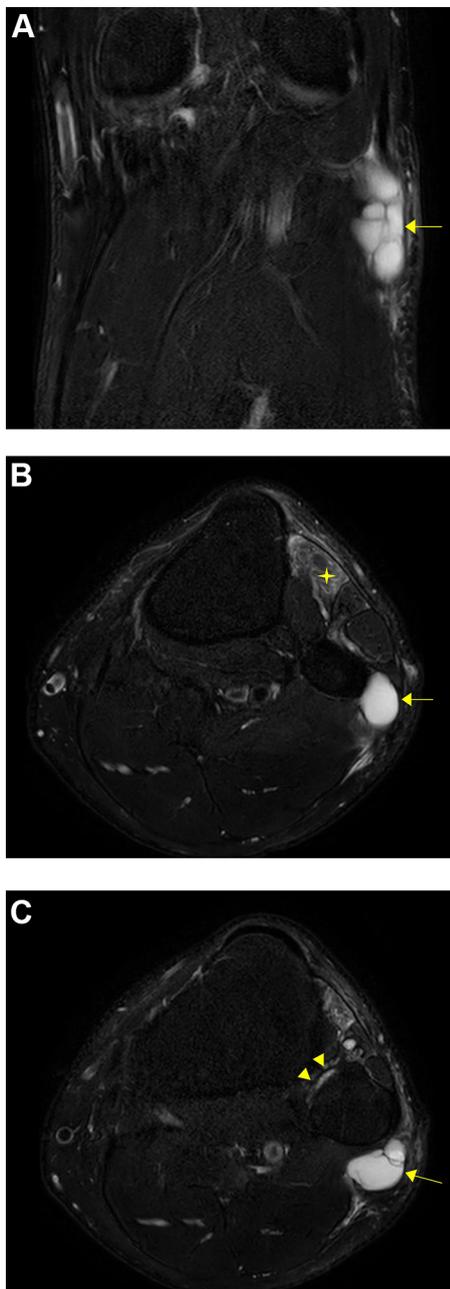
During reoperation, using standard microsurgical techniques, the nerve was carefully followed and dissected to the tibiofibular joint, and the

communicating articular branch was discovered, thus confirming MRI findings. The articulating trunk was transected and evacuated of the ganglion cyst, which extended into the joint. The distal portion was then ligated with silk suture. Intraoperative neurophysiologic monitoring was used throughout the procedure. After resection, both the superficial and deep peroneal nerve were stimulated at 10 mA, revealing complete function of all nerve fibers.

After the reoperation, the patient still exhibited weakness of the dorsiflexor compartment 4 weeks postoperatively. The patient was enrolled in physical therapy, where electrical stimulation was performed to preserve muscle function. One year postoperatively, sensation to pinprick and light touch returned to the common peroneal nerve distribution. The dorsiflexor compartment muscles regained full strength with the assistance of physical therapy and electrical stimulation. The patient now ambulates normally without the use of an ankle-foot orthosis.

Discussion

Although ganglion cysts are relatively common entities, intraneural origin of peripheral nerves has been rarely described in the literature. Peripheral nerve involvement was first described by Beauchêne in 1810 involving the median nerve.¹ It was not until 1921 that Sultan recounted the first case involving the peroneal nerve.¹⁴ Since then, our knowledge and understanding of the etiology of intraneural ganglion



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Figure 2. Subsequent magnetic resonance images obtained 3 weeks after decompression and external neurolysis of the common peroneal nerve revealing progression and frank development of a large intraneural ganglion cyst. (A) A T1-weighted coronal image showing the multilocular cyst measuring 4.8 cm from proximal to distal and as much as 1.5 × 1.9 cm transversely. (B) A T2-weighted fast spin echo axial image with fat suppression demonstrating an intraneural cyst (arrow) with denervation changes in the anterior compartment musculature (asterisk). (C) A T2-weighted fast spin echo axial image with fat suppression revealing multilocular intraneural cyst, including a signet ring sign (arrow) and narrowing at its site of origin to the anterior recurrent branch of the common peroneal nerve to the ventral synovial lining of the tibiofibular articulation (“tail sign,” arrowheads).

has expanded dramatically, most notably by the work of Spinner and colleagues.⁶⁻⁹

In this report, we describe a unique case in which an occult common peroneal intraneural ganglion cyst was not initially recognized and diagnosed on preoperative MRI. After surgical decompression and external neurolysis, postoperative MRI revealed a frank intraneural ganglion cyst, demonstrating the effect of extraneural pressure gradients on the accentuation of cyst propagation.

The etiology of intraneural ganglion cysts was historically controversial because of its poorly understood pathogenesis. Many proposed theories explained only one aspect of the pathogenesis leading to the formation of this clinical entity. In the degenerative theory, cysts arise from joint damage, causing metaplasia of periarticular tissues secondary to spindle cell irritation. These spindle cells transform to spheroidal cells filled with mucoid vacuoles that are emitted to the intraneural tissue.³ Other reports describe its degenerative origin from resorption of intraneural hemorrhage secondary to trauma.⁴ The tumoral theory associates cyst formation with nerve sheath tumors.⁵ Antoni type B schwannomas, for example, can undergo cystic degeneration that can become confluent, expressing themselves as intraneural ganglions.³ The synovial theory, which was classically most favored, linked lesions to articular origin based on anatomical evidence. The foundation for this theory rested on intraoperative findings of pedicles articulating the cyst to the joint by means of articular branches of the associated nerve.^{3,11,15} However, each theory proposed offered only partial explanation for this clinical entity. It was not until 2003 that Spinner and colleagues⁶ first described the unifying articular theory to clarify the underlying mechanisms and formation of intraneural ganglia.

Based on shared characteristics of each theory, Spinner et al⁸ described the origination of the cyst from a previously damaged joint, causing dissection of fluid along the communicating articular branch through the least resistant tissue. This theory was further expanded in 2007 to account for the additional effects of pressure gradients and pressure fluxes.⁸ In this study, three cases were presented involving intraneural ganglion of the popliteal fossa in which the patients experienced fluctuating clinical symptoms in relation to the lesion. Serial MRI confirmed spontaneous regression and proximal migration of the ganglia based on their connection to the superior tibiofibular joint. This finding added a dimension to the unifying articular theory: origination and propagation of these lesions are dynamic and respond to articular and extraneural pressure fluxes.

The morphology of intraneural cysts is constantly changing in concordance with pressure gradients within the intravascular and extraneural compartment, and the superior tibiofibular joint. The life cycle of this lesion, by shrinking and growing over time, demonstrates a “roller-coaster” phenomenon in terms of size and morphology.¹³ In many cases, subtle imaging findings that are evident are easily missed unless there is suspicion of such disease prior. However, peroneal intraneural ganglia are increasingly being acknowledged as a prevalent cause of peroneal neuropathy, reported in as many as 18% of cases.¹⁶ In our case, the evidence of an intraneural ganglion was missed on preoperative imaging. However, on retrospective examination, closer inspection of these films revealed occult cystic changes to the common peroneal nerve.

This case strengthens the argument for the dynamic articular theory and the roller-coaster phenomenon. While taking into consideration the fact that the pathogenesis of this entity is an active process, it is essential to take other factors into consideration when determining its etiology. This could include intraarticular or stromal pressure fluxes over time. Through thorough postoperative physical examination and imaging studies, an intraneural ganglion cyst of the common peroneal nerve was subsequently identified. Operative interventions such as surgical neurolysis and decompression can also alter pressure dynamics that determine the size and morphology. As demonstrated in this case, the recurrent cystic lesion exhibited a different size and morphology than the initial image.

In patients who present with fluctuating deep peroneal nerve predominant symptoms, including denervation changes to the tibialis anterior muscle manifesting clinically as acute footdrop, peroneal intraneural ganglion should be included in the differential diagnosis. It is important to carefully inspect atypical cases to further recognize and appreciate this dynamic pathologic entity. This report emphasizes the importance of understanding the pathoanatomical and hydraulic factors to appropriately identify, diagnose, and treat intraneural ganglion cysts.

Conclusions

Intraneural ganglion cyst of the common peroneal nerve is a rare cause of footdrop. Its pathogenesis has been highly controversial, and three theories have classically been postulated, including the degenerative, tumoral, and synovial theories. However, based on careful examination of these intraneural cysts through clinical, imaging, and operative findings,

Spinner and colleagues⁶⁻⁹ proposed a unifying articular theory to account for their origin. It is important to recognize the dynamic nature of these lesions, as both articular and extraneural pressure fluxes contribute to their progression. Without an appreciation of such fluid dynamics, these peripheral nerve lesions can be misdiagnosed or underdiagnosed, leading to debilitating consequences if unrecognized. These clinical features can also be implicated intraoperatively to prevent recurrence.

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Conflict of Interest: None reported.

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