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Sonographic Appearance of Periprocedural Popliteal Fossa Nerve Injuries

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Background: Peripheral nerve injuries can occur perioperatively and postoperatively. While ultrasound cannot determine etiology, it helps evaluate suspected peripheral nerve injury. This paper describes sonographic findings in 2 patients who developed peripheral nerve injuries and subsequent complex regional pain syndrome type II following lower extremity surgery with popliteal catheter placement.

Case Report: Case 1: A 53-year-old woman developed neuropathic pain after lower extremity surgery with popliteal catheter placement. Ultrasound performed at 6 weeks revealed disrupted fascicular architecture, altered echogenicity, and enlargement of the right fibular and tibial nerves, each more than twice the cross-sectional area of the corresponding contralateral nerve. She was subsequently diagnosed with complex regional pain syndrome type II.
Case 2: A 27-year-old man developed neuropathic pain and paresthesia following lower extremity fixation with popliteal catheter placement. Ultrasound performed at 9 weeks demonstrated distorted fascicular architecture, altered echogenicity, and enlargement of the right sciatic, tibial, and fibular nerves compared with the corresponding contralateral nerves. He was subsequently diagnosed with complex regional pain syndrome type II.

Conclusions: Ultrasound serves as a valuable adjunct to electrodiagnostic testing for comprehensive peripheral nerve injury evaluation.

Key words: Ultrasonography; peripheral nerve injury; electrodiagnostic testing; complex regional pain syndrome; sciatic nerve; case series

BACKGROUND

Peripheral nerve injuries (PNIs) can arise from various perioperative and postoperative factors, and identifying the underlying etiology is often challenging. Continuous catheter-based analgesia represents one of several potential contributors to postoperative neurologic symptoms, whether through mechanical trauma, local anesthetic chemical toxicity, or both (1-8). Anatomic variability, preexisting conditions, surgical considerations, and other periprocedural factors may also increase risk (5,8,9). Current standard PNI assessment involves electrodiagnostic testing, including nerve conduction studies (NCS) and electromyography (EMG) (10,11). However, these modalities cannot always definitively localize lesions, characterize structural abnormalities, or reliably detect all peripheral nerve injuries because of limited sensitivity and specificity (12).

Ultrasound has been increasingly recognized as a valuable adjunctive tool, enabling dynamic evaluation of nerve characteristics to help localize the level of pathology and inform further workup (13,14). Sonographic features of peripheral nerve pathology have been described in contexts such as trauma, entrapment, and infection (15), yet detailed characterization

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of periprocedural nerve injury following operations requiring continuous catheter-based popliteal blocks remain limited.

In this context, we describe sonographic findings of PNI and discuss their correlation with electrodiagnostic studies and the patients' clinical courses, acknowledging that the underlying etiology remains unclear. These cases highlight the potential role of ultrasound in early evaluation of suspected PNI. Written consent was obtained from both patients.

CASE REPORT

Case 1

A 53-year-old woman presented 6 weeks postoperatively with new-onset right foot neuropathic pain following a traumatic right proximal phalanx fracture treated with open reduction and internal fixation without intraoperative complication. Before surgery, she received an ultrasound-guided right saphenous single-shot nerve block via the adductor canal using a 21-gauge block needle, followed by an ultrasound-guided right sciatic continuous nerve block in the popliteal fossa using an 18-gauge block needle and 20-gauge catheter placed with a catheter-over-needle technique for ropivacaine administration.

The patient reported no pain following catheter placement. The catheter was removed on postoperative day 4, after which sensation began to return to her right leg, and she noted significant pain localized to the popliteal fossa. Over the next week, the pain intensified into moderate-to-severe burning dysesthesia with numbness involving the lateral calf and the plantar and dorsal surfaces of the foot and toes. The patient denied motor changes but reported persistent lower extremity edema and discoloration. At 6 weeks postoperatively, the orthopedic hardware was removed because of ongoing symptoms; however, no improvement was noted, prompting referral to the peripheral nerve clinic for further evaluation.

At the peripheral nerve clinic evaluation, physical examination demonstrated right lower extremity weakness limited by pain. Sonographic assessment revealed abnormal findings at the level of the popliteal fossa; the right common fibular nerve demonstrated significant focal enlargement and prominent fascicles, with increased cross-sectional area (CSA) beyond the upper limit of normal. The right tibial nerve, although within the normal CSA range, demonstrated a significant side-to-side difference beyond the upper limit of normal (Table 1). CSA measurements and appearance were normal in the right tibial and common fibular nerves immediately distal to the popliteal fossa. The right superficial fibular and sural nerve branches demonstrated normal CSA throughout

Table 1. Side-to-side cross-sectional area comparison of lower extremity nerve ultrasound.

Nerve	Site	Patient 1 Data		Patient 2 Data		Normative Data*		
		CSA Left-sided (mm^2)	CSA Right-sided (mm^2)	CSA Left-sided (mm^2)	CSA Right-sided (mm^2)	Mean (mm^2)	Reference range (mm^2)	Side-to-side difference upper limit (mm^2)
Ciatic	Distal thigh	-	-	37	62	52.6	24.6-80.6	18.9
Common Fibular	Popliteal fossa	11	23	7	12	11.7	2.5-20.9	9.5
	Fibular head	14	17	8	11	11.2	4.6-17.8	4.9
Tibial	Popliteal fossa	19	42	-	5	35.3	14.7-55.9	15.7
	Poximal calf	-	22	25	40	25.3	10.7-39.9	10.8
	Ankle	-	-	-	22	13.7	5.1-22.3	5.7
Sural	Distal calf	4	3	8	18	5.36	1.7-8.9	2.6
Superficial Fibular	Ankle	3	2	-	4	-	-	-

CSA measurements of both patients shown, alongside normative means, reference ranges, and upper limits for side-to-side differences. Bolded values indicate abnormalities. Dashed lines indicate sites where measurement was not obtained.

*Normative data reference: Cartwright MS, Passmore LV, Yoon JS, Brown, ME, Caress JB, Walker FO. Cross-sectional area reference values for ultrasonography. Muscle Nerve. 2008 May; 37(5) 566-71. doi: 10.002/mus.21009. PMID: 18351581.

their course (Fig. 1). EMG was not performed because of the patient's severe hypersensitivity.

The patient was subsequently referred to pain management, where she met the Budapest Criteria for CRPS. A diagnosis of CRPS type II of the right lower extremity was made based on her clinical history and suspected underlying nerve involvement, with available ultrasound findings suggestive of nerve injury (Fig. 1) and electrodiagnostic findings providing additional diagnostic support. At 1 year postoperatively, she continued to experience significant symptoms despite extensive trials of multiple pharmacologic and nonpharmacologic modalities.

Case 2

A 27-year-old man presented 9 weeks postoperatively with severe right lower extremity neuropathic pain after a displaced closed right calcaneus fracture, status post closed reduction and internal fixation without intraoperative complication. Before surgery, he received an ultrasound-guided right saphenous single-shot nerve block via the adductor canal using a 21-gauge block needle, followed by ultrasound-guided placement of a right sciatic continuous nerve block catheter in the popliteal fossa using a 21-gauge block needle and 24-gauge catheter with a catheter-over-needle technique for ropivacaine administration. He reported paresthesia throughout the distal right lower extremity immediately following block placement. The continuous nerve block catheter was removed on postoperative day 5.

Over the next 8 weeks, the patient developed progressive neuropathic pain, cold sensations, numbness, and weakness of his distal right lower extremity. He was subsequently referred for peripheral nerve clinic evaluation and underwent nerve ultrasound at 9 weeks postoperatively, followed by electrodiagnostic testing at 11 weeks.

Ultrasound revealed enlarged fascicles and a partially hypoechoic appearance of the right sciatic nerve, with significantly increased CSA at the distal thigh just proximal to the bifurcation in the popliteal fossa compared with the contralateral side at the same level (Fig. 1). The right tibial nerve demonstrated borderline increased CSA at the popliteal fossa compared with the contralateral location, with prominent fascicles that normalized in size and appearance approaching the proximal calf. At the ankle, the tibial nerve CSA was more than twice that of the left side. The right common fibular nerve demonstrated mild hypoechogenicity and increased CSA at the popliteal fossa compared with the left common fibular nerve at the same location, normalizing in size and appearance at the fibular head (Table 1). The right superficial fibular, right sural, left sciatic, left tibial, and left common fibular nerves were normal in size.

NCS and EMG performed at 11 weeks revealed severe,

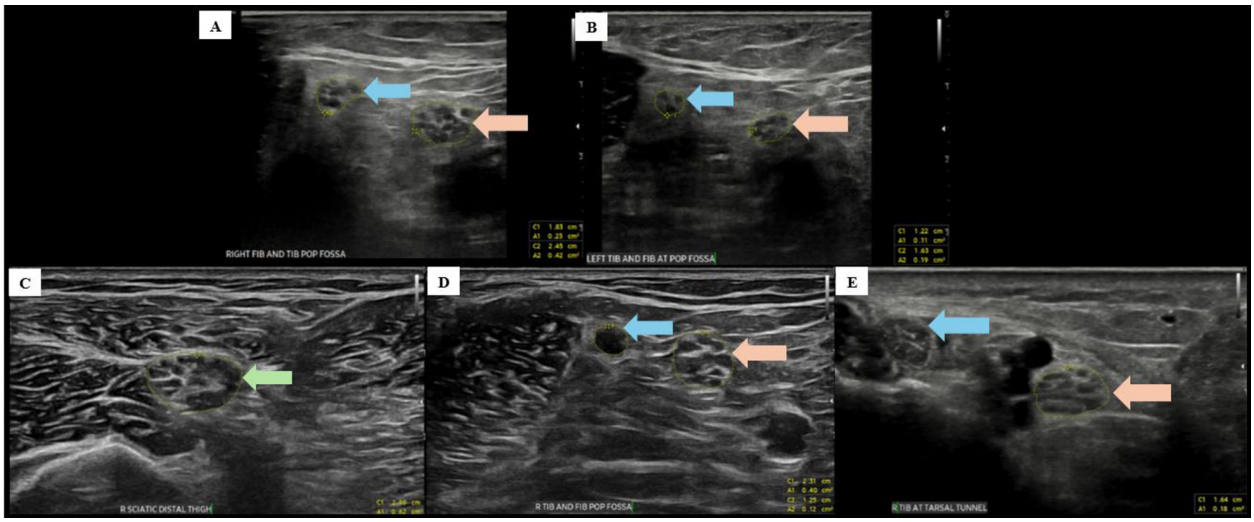


Fig. 1. Ultrasound images of the lower extremity distal to the popliteal fossa in patient 1 (A/B) and patient 2 (C/D/E). (A) Abnormal right tibial (orange arrow) and common fibular (blue arrow) nerves showing enlarged, hypoechoic fascicles. (B) Normal left tibial and fibular nerves with typical hyperechoic fascicles. (C) Right sciatic nerve (green arrow) at the distal thigh demonstrating enlarged, partially hypoechoic fascicles, with increased CSA proximal to the bifurcation. (D) Right tibial nerve with enlarged fascicles and increased CSA in the popliteal fossa; hypoechoic right fibular nerve with increased CSA. (E) At the ankle, the right tibial nerve exhibits increased CSA; the right superficial fibular nerve appears normal.

acute sensorimotor axonal neuropathies of the right tibial and fibular nerves (Fig. 2). The patient was ultimately referred to pain management and diagnosed with CRPS type II. Although symptom onset temporally coincided with catheter placement, a direct catheter-related injury could not be established. Potential contributing factors included local anesthetic toxicity, mechanical compression, and the inciting trauma or surgical intervention. The precise etiology therefore remained unclear, although the timing suggested a perioperative injury

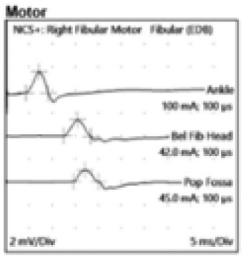
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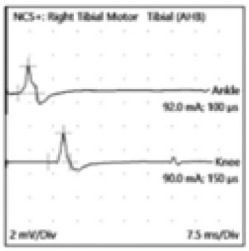
Site	Onset (ms)	Onset Normal	O-P Amplitude (mV)	O-P Amplitude Normal	Duration (ms)	Area (ms*mV)	Temperature (°C)
Right Fibular (EDB) Motor							
Ankle	4.3	<6.0	*1.84	>2.0	4.9	4.7	32.7
Below Fibular Head	12.5	-	1.44	-	5.8	4.3	32.6
Popliteal Fossa	14.5	-	1.04	-	5.5	3.0	32.7
Right Tibial (AHB) Motor							
Ankle	3.8	<6.5	*2.3	>3.0	5.9	4.9	22.1
Knee	13.4	-	2.4	-	6.8	4.1	21.8

Legend: O-P = Onset-Peak; ms = milliseconds; mV = millivolts; °C = Celsius; EDB = extensor digitorum brevis; AHB = abductor hallucis brevis

Segment	Delta-Onset (ms)	Distance (cm)	Conduction Velocity (m/s)	Normal
Right Fibular (EDB) Motor				
Below Fibular Head-Ankle	8.2	35	43	>41
Popliteal Fossa-Below Fibular Head	2.0	8.5	43	>38
Right Tibial (AHB) Motor				
Knee-Ankle	9.6	41	43	>41

Legend: ms = milliseconds; cm = centimeters; m/s = meters/seconds; EDB = extensor digitorum brevis; AHB = abductor hallucis brevis

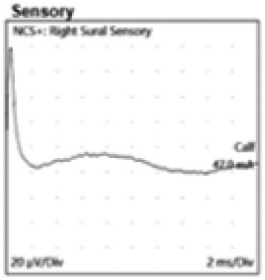




B

Site	Onset (ms)	Onset Normal	Peak (ms)	Peak Normal	O-P Amplitude (µV)	O-P Amplitude Normal	Temp °C	Distance cm	CV m/s	CV Normal
Right Sural Sensory										
Calf-Lateral Malleolus	*NR	-	*NR	<4.5	*NR	>6	32.6	14	*NR	-

Legend: ms = milliseconds; µV = microvolts; °C = Celsius; cm = centimeters; m/s = meters/seconds; NR = non-reactive



C

Muscle	Nerve	Root	Insertional Activity	Fib/PSW	Fasciculations	Amplitude	Duration	Polyphasia	Recruitment
Biceps Femoris Short Head	Sciatic	L5-S1	Nml	Nml	None	Nml	Nml	Nml	Nml
Gastrocnemius Medial Head	Tibial	S1-S2	Nml	*3+	None	Nml	Nml	Nml	Nml
Tibialis Anterior	Fibular	L4-L5	Nml	*3+	None	Nml	Nml	Nml	Nml

Legend: Fib/PSW = fibrillations/positive sharp waves; Nml = normal

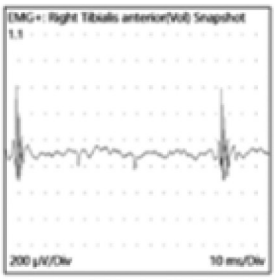


Fig. 2. Electrodiagnostic results of patient 2, including (A) motor nerve conduction studies (NCS), (B) sensory NCS, and (C) electromyography (EMG) results. (A) NCS revealed normal distal latencies and reduced amplitudes in the right fibular motor response to the extensor digitorum brevis and right tibial motor response to the abductor hallucis, with normal conduction velocities throughout. (B) The right sural sensory response was absent. (C) Needle EMG of the right tibialis anterior and medial gastrocnemius showed abnormal spontaneous activity and limited motor unit analysis because of pain-limited activation. Overall, there is evidence of severe, acute sensorimotor axonal neuropathies of the right tibial and fibular nerves, with normal EMG of the biceps femoris short head, localizing the injury distal to its branch point.

rather than a pre-existing or delayed process. At 1 year postoperatively, he continued to experience significant symptoms despite trials of multiple pharmacologic and nonpharmacologic modalities.

DISCUSSION

Iatrogenic PNI is rare but can be functionally disabling and painful. This paper describes 2 patients who developed nerve deficits following lower extremity surgery with popliteal catheter placement, although the underlying etiology of their nerve injuries remains unclear. In both cases, ultrasound served as an adjunct to comprehensive physical examination, allowing visualization and early detection of peripheral nerve pathology and highlighting its diagnostic potential in early PNI evaluation.

Although NCS and needle EMG remain the gold standard for assessing PNIs (11), additional information is often required to further understand etiology and structural abnormalities. Electrodiagnostic testing can also be uncomfortable, particularly for individuals with CRPS, time-consuming, and prone to technical variability (3,16). In contrast, ultrasound is low cost, portable, and dynamic, offering immediate visualization of nerve anatomy and precise localization of pathology (3,13,17). Ultrasound involves no radiation, has virtually no contraindications, permits quick contralateral comparisons, minimizes metal artifacts that can hinder magnetic resonance imaging, and provides real-time guidance for interventional procedures (16). Ultrasound can also identify characteristic PNI pathologies before electrodiagnostic changes appear (11,16). However, ultrasound is limited by operator skill, technical variability, and bone or hardware, which can limit penetration to deep tissue and obstruct the view (16). Therefore, electrodiagnostic testing and ultrasound serve complementary roles in the comprehensive evaluation of suspected PNI.

The correlation between ultrasound findings and Sunderland classification highlights its potential value in estimating injury severity. Normal sonographic appearance of an uninjured peripheral nerve resembles an "insulated electrical cable," with fascicles surrounded by a hyperechoic epineurial rim (16). Longitudinal-axis imaging also assesses continuity and architectural changes. Injured nerves may demonstrate features of demyelination and structural disruption, including fascicular swelling, side-to-side variability, loss of echogenicity with hypoechoic fascicles surrounded by a hyperechoic rim, increased Doppler signal, irregular fascicular patterns with loss of the classic "honeycomb" appearance, or discontinuity (18). These sonographic characteristics, considered together with electrodiagnostic findings, may provide an estimate of injury severity corresponding to the Sunderland classification (19,20). Both cases demonstrated ultrasound findings suggestive of a Sunderland grade II–III injury (Supplementary Table 1). Because Sunderland grading is histologically defined, these classifications represent interpretive estimates based on imaging and electrodiagnostic findings rather than formal histopathologic diagnoses.

By identifying discontinuity or fascicular distortion noninvasively, ultrasound can help stratify patients for conservative versus surgical management. Integrating ultrasonography with electrodiagnostic data may provide a more comprehensive framework for early PNI classification, especially when magnetic resonance neurography is unavailable or contraindicated.

In both cases, ultrasound identified structural nerve pathology and contributed to the evaluation of potential secondary pain syndromes (21,22). CRPS type I occurs without a distinct nerve injury, whereas CRPS type II is associated with a distinct nerve injury. Ultrasound has been used to assess intramuscular changes associated with CRPS type I and nerve changes in CRPS type II (21). However, no ultrasound-based standard of care exists for diagnosing CRPS type II; diagnosis is primarily clinical using criteria such as the Budapest criteria (22).

Advances in the clinical use of ultrasound for early PNI assessment may potentially translate to earlier CRPS type II recognition and treatment.

Limitations

This case series has limitations. The retrospective design resulted in incomplete or unobtainable details regarding the patients' clinical courses. Additionally, these cases came from a single institution, which may limit generalizability because other institutions may lack the resources or specialized expertise of PNI diagnostic or surgical teams. This may also affect the accuracy of findings because ultrasound performance and assessment are operator dependent.

CONCLUSIONS

These findings have implications for clinical systems and patient care. Incorporating ultrasound into early evaluation as an adjunct to electrodiagnostic testing may expedite diagnosis and inform referrals to appropriate specialists. Shortening diagnostic delays may

help guide management and improve rehabilitation outcomes.

Future work should focus on developing standardized ultrasound protocols and grading criteria for PNI. Prospective multicenter studies could help clarify diagnostic accuracy and define optimal timing for ultrasound relative to electrodiagnostic testing.

Author Contributions

SHV: conceptualization, investigation, methodology, data curation, writing, review, editing, visualization.

KZ: conceptualization, investigation, methodology, data curation, writing, review, editing, visualization.

YKL: review, editing, supervision.

SMS: review, editing, supervision

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Supplementary Table 1.

Sunderland Classification	Injury Description	Ultrasound Findings
Grade I: Neurapraxia	Focal segmental demyelination without axonal damage	No pathologic findings visualized on ultrasound, or only mild swelling of the nerve with an intact fascicular (honeycomb-like) pattern.
Grade II: Axonotmesis	Axonal damage with intact endoneurium	Enlarged CSA of affected fascicles and nerves at the lesion site due to axonal edema. The nerve may appear mildly bulky. Mildly altered echotexture, but the overall fascicular pattern remains visible and organized.
Grade III: Axonotmesis	Damage to axons and endoneurium with intact perineurium	Loss of normal nerve architecture and echotexture with disrupted internal fascicular pattern. The nerve often appears enlarged and hypoechoic due to internal edema and scarring. It may appear bulky or focally swollen.
Grade IV: Axonotmesis	Axons, endoneurium, and perineurium damaged with intact epineurium	Marked disruption of normal nerve architecture. Fascicular bundles are disorganized and no longer distinct. This is often described as a "neuroma-in-continuity." The nerve is significantly enlarged and hypoechoic, with a loss of the typical "honeycomb" fascicular pattern. The complete disruption of the perineurium causes severe disorganization within the nerve.
Grade V: Neurotmesis	Complete nerve transection	Lesion shows transection of the nerve with a loss of nerve continuity.