

Crystallizing the Diagnosis: Acute Knee Pain After Peripheral Nerve Stimulator Placement: A Case Presentation

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Introduction

Peripheral nerve stimulation (PNS) is an opioid sparing technique utilized in both acute post-surgical and chronic pain. The mechanism, while elusive, is largely thought to be via gate control theory proposed by Melzack and Wall in 1965. This theory suggests that non-noxious stimuli applied to low-threshold, non-nociceptive, large diameter A-beta fibers leads to activation of inhibitory interneurons and inhibition of the nociceptive A-delta and C fibers as well as transmission of pain centrally via the spinal cord [1]. While very effective in some patients, PNS is not universally helpful or without risk [2]. We present a case of acute knee pain after PNS placement to illustrate the diagnostic puzzle in evaluating acute pain in a patient with an implanted stimulator and multiple comorbidities. While pain specialists are often most concerned with infection or hardware related complications, this case illustrates that the differential should remain broad yet patient specific.

Methods

Informed consent for case-report was obtained. IRB review was not required per policy of the institution.

Case Presentation

A 67-year-old male with past medical history of hypertension, diabetes, coronary artery disease, and EMG/NCS-proven diabetic peripheral neuropathy and radicular pain presented for treatment of chronic pain in his bilateral feet. Previous treatments that were either not tolerated or provided partial or no relief included medications (Norco, gabapentin, duloxetine), and lumbar epidural steroid injections.

Case Presentation Continued

The patient did not desire trial and implant of a dorsal column stimulator for treatment of his symptoms, but he was amenable to temporary, percutaneous, PNS. Based on the areas of greatest pain in his feet, ultrasound guidance was used to place PNS leads (Sprint Therapeutics) in proximity to the bilateral, sciatic nerves using supra-popliteal approach. Both leads were set to an intensity of 60 and provided an impressive 50% reduction in pain and ability for the patient to sleep through the night for the first time in “years”. Over the following weeks pain relief was less impressive, and stimulation intensity was increased to 75 bilaterally. It is common for patients to need adjustment of their stimulation parameters during the treatment period, and this did not represent an abnormal increase. However, 4 weeks after implant, the stimulation intensity was increased to 90 and 87 in a period of one week, which is a large increase for such a short interval. At that point, even with increases in stimulation, his pain was only 15% improved compared to baseline. Interestingly, he reported improved sensation in his toes, not present prior to PNS, despite the pain.

On his sixth week of PNS treatment, the patient presented to the emergency department complaining of two days of acutely worsened bilateral, posterior knee pain. He endorsed a “blunt knife”, “stabbing” pain that radiated up the thighs into the groin, precluding ambulation. This was different than his usual leg pain below the knees. The pain was unrelieved by his chronic pain medications, rest, ice, compression, and elevation. Activity was limited in the days leading to the ED visit. He denied trauma to either lower extremity. No fever or redness, warmth, induration, or drainage had been present at his lead insertion sites. Bilateral lower extremity edema was present at the knees and below. Physical examination revealed swollen and tender bilateral knees, with normal sensory and motor function. Lead sites showed mild mechanical irritation at the insertion sites, but no streaking, erythema, or discharge. Laboratory analysis was remarkable for ESR 31 and CRP 30 and slightly elevated uric acid. X-ray of the bilateral lower extremities showed mild degenerative changes but no acute abnormality. Bilateral lower extremity ultrasound was negative for deep vein thrombosis or Baker cysts, but positive for joint effusions. The initial suspicion was for PNS infection versus overstimulation, so the patient was admitted, started on vancomycin and ceftriaxone, and leads were removed. Further discussions with the patient revealed a history of gout, although it had never affected his knees. Bilateral knee joint aspiration produced 45mL of straw-colored fluid. Lidocaine and triamcinolone were also injected by the treating service. Synovial fluid analysis was positive for neutrophils and monosodium urate crystals. Cultures were negative for bacterial growth, though empiric antibiotic therapy had already been initiated. The patient was diagnosed with a gout flare and prescribed colchicine and NSAIDs with resolution of his symptoms.

Table 1: Differential diagnosis of knee pain

Baker cyst	Ligament injury	Pseudogout
Bone injury	Meniscus injury	Reactive arthritis
Bursitis	Nerve entrapment	Referred pain
Deep vein thrombosis	Osteoarthritis	Rheumatoid arthritis
Gout	Popliteal artery aneurysm	Sickle cell
Infectious arthritis	Popliteal artery entrapment	Tumors

Figure 1: Differential diagnosis of knee pain

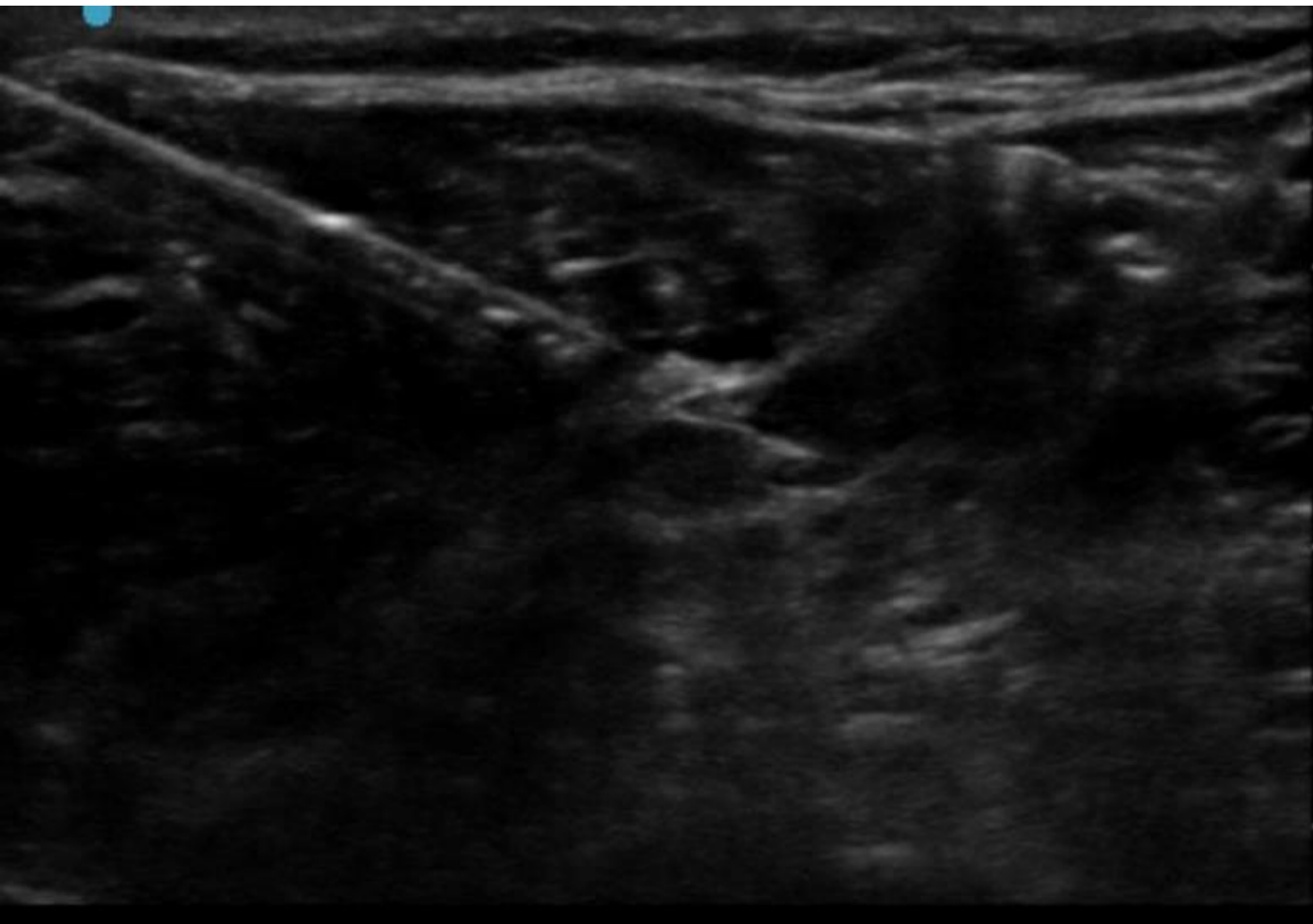


Figure 2: Ultrasound image of needle placement on sciatic nerve, above popliteal fossa on patient left side

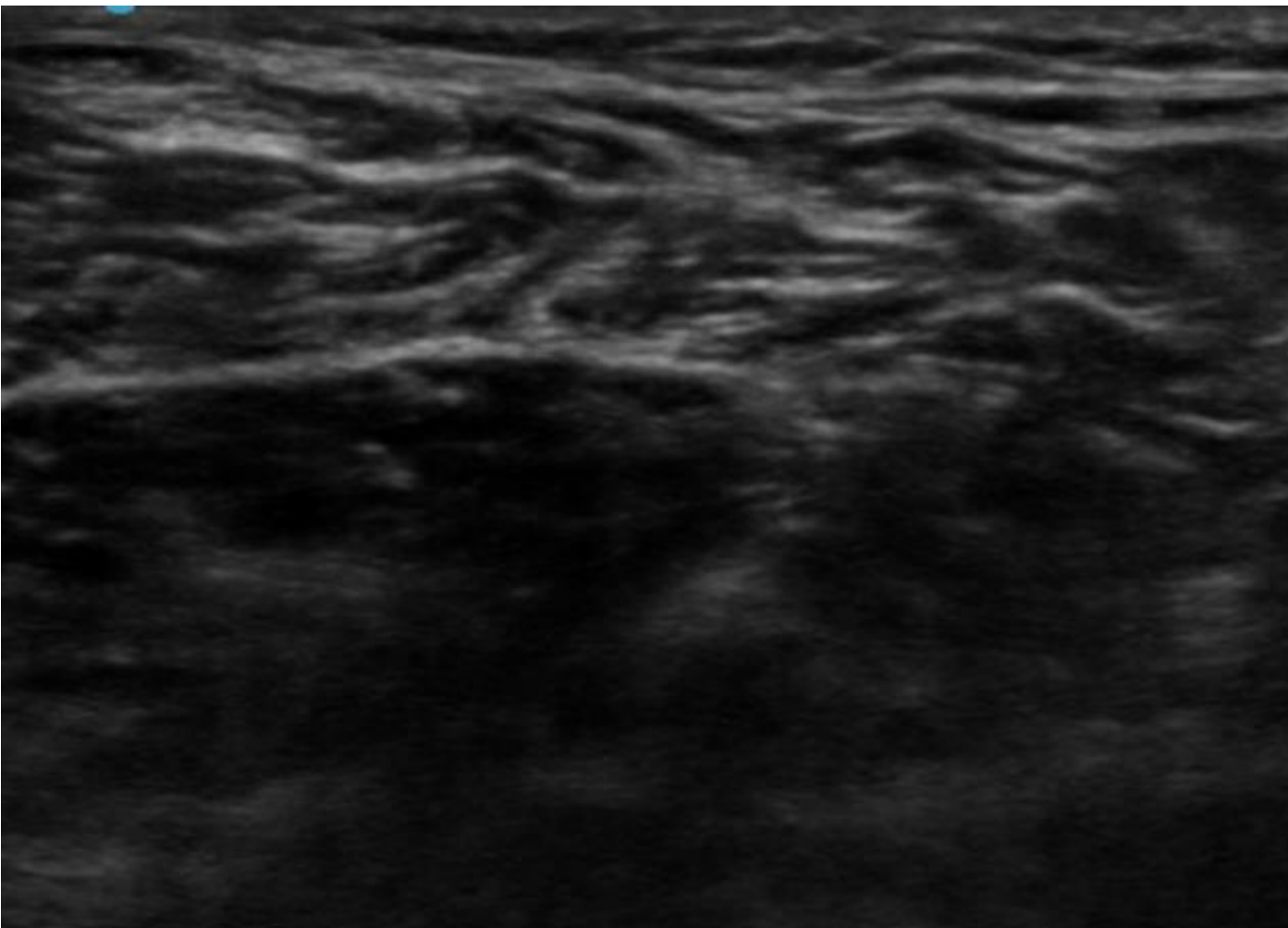


Figure 3: ‘Ultrasound visualization of of Sprint PNS system at level of sciatic nerve, above the popliteal fossa, patient left side.

Discussion

There are many etiologies of knee pain, with varying patterns of pain and presentation [3]. (See table1) While a thorough history and physical examination can usually differentiate or rule out common and rare causes, arriving at the correct diagnosis for acute knee pain after this case of popliteal sciatic PNS placement was more nuanced. Pain specialists may initially be more concerned for a potential complication of the PNS—lead dislodgement, overstimulation, device failure, infection [2]. Several factors blurred the clinical picture and delayed diagnosis in this case: 1) an incomplete past medical history—reviewing the patient’s medication list and asking the patient why he took indomethacin revealed a diagnosis of gout that had been lost in recent medical records, and 2) overlap of findings in his history and physical examination and laboratory markers with multiple etiologies for knee pain. Pain, local swelling, warmth, slight irritation around the lead sites, and elevated inflammatory markers were non-specific, and etiologies that share this constellation of findings include infectious, traumatic, and rheumatologic diagnoses, among other causes. When evaluating pain after PNS placement it is important to consider the patient’s whole clinical picture. This case acts as a reminder to the pain physician to keep a broad differential, including medical diagnoses that are not commonly treated in our specialty.

References

References:

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