

# Ultrasonography-guided pulsed radiofrequency of sciatic nerve for the treatment of complex regional pain syndrome Type II

## ABSTRACT

Although the major mechanism of complex regional pain syndrome (CRPS) involves dysfunctional central or sympathetic nervous system activation, the peripheral nervous system also contributes significantly to its clinical manifestations. Pulsed radiofrequency (PRF) is a recently developed treatment option for neuropathic pain syndromes. Here, we report a case of CRPS Type II after a femur fracture and sciatic nerve injury, in which the pain was treated successfully with ultrasonography-guided selective sciatic nerve PRF application.

**Key words:** Complex regional pain syndrome; pulsed radiofrequency; ultrasonography

## Introduction

Complex regional pain syndrome (CRPS) is a pain syndrome characterized by a combination of sensory, motor, vasomotor, and sudomotor dysfunction together with trophic signs. Among these variable symptoms, the most important clinical manifestation is an intractable pain, which results in limited living activities and decreased the quality of life. Although the exact mechanism of CRPS is not well understood, dysfunctional activation of the central nervous system (CNS) and autonomic nerve system (ANS), including the sympathetic nervous system, is regarded as its major pathogenic mechanism.<sup>[1]</sup> According to this concept, the classic treatment of CRPS is focused on the sympatholytic blockade, whereas treatment targeting the peripheral nervous system (PNS) is not regarded as a fundamental part of its management in current practice. Here, we describe a case of CRPS Type II after sciatic nerve injury caused by a femur fracture in which the pain was

treated successfully by ultrasonography (US)-guided pulsed radiofrequency (PRF) application.

## Case Report

A 43-year-old male was referred to our pain clinic for disabling pain and a tingling sensation in his right foot and calf. Three months earlier, the patient had undergone open reduction and internal fixation with nail pinning of the right femur head and mid-shaft fracture (Pipkin Type I) from a traffic accident. His right foot pain continued after the surgery due to concomitant sciatic nerve injury and indeed progressed.

On the day, the patient was referred to our pain clinic and his pain was at the dorsum, lateral sole, and toes of his right foot and the posterior side of the calf. This pain was of a

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continuous nature and the severity ranged from 6 to 7 on a visual analog scale (VAS) with the intermittent breakthrough pain of VAS 9. He also complained of severe allodynia in the affected limb, especially in his sole, toes, and heel. The severe pain with allodynia made the patient unable to bear his body weight and walk with the affected limb. He could not even walk on the healthy left leg with crutches because the severe allodynia caused him to drag his right foot on the ground. His motor power-ankle plantar flexion and dorsiflexion were assessed as grade 0/5, and his sensory function was impaired in a neurological examination. Other associated features including changes in temperature, sweating abnormalities, edema, trophic changes, and skin color changes were found in a physical examination.

A nerve conduction study and electromyography revealed an absence of sciatic nerve responses in the right leg below the knee area. Integrating the clinical, physical, three-phase bone scintigraphy, digital infrared thermal imaging test, and laboratory findings above, the patient was diagnosed with CRPS Type II according to the diagnostic criteria of the International Association for the Study of Pain.

The patient did not respond to conventional treatments including medications, sciatic nerve blocks, and sympathetic blocks. The dynamic allodynia was so severe that the patient could not receive physical therapy. Thus, we decided to apply PRF to the affected sciatic nerve with ultrasound guidance. Informed consent was obtained from the patient before the procedure.

The sciatic nerve was identified under ultrasonographic guidance just above the bifurcation of the nerve at the popliteal fossa [Figure 1a] and the final location of the needle tip was close to the sciatic nerve in the popliteal fossa [Figure 1b]. A sensory stimulation test was performed. PRF was applied at a temperature of 42°C for 120 s and repeated 4 times. After the procedure, 0.3% ropivacaine (10 mL, mixed with triamcinolone, 8 mg) was injected to prevent peri-neural inflammation.

After the US-guided PRF, the patient's spontaneous pain was decreased to a VAS score of 3. The allodynia and breakthrough pain were also significantly improved after the procedure. The other symptoms and signs were not changed immediately. The patient was able to drag his affected foot on the ground and load his weight on the affected limb temporarily. He was able to walk with crutches and to participate in a rehabilitation program. The effects of PRF were maintained for 8 months after the first application. In the follow-up interval, the VAS score

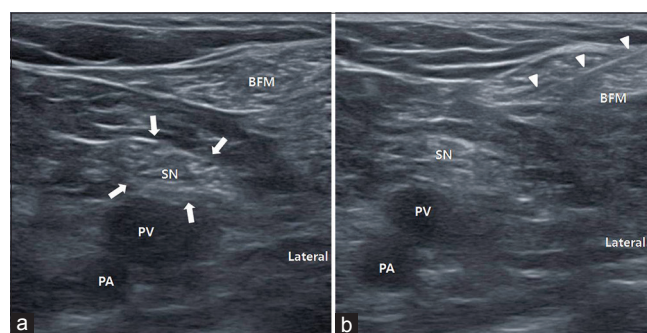
increased to six again. Thus, we applied PRF to the sciatic nerve in the same manner as before, and the VAS score was again reduced to 2–3.

## Discussion

Our case demonstrates that PRF, targeting primarily the PNS, is effective for the management of intractable CRPS Type II. There is no established standard treatment for CRPS to date, reflecting the fact that there is no consistently effective treatment and that treatment responses are often unpredictable. Despite many controversies, most clinicians regard abnormal CNS and ANS activation as the main feature of its pathogenesis. In this hypothesis, continuous nociceptive input due to hypoxia, inflammation, or sympathetic stimulation may lead to sensitization and alterations in the cortical organization of sensory and motor units, inducing CRPS.<sup>[1]</sup>

Based on the current concept of pathophysiological changes in CNS and ANS activation, the classic treatment for CRPS has been sympatholytic blocks, aimed at attenuating the pain and vasoconstriction induced by sympathetic hyperactivity.<sup>[2]</sup> However, this approach is not based on firm clinical evidence, and others have argued that many CRPS patients do not benefit from sympathetic blocks regarding pain management, especially.<sup>[3]</sup>

Many previous reports already demonstrated the usefulness of PRF in the management of peripheral neuropathies but not in CRPS.<sup>[4-6]</sup> The exact mechanism of PRF in the management of neuropathic pain is not well understood. However, the current hypothesis is that PRF generates a strong electromagnetic field around the electrode tip, which could



**Figure 1:** A 12–5 MHz transverse ultrasound image just above the bifurcation of the sciatic nerve at the popliteal fossa. (a) The sciatic nerve (arrows) is just above and lateral to the popliteal vessels. (b) A sono-guided in-plane lateral side approach to the sciatic nerve with the RF needle (arrowheads). A sensory test with 50 Hz stimulation confirmed a satisfactory needle position, evoking paresthesia in the identical area as the preexisting foot pain. BFM: Biceps femoris muscle; PA: Popliteal artery; PV: Popliteal vein; SN: Sciatic nerve

impede the transmission of pain impulses by disrupting the neuronal membrane, thereby interfering with the generation of action potentials and ectopic firings in nociceptive A- $\delta$  and C neurons.<sup>[7-9]</sup> In this regard, we suppose that PRF not only relieved the patient's somatic pain, it interrupted the pathophysiological changes in the CNS and ANS system, described as central sensitization.

In our case, US was used as a guide for the procedure for diagnostic purposes and for accurate needle positioning. We felt that exact real-time localization by this approach would lead to procedural success and avoid possible complications. In conclusion, US-guided PRF may be an effective and durable treatment modality for the management of refractory pain in CRPS Type II.

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#### **Conflicts of interest**

There are no conflicts of interest.

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