



Peter's Persistent Pedal Pain



Chris Riddle, BSc; and Steven Curry, MD, CCFP

Peter's case

Peter, 35, presents to the ED with severe (8/10) pain in his right foot. The pain is of a burning quality, is non-localizable and is present throughout the foot, including the ankle joint.

Five weeks ago while playing hockey, Peter was hit with the puck over the right medial malleolus. Initially, he could walk, but upon arriving at the hospital, he could no longer bear weight. Initial x-rays and films done 10 days later are both negative. Over the next month, the bruising resolved, but severe burning pain and significant swelling developed. Furthermore, Peter reports that his right foot has become extremely sensitive to touch. He cannot sleep at night, as even the sheet brushing against his leg results in intense pain. He can walk, but with much difficulty. Acetaminophen has not alleviated the pain.

His past medical history is unremarkable except for a torn rotator cuff several months ago. He is not taking any medication (prescribed or OTC).

Physical Exam

He has an obvious antalgic gait. Diffuse swelling is present over the entire right ankle joint and proximal forefoot. There is marked tenderness (Grade 3) to light palpation over the right ankle and midfoot. No bony abnormalities, bruising or erythema are visible and there are no palpable temperature differences between the right and left foot. The right foot feels moist compared to the left. Normal pedal pulses are present bilaterally. Sensory and motor testing are both normal, though painful.

Lab investigations and Imaging

Both normal.

Turn to page 4 for more on Peter.

Questions & Answers

1. What is the diagnosis?

This is likely complex regional pain syndrome (CRPS) Type I, formerly known as reflex sympathetic dystrophy (RSD). CRPS is a chronic condition characterized by spontaneous severe burning pain, allodynia and hyperalgesia. Autonomic, trophic and motor system changes are also present. The most common causes of CRPS are limb injury (by some estimates accounting for up to 50% of CRPS patients) and stroke; however, it may develop from a variety of other events including:

- MI
- Frostbite
- Surgical procedures
- Prolonged immobilization

The pathophysiology of CRPS is often contested and not yet fully understood. However, it is generally accepted that functional disturbances of the central nervous system and peripheral inflammatory processes both play key roles. CRPS is classified as either Type I (no identifiable peripheral nerve damage) or Type II (detectable peripheral nerve damage).

2. How does CRPS present?

The clinical picture of CRPS results from involvement of the sensory, autonomic, trophic and motor systems of the affected limb. Characteristic signs and symptoms include a combination of:

- Spontaneous burning pain
- Mechanical and thermal allodynia

Table 1

IASP criteria for the diagnosis of complex regional pain syndrome

1. Continuing pain (with allodynia or hyperalgesia) that is disproportionate to any inciting event*
2. Presence (at some point, not necessarily at presentation) of edema, altered blood flow and sudomotor changes in the area of the affected limb
3. Pain and dysfunction that cannot be attributed to any other cause

* The presence of an initial traumatic event is useful, but not required for diagnosis, as up to 10% of patients will lack any such incident.

IASP: The International Association for the Study of Pain

- Edema
- Hyperhidrosis and/or hypohidrosis
- Increased and/or decreased temperature
- Increased and/or decreased hair growth
- Muscle weakness

The pain is typically worse when the limb is in a dependent position and does not follow a definite dermatomal distribution. The course of CRPS is commonly divided into three separate stages. The acute stage is characterized primarily by pain, vasomotor dysfunction, edema and sudomotor changes and typically lasts for up to three months. The dystrophic stage consists of more intense pain, continued vasomotor dysfunction and the development of motor dysfunction. The atrophic stage is identified by decreased pain, continued vasomotor disturbance and pronounced motor changes (which are often irreversible). It is important to note that this staging system may be useful for therapeutic purposes, but does not accurately reflect the progression of CRPS in most patients.

3. How is the diagnosis made?

CRPS is a clinical diagnosis, relying almost entirely on a thorough history and detailed physical exam. There are currently no diagnostic tests. Initially, alternative diagnoses that may explain the clinical picture must be excluded. An elevated erythrocyte sedimentation rate, fever, leukocytosis, elevated antibody titre or specific antigen all point to alternative pathology, such as cellulitis or other infections. Plain films, while not generally useful, may show a poorly healed fracture or other bony disturbances capable of eliciting the observed signs and symptoms. In the early stages, three-phase bone scintigraphy may be useful in distinguishing CRPS from arthritis or osteomyelitis. The former shows tracer accumulation within the entire extremity, while the latter results in tracer accumulation confined to a particular region within the limb.

Although not generally available in the ED or FP's office, confirmation of the observed signs may be done

Mr. Riddle is a Dalhousie Medical Student, Class of 2009, Halifax, Nova Scotia.

Dr. Curry is an Emergency Physician at the QEII Health Sciences Centre, Halifax, Nova Scotia.

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STA Communications Inc.
955 boulevard St-Jean, Suite 306
Pointe-Claire, QC, H9R 5K3

Peter's case cont'd...

Peter is given immediate release oral morphine (30 mg tablet) in the ED and discharged home with morphine sulfate controlled-release. He is instructed to follow up with his family doctor one week later.

At the follow up, the pain relief from the opioids is significant, however Peter requires gabapentin for long-term pain control.

Four months after his initial presentation, he no longer requires analgesics and is able to start playing hockey again.

through several quantitative tests. Skin temperature can be measured by Doppler flowmeters, sweating may be assessed by quantitative sudomotor axon reflex testing and sensory abnormalities may be assessed through quantitative sensory testing. Once other possibilities have been ruled out, specific diagnostic criteria are applied. The International Association for the Study of Pain (IASP) criteria (Table 1) are used most often, though Bruehl's or Veldman's criteria are acceptable alternatives.

4. *What is the treatment for CRPS?*

The overall goal of treatment is to achieve functional restoration and desensitization. An interdisciplinary approach involving physicians (*e.g.*, physical medicine specialists, pain specialists, anesthesiologists), psychological therapy, occupational therapy, physical therapy, recreational therapy and vocational rehabilitation is essential if this goal is to be reached.

It is difficult to predict the outcome for patients with CRPS. Some patients recover fully within a matter of weeks, while others live for years with the characteristic pain and resultant decline in function.


The primary responsibility of the emergency or primary care physician is to:

- recognize the condition,
- provide immediate pain management and
- refer the patient to colleagues who can then begin the process of desensitization and functional restoration.

The importance of an early diagnosis cannot be understated and is essential in preventing irreversible damage.

Case of the Month

Unfortunately, there are no guidelines for the specific treatment of CRPS and therapy must therefore be tailored to the individual. In the ED, the focus is on alleviating pain. Narcotics are often the first-line pharmacotherapy for temporary pain relief. Morphine sulfate is the drug of choice because of its predictable and reliable side-effects, as well as reversibility with Naloxone. An initial dose of 2 mg is administered (either through IV, intramuscularly, or subcutaneously) and titrated as needed. Hydromorphone may also be used. For longer term pain control, it is sensible to start with gabapentin, moving to tricyclic antidepressants if the response is inadequate. Corticosteroids and bisphosphonates have been effective for some patients.

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Resources

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4. Intenzo CM, Kim SM, Capuzzi DM: The Role of Nuclear Medicine in the Evaluation of Complex Regional Pain Syndrome Type I. *Clinical Nuclear Medicine* 2005; 30(6):400-7.
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