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Dealing with CRPS

What is the current best management for complex regional pain syndrome?

Question submitted by:
Steve O'Brien, MD
Charlottetown,
Prince Edward Island

Reflex sympathetic dystrophy, or complex regional pain syndrome (CRPS), is an example of neuropathic pain—pain that is mediated by a lesion or dysfunction in the nervous system. Typically it is precipitated by trauma and immobilization. Often, the trauma appears minor. It can be subclassified as CRPS 1 (without nerve injury) and CRPS 2 (with nerve injury).

Pathophysiology

It is thought that CRPS occurs as a result of a normal sympathetic response to trauma that does not shut off, leading to prolonged vasoconstriction and the continued release of proinflammatory mediators. The end result is pain, swelling, trophic changes and disuse of the affected area. However, the sympathetic role in CRPS has recently been called into question.

Natural history

CRPS is characterized by pain, trophic changes and autonomic dysfunction. Typically, on examination, there is evidence of allodynia, hyperalgesia and hyperpathia. The early phase of the disorder is characterized by pain that is disproportionate to the degree of trauma. There is evidence of swelling, redness and vasomotor instability, hyperhidrosis and coolness to the touch. Demineralization begins due to disuse.

Over time, redness gives way to cyanosis of the skin, fibrosis affects the joints and demineralization becomes pronounced, leading to osteoporosis.

Investigations

Although tests ranging from three-phase bone scan and thermography to laser Doppler fluxmetry have been used as investigative tools in making the diagnosis of CRPS, the key to diagnosis is the history and physical examination.

Treatment

Outcome is improved by early identification of this disorder and early treatment. Typically,

treatment focusses on early physiotherapeutic intervention. Evidence suggests that physiotherapy should be the first line of treatment for adolescent patients with CRPS.

Evidence to support specific pharmacotherapeutic treatments is limited. These interventions include the use of non-steroidal anti-inflammatory drugs, tricyclic antidepressants, anticonvulsants, steroids, alpha-2 agonists, calcium channel blockers, clonidine, capsaicin, calcitonin and opioids.

Surprisingly, the evidence for sympathetic blocks and sympathectomy is poor. There is support for spinal cord stimulation and thalidomide and etanercept are under investigation as treatment options.

Finally, the development of pain management skills is critical for patients who have to deal with this poorly understood, and often misdiagnosed, disorder.

Answered by:
Jeff Ennis, MSW, MD, FRCP(C)
Director
East End Multidisciplinary Pain
Management Program
Dundas, Ontario