

key points

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Complex regional pain syndrome (CRPS) is the current term for the condition formerly known as reflex sympathetic dystrophy. It is divided into type I, which occurs without obvious nerve injury and type II, in which a peripheral nerve injury is present, although the pain may not be limited to the distribution of that nerve. The pathophysiological nature of the disorder is still not clear. There is no reliable diagnostic test, and diagnosis rests on the presence of a constellation of symptoms and signs.

The condition typically starts with an injury to an extremity which is often seemingly trivial, followed by immobilisation, such as an ankle sprain or Colles' fracture. Instead of the expected resolution of symptoms, persistent pain and dysfunction develop. Symptoms and signs include extreme sensitivity, changing skin colour and temperature, trophic changes in the skin, nails and hair, and a decreased range of motion and loss of function. All these features are accompanied by considerable pain for which no other unifying diagnosis can be made.

CRPS usually has an early warm phase that suggests an inflammatory process, with local swelling, erythema and heat. It then progresses to a cold phase, which often goes on for months or years. No single mechanism has been found to account for this complex syndrome, and different mechanisms are likely to be active in different individual cases, and at different times, in its presentation.

A study using a large Dutch longitudinal database holding more than 600,000 patients found: the highest incidence in the seventh decade; the condition was 3.4 times more common in women than men; 44% of cases occurred after a bony fracture; and an estimated overall incidence rate of 26.2 per 100,000 person-years.⁵

Most authorities agree that an important element of treatment is to keep the affected part moving as much as possible, especially in the early stages. Sensory stimulation, particularly by self-massage, is advised. Patients should be encouraged to take analgesia before periods of movement likely to cause pain. The seemingly inflammatory nature of the acute phase has led to its treatment with agents known to suppress acute inflammation, such as NSAIDs and corticosteroids. There is some evidence supporting the use of oral gabapentin, nifedipine and phenoxybenzamine in the acute phase, along with intravenous ketamine. Bisphosphonates have been the subject of a number of small randomised controlled trials in CRPS with improvement in pain and mobility. Spinal cord stimulation has had remarkably good results in non-randomised studies.

Aggressive treatment of the condition in the early stages improves prognosis. Many cases, especially those with relatively minor symptoms, will resolve spontaneously. Patients who are symptomatically deteriorating, despite regular analgesia, neuropathic agents and physiotherapy, should be referred for assessment by a specialist.