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Complex Regional Pain Syndrome - CRPS

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Abbreviations: CRPS: Complex Regional Pain Syndrome, IASP: International Association for the Study of Pain

CRPS was first studied in the early 1800s by Claude Bernard. During the American Civil War, it was observed that soldiers after injury were often led to neuropathic pain, which was called causalgia by Silas Weir - Mitchell. The terms algodystrophy, Sudeck 's syndrome, persistent burning pain have been used from time to time. For this reason, the IASP (International Association for the study of pain) proposed to call the syndrome Complex Regional Pain Syndrome and to separate it into 2 groups: Type I: no obvious nerve damage. It was formerly characterized as reflex sympathetic dystrophy, reflex neurovascular dystrophy, algodystrophy, or Sudeck - type atrophy. Type II: there is clearly a nerve injury. It used to be called heartburn. There is complete disagreement regarding the pathophysiology of the syndrome. Numerous pathophysiological components of the disease have been identified, including neurogenic inflammation, peripheral and central sensitization, and disturbed sympathetic function. In cases of injury, local inflammation causes the production of inflammatory cytokines and neuropeptides. Cytokines stimulate osteoclasts of adjacent bones, bone remodelling increases, and osteoporosis is induced. Pain is attributed to stimulation of nociceptors by acidic enzymes released by osteoclasts to dissolve bone tissue. Its features include pain, sensory disturbances, edema, autonomic dysfunction, motility disorders, and trophic changes. Usually, automatic pain or allodynia is not limited to the territory of a single peripheral nerve and shows a disproportion with respect to stimulation. Stage I: sympathetic stimulation, burning sensation, muscle spasm, vasoconstriction, joint stiffness, reduced hair growth. Stage II: muscle atrophies, osteoporosis. Stage III: irreversible damage, limb deformities.

Type I (IASP diagnostic criteria): History of trauma or prolonged immobility; Presence of allodynia or hyperalgesia; Presence of swelling, local vascular disorders, skin color and hydration; The presence of any other disease that could justify the clinical picture excludes the diagnosis. Type II: Persistent allodynia or hyperalgesia after nerve injury; Edema, disorders of local vascularity, color and hydration of the skin; The presence of any other disease that could justify the clinical picture excludes the diagnosis. Diagnosis can be made by thermography, sweat reflex testing, x-ray, bone scintigraphy, bone mass measurement, electromyogram and nerve conduction study. Treatment of the syndrome includes non-steroidal anti-inflammatory drugs, oral and intra-articular cortisone, calcium antagonists, calcitonin, bisphosphonates, opioids, anticonvulsants, anticonvulsants, local nerve grafts, sympathectomy, ketamine, spinal cord neurostimulation, hypnosis, and limb amputation **[1-4]**.

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