Editorial

Complex Regional Pain Syndrome Type 1 is a Disorder to Prevent and Treat

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The Complex Regional Pain Syndrome type 1 (CRPS1) is a reflex sympathetic dystrophy syndrome, that occurs after an illness or injury that didn’t directly damage the nerves in your affected limb [1].

The typical areas that CRPS can affect are the arms, legs, hands, or feet. The condition usually occurs after some kind of injury or trauma to the area. It is a syndrome characterized by pain at even rest, functional impotence, edema, micro vascular alterations skin, alloying and hyperalgesia. The clinical criteria currently used for the diagnosis of CRPS 1 are those defined by Harden and Bruehel in 2007 (“Budapest clinical diagnostic criteria for CRPS”) [2] [Table 1].

The incidence of CRPS1 varies from 5 to 45 / 100,000 / year in depending on the various studies. The female is more frequently affected with a female/ males ratio of 4 to 1 and most affected is middle age [3,4].

The trauma is a causative factor in 40-70% of cases and it can be represented by a joint sprain, a comminuted fracture, a crushing trauma, a micro repeated trauma. Contributing factors are represented by the application of a tight cast, the anti-physiological position in which it immobilizes a limb, from prolonged immobilization and the absence of load [5]. All the so increase the patient’s disuse of the extremity and promoting fear-avoidance, which may progress into a neurological neglect-like syndrome. The pathogenetic mechanism is related to the release of neuro-inflammatory peptides and cytokines. This is the event that triggers and maintains the first stages of the process, while in the chronic phase prevails the disturbance of the micro circle [6].

In our orthopedic departments 12.5% of ankle fractures (surgical and not surgical) and about 18% of wrist fractures (surgical and not surgical) are affected by CRPS1. Patients report pain, swelling and functional impairment for about 7/8 months after injury and delay the resumption of normal limb activity, although the axis and limb anatomy is well reconstructed.

The clinical presentation is characterized mainly from continuous pain, which is aggravated by the load and it is present even at night, difficulty and/or inability in using the affected extremity, rapid fatigability, range of motion limited, edema, allodynia and hyperalgesia, altered skin temperature, hypotrichosis, altered nail growth.

The risk of over diagnosing CRPS must be taken into account. A detailed history and physical examination, as well as the for mentioned specifications, including testing, are necessary to differentiate CRPS from other neuropathic and pain syndromes: diabetic polyneuropathy, autoimmune or hematological disorder, raynaudphenomena, entrapment (eg. carpal tunnel, cubital tunnel), multiple sclerosis, etc... For this reason it is essential to collect well the patient’s history and determine the relationship between trauma and CRPS1.

No specific diagnostic tests confirm the presence of CRPS. Blood tests and electromyography may exclude haematic or neuropathic diseases. The X-ray shows revealendoosteal and intra cortical excavation, resorption of subperiosteal and trabecular bone, localized bone demineralization, and/ or osteoporosis.

Surely it is always essential prevention, avoiding performing repeated reductive maneuvers, to apply too tight plaster cast, to immobilize the limb in anti-physiological and administering preventive Vitamin C objective position, because of its antioxidant properties.

The most used drugs are the major analgesics (for pain control), corticosteroids (systemic route to reduce edema), Calcium-regulating drugs (they have been shown to significantly improve pain, swelling, and range of movement in patients with acute CRPS), NSAIDs (for pain control) [7].

Drugs lass that for us seems to offer the greatest effective guarantees is represented by neridronate, the only bisphosphonate indicated for the treatment of CRPS, administered intravenously at a dose of 100 mg per day every three days for a total of four infusions.
The neridronate has the ability to bind the hydroxyl apatite and preventing its dissolution; it acts by inhibiting the anaerobic metabolism, the production of lactic acid, and then the stimulation nociceptive generated by reduced local pH. The effectiveness the action could also be mediated interference with the production of GTPases, and then with the genesis and production of the painful stimulus [8]. The physio-kinesi therapy and particularly the magnetic and the active and passive mobilization assisted, it is recommended in the initials stages of the disease for the purpose of reducing local edema and improve function.

CRPS 1 is a common disease with multifactorial pathogenesis. Trauma (fracture or joint sprain) can be found in over half the cases. The localized osteoporosis is due not only to an osteoclastactivity, but also to a roll-osteoclastic bone resorption mediated by the dissolution of hydroxyl apatite crystals due to hypoxia and lowering of local pH. An early medical therapy neridronate and physio-kinesi therapy is typically favorable with a restitution ad integrum in 80-90% of cases.

The purpose of the orthopedic surgeon is still preventing the disease, knowing that can be prevented by eliminating risk factors.

References

Table 1: Budapest clinical diagnostic criteria for CRPS.

| 1. | Continuing pain, which is disproportionate to any inciting event |
| 2. | Must report at least one symptom in three of the four following categories |
| • Sensory: reports of hyperesthesia and/or allodynia |
| • Vasomotor: reports of temperature asymmetry and/or skin color changes and/or skin color asymmetry |
| • Sudomotor/edema: reports of edema and/or sweating changes and/or sweating asymmetry |
| • Motor/trophic: reports of decrease range of motion and/or motor dys function (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin) |
| 3. | Must display at least one sign at time of evaluation in two or more of the following categories: |
| • Sensory: evidence of hyperalgesia (to pinprick) and/or allodynia (to light touch and/or deep somatic pressure and/or joint movement) |
| • Vasomotor: evidence of temperature asymmetry and/or skin color changes and/or asymmetry |
| • Sudomotor/edema: evidence of edema and/or sweating changes and/or sweating asymmetry |
| • Motor/trophic: evidence of decrease range of motion and/or motor dys function (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin) |
| 4. | There is no other diagnosis that better explains the signs and symptoms |