LETTER TO THE EDITOR

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Evaluation and management of the CRPS II

Sir

Complex regional pain syndrome (CRPS) describes a diversity of painful conditions which usually follow trauma with nerve injury. CRPS type II is characterized by somatic and vegetative functional deterioration [1-4], with neuro-inflammatory pathogenesis [3].

We would like to report our clinical experience with CRPS type II of the upper limb. In the last years, we observed that the lidocaine use was associated with CRPS onset, influencing the neuro-inflammatory mechanisms that underlie CRPS. Therefore, we developed therapeutic strategy in which the patients with CRPS II of the upper limb, who agreed to our study protocol, underwent the association of surgery and lidocaine infiltration (LI). 12 patients with CPRS II due to a peripheral nerve lesion of the upper extremities (Tab. I) presented edema, burning pain, hyperalgesia, allodynia, altered ROM with flexor deficit. They underwent three steps: preop-

TABLE I - *Case histories and outcomes.*

Case	Lesion Type	Injured Nerve	Follow-up 1-month	3-months	6/12/24/36-months
1	Cut	Digital	< pain; < allodynia; < tender area; < trigger point	absence of pain, allodynia, tender area and trigger point	absence of pain, allodynia, tender area and trigger point
2	Crushing	Digital	< pain; < allodynia; < tender area; < trigger point	absence of pain, allodynia, tender area and trigger point	absence of pain, allodynia, tender area and trigger point
3	Cut	Digital	< pain; < allodynia; < tender area; < trigger point	absence of pain, allodynia, tender area and trigger point	absence of pain, allodynia, tender area and trigger point
4	Iatrogenic	Median (sensory)	< pain; < allodynia; < tender area; < trigger point	absence of pain, allodynia, tender area and trigger point	absence of pain, allodynia, tender area and trigger point
5	Crushing	Median and Ulnar	< pain; < allodynia; < tender area; < trigger point	absence of pain, allodynia, tender area and trigger point	absence of pain, allodynia, tender area and trigger point
6	Crushing	Ulnar	< pain; < allodynia; < tender area; < trigger point	absence of pain, allodynia, tender area and trigger point	absence of pain, allodynia, tender area and trigger point
7	Iatrogenic	Median	< pain; < allodynia; < tender area; < trigger point	absence of pain, allodynia, tender area and trigger point	absence of pain, allodynia, tender area and trigger point
8	Iatrogenic	Median	< pain; < allodynia; < tender area; < trigger point	absence of pain, allodynia, tender area and trigger point	absence of pain, allodynia, tender area and trigger point
9	Iatrogenic	Median		< pain; < allodynia; < tender area; < trigger point	absence of pain, allodynia, tender area and trigger point
10	Cut	Digital		< pain; < allodynia; < tender area; < trigger point	absence of pain, allodynia, tender area and trigger point
11	Crushing	Digital		< pain; < allodynia; < tender area; < trigger point	absence of pain, allodynia, tender area and trigger point
12	Cut	Digital		< pain; < allodynia; < tender area; < trigger point	absence of pain, allodynia, tender area and trigger point

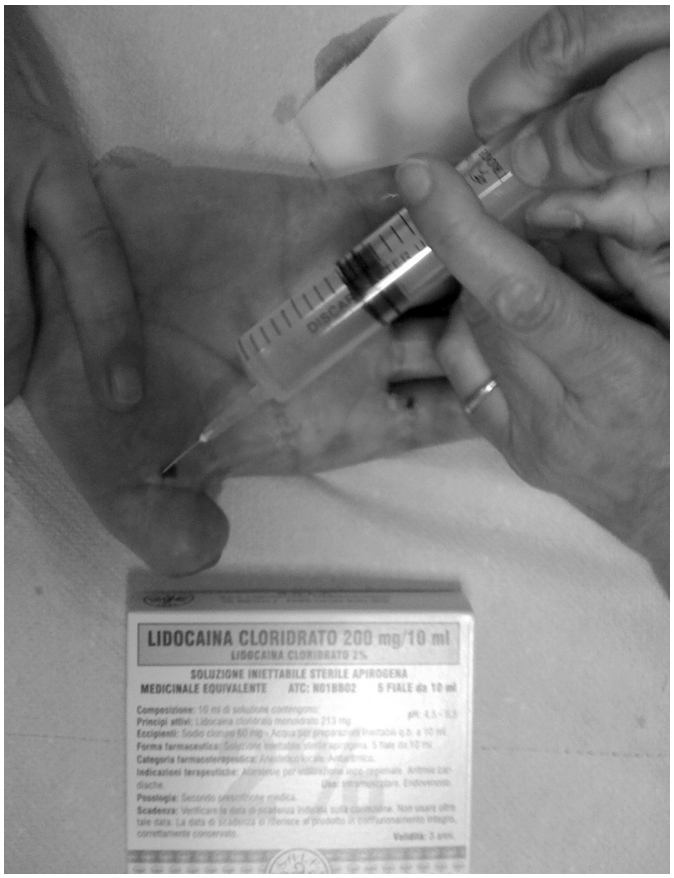

Fig. 1: Lidocaine 2% infiltration.

erative LI blocking; surgical nerve reconstruction after anesthetic blocking of the brachial plexus by LI; postoperative LI blocking. The temporary peripheral neuronal injection by lidocaine 2% of the ulnar, radial, median and digitalis nerves and even when necessary of the ganglion determined a neuromodulation. In postoperative, the injection protocol were performed every twice a week for 1 to 3 months, both around the previous lesion and on the anatomical neuronal projection area (Fig. 1). The follow-up was at 3, 6, 12, 24, 36 months. All the evaluations followed the IASP protocol [2]. At short-term follow-up, 8 patients were CRPS-free. At long-term follow-up, all the patients were CRPS-free (Tab. I).

The reasons for this successful therapy are to be found in the CRPS physiopathology characterized by (a) the increasing electro-tonic activity in the anatomical site of the nervous lesion, with a rise in incidence of the sodium channels causing a stable depolarization of the neuronal cells involved [1]; (b) the mechanism of the pathological excitation triggered by glutamate, and the inflammation caused by neuropeptides and prostaglandins [5]. From these, neuromodulation with LI, before and after surgical procedure might be appropriate to regulate the traffic of the afferent nerve in patients with upper limbs CPRS II. Lidocaine modulates the expression of sodium channels activity thus restricting the nerve depolarization and determining an increased extracellular title of glutamate and potassium [6]. This modulation stabilizes the depolarized neuronal membrane and reduces the discharge activity of the neuronal cell and its secretion. De Mos et al. showed that the interaction with the catabolism of neuropeptides influencing the neuro-inflammatory mechanisms that underlie CRPS [3]. According to Wörz, that utilized lidocaine plasters in chronic pain, the administration of this local anesthetic may modulate the neurophysiopathology process that after surgery could determine worsening of CRPS II [7]. We believe that an early reconstructive surgical procedure, without this neuromodulation, may cause worsening of nerve suffering. So, we propose that the surgical reconstructive procedure is to be performed after the reduction of the CPRS II. In our study, patients treated with LI and surgery did not have disease relapse. Our good results need to be confirmed by more data; nevertheless this neural-therapy appears extremely encouraging.

References

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