

Postoperative pain: What can we do?

ABSTRACT

Chronic postoperative pain (CPOP) is a potentially devastating consequence of a surgical procedure. It leads to increased medical costs, painful, and stress experience to the patients. After a surgical decompression performed in a patient with a non-traumatic compartment syndrome, a muscle biopsy confirmed McArdle disease, and after surgery, severe pain of neuropathic characteristics developed in the arm decompressed. Advanced techniques up to neuromedullary stimulation failed to improve the clinical status, after which repeated treatment with capsaicin patch ameliorated the patient's condition. This case report illustrates the need for a high index of suspicion for metabolic diseases in patients who present compartment syndrome without prior history of trauma and also the challenges in treating neuropathic pain after surgery.

Key words: Capsaicin, chronic postoperative pain, McArdle disease, neuropathic pain

Introduction

Chronic postoperative pain (CPOP) is a poorly recognized condition and is defined as persistent pain after surgery of greater than three months' duration. It can vary from mild to severe loss of quality of life so it is important to recognize it to reduce the suffering of patients and also the economic consequences that ensue from it.

McArdle disease is a rare metabolic myopathy that predisposes patients to exercise intolerance, fatigue, and painful muscle cramps.

This article discusses both conditions, aiming to demonstrate the great challenge that it is to control chronic pain developed after surgery.

Case Report

A 23-year-old woman presented to the emergency department with paralysis and severe pain of the right hand and forearm without an identifiable traumatic incident. On physical examination, the right forearm was firm to touch and passive movement elicited extreme pain. Radiographs were negative for fracture. A Doppler ultrasound revealed normal blood flow. Laboratory tests revealed myoglobinuria and elevated serum creatine kinase. Fluid resuscitation was initiated as well as analgesia. Ultimately atraumatic compartment syndrome was diagnosed and decompressive fasciotomies performed. She was discharged four days after surgery but maintained paresis and neuropathic pain at the right forearm and hand.

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A multidisciplinary team was put in place: an electromyography, a muscle biopsy, and a genetic test were performed and were compatible with McArdle disease.

The main objective in the follow-up of the patient was to decrease her pain as she described it as constant and unbearable (numeric pain scale 9/10), with neuropathic characteristics like burning and electric shocks. On examination her elbow was flexed, allodynia and hypoaesthesia were present and muscle strength was reduced.

This pain had an enormous impact on her life as she was not able to keep her job and had a poor quality of sleep.

To treat her pain, multiple conventional approaches were conducted: gabapentin, pregabalin, imipramine, paracetamol, non-steroidal inflammatory drugs, metamizole, deflazacort, tramadol, tapentadol, and oxycodone. However, they have little efficacy or were abandoned due to side effects. At the same time, the patient initiated occupational therapy, acupuncture, and psychological follow-up that she felt were of no benefit.

As conventional therapy didn't work, invasive treatments were attempted. First, an ultrasound-guided block of the right stellate ganglion was accomplished with the injection of lidocaine 2% and 80 mg of methylprednisolone, without efficacy. Secondly, a right brachial plexus block via supraclavicular approach was performed with 20 ml of ropivacaine 0.2%, again without pain relief. One year and three months after her injury and without any substantial improvement in her pain, a spinal cord neurostimulation was attempted. Twenty days after its placement and with the lack of positive results, the spinal cord stimulation was removed.

After all this journey, the patient was more and more helpless. At this point, a topical application of capsaicin was proposed to the patient, which she accepted. This application, two patches 280 cm² and two patches 179 cm², was performed under sedation with midazolam, a perfusion of ketamine, and some bolus of propofol when needed. The application went without complications, but at the end of the procedure the patient compliant of burning sensation at the place where the patches were applied which improved with local ice and oramorph.

Ten days after the application of the capsaicin patches, the patient reported improvement of mobility and sensibility at the hand.

Discussion

McArdle disease is a rare genetic disease with an anomaly at the breakdown of muscle glycogen as a consequence of myophosphorylase-deficient activity.^[1] That's why these patients commonly present fatigue and muscle pain.

Despite surgery to resolve the compartment syndrome, the patient started to experience extreme pain of neuropathic characteristics that had a serious and negative impact on her quality of life. CPOP affects 2–10% of adults undergoing surgery although incidence varies with the definition used and the type of surgery performed.^[2] The criteria to establish a diagnosis of CPOP are 1) the pain develops after a surgical procedure or increases in intensity after the surgical procedure, 2) the pain should be of at least 3–6 months' duration and significantly affect the health-related quality of life, 3) the pain is either a continuation of acute post-surgery pain or develops after an asymptomatic period, 4) the pain is either localized to the surgical field, projected to the innervation territory of a nerve situated in the surgical field, or referred to a dermatome (after surgery in deep somatic or visceral tissues), 5) other causes of the pain should be excluded.^[3] In the case presented, all these criteria were met.

Currently, there have been identified some risk factors for the development of CPOP, as the treatment of already established CPOP is difficult a prevention policy is the best approach.^[4]

CPOP is frequently neuropathic, and so it is more prone to higher-intensity pain and chronification. Neuropathic pain is caused by a lesion or disease of the somatosensory system.^[5] Due to the complexity of neuropathic mechanisms, treatment decisions are often challenging. To prevent CPOP, one must achieve effective early postoperative analgesia and include agents that prevent sensitization (e.g., gabapentinoids or N-methyl-D-aspartate receptor antagonists) and not just treatment of somatic pain (e.g., opioids).^[6]

In this case, first conventional treatments failed and it was necessary to try more invasive treatments. Among these, the only one that showed to be beneficial was topical capsaicin. Capsaicin leads to the activation of transient receptor potential cation channel subfamily V member 1 receptors in the noxious area which then results in sensory neuronal depolarization leading to the sensations of heat, burning, stinging, or itching at the moment of application. High concentrations of capsaicin or repeated applications produce a persistent local effect on cutaneous nociceptors that reduce spontaneous activity and a loss of responsiveness

to a wide range of sensory stimuli in that area leading to the improvement of the pain.^[7]

Due to its relevance, CPOP should be kept in mind as a potential complication following surgery, and it is crucial to raise awareness to this entity to reduce its incidence and potential complications.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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