**Case Report**

**Management of complex regional pain syndrome type II using lidoderm 5% patches**

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We report a case of a patient developing complex regional pain syndrome of the upper limb after a laceration injury with glass. The pain in his hand was resistant to all conventional modes of treatment. The pain reduced dramatically after a diagnostic lidocaine infusion and the reduction in pain lasted for 3 days. Following this the patient responded well to lidoderm 5% patches and achieved 80% pain relief with an improved range of movement in his hand.

**Keywords:** complications; complex regional pain syndrome; lidoderm 5% patches; rehabilitation; physical; safety

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**Case report**

A 35-yr-old former welder presented to our pain clinic with a tender thenar eminence and palmar surface of the right thumb. His original injury was a glass laceration one and a half years previously, after which he underwent an operation for repair of the right radial digital nerve and a full thickness skin graft. He underwent a further operation a few months later for removal of a neuroma in the thumb. His pain did not resolve in spite of physiotherapy and simple analgesics. He was therefore referred to the pain clinic.

He was otherwise fit and well and had no allergies. There was no ongoing litigation regarding his injury. On examination, the patient had an extremely tender thenar eminence and thumb. The pain was disproportionate in intensity and duration relative to the inciting event and radiated to the rest of his palm and the wrist. He had mechanical allodynia and hyperalgesia. The swelling present in the acute phase of his injury was resolving. His hand was white and cold compared with the uninjured hand and had a restricted range of movement at the wrist joint. The skin of his palm was glossy with increased nail growth. There was also reduced prominence of the thenar muscles compared with the other hand. The patient was diagnosed clinically to have complex regional pain syndrome (CRPS) type II according to the IASP criteria and was started on pregabalin 50 mg bd, amitriptyline 10 mg noxte, and cocodamol 8/500; two tablets qds. He was able to increase the amitriptyline up to 50 mg nocte over a period of 3 months but found it difficult to increase the dose of pregabalin above 75 mg bd because of somnolence and hence was started on topiramate 25 mg daily. Pregabalin and amitriptyline were continued. The patient did not show any signs of improvement after 5 months and was given a trial of series of three i.v. blocks using prilocaine 0.5%, ketorolac, and fentanyl. These drugs were used for all the three blocks, but only had short lived effects. He also underwent a series of three stellate ganglion blocks using levobupivacaine 0.5% in each with minimal benefit.

Because of the failure of conventional neuropathic pain medications and sympathetic blocks, a diagnostic infusion of 3 mg kg⁻¹ of lidocaine administered over 1 h was performed. This was repeated a week later. Following the infusions, the patient displayed a dramatic improvement of more than 50% in the movement, grip, and pain in the hand. This effect lasted for about 3 days after which the pain recurred. Following this success, he was given lidoderm 5% patches for a trial period of 2 weeks to be used in concert with his other neuropathic medications. He was advised to wear three patches for 12 h a day. A review 2 weeks later showed improvement in his pain and movements by about 80%.

**Discussion**

CRPS can be difficult to treat. Management focuses on patient education and a strategy of encouraging use and movement of the limb through paced and goal-directed
The pathophysiology of neuropathic pain, including CRPS, is poorly understood. There are a number of theories advanced to explain its development. These include nociceptive-sympathetic cross talk and wind up and overexpression of voltage-gated sodium channels, including PN3, causing increased current density and a lowered action potential threshold. The overexpression of sodium channels suggests a possible role for local anaesthetics in the treatment of CRPS. In 1982, Boas and colleagues showed that low doses and blood concentrations of lidocaine had no effect on ischemic pain, but suppressed the clinical neuralgia pain in the same patients. They suggested that that deafferentation disorders responded favourably to i.v. lidocaine, whereas pain of peripheral origin was unaffected.

A pilot study by Devers and colleagues in 2000 assessed the effectiveness and tolerability of topical lidocaine patches for treatment of neuropathic pain conditions other than post-herpetic neuralgia. Moderate pain relief was reported by 81% of the participants. Fifteen out of the 16 patients in the study had a mean duration of patch use of 6.2 weeks with continued pain relief without any side effects. The lidoderm patch provided clinically meaningful pain relief in most of these refractory cases.

The use of i.v. lidocaine infusions for the treatment for neuropathic pain in cancer patients has been described. Ten patients received 5 mg kg\(^{-1}\) of lidocaine infusion in 1 ml kg\(^{-1}\) of isotonic normal saline over 1 h under continuous monitoring. Seven of the 10 patients had significant pain relief immediately after the infusion. Pain relief was sustained for an average of 14 days. The patients had no side effects and signs of toxicity.

The safety, tolerability, and pharmacokinetics of 5% topical lidocaine patches applied for 72 h were studied in 2002. This was a randomized, prospective, multiple-dose pharmacokinetic study that studied two groups of healthy men and women. One group had four lidocaine patches every 24 h and the other four patches every 12 h. In both groups, plasma lidocaine concentrations remained well below those that would produce anti-arrhythmic or other toxic effects.

Wallace and colleagues used a computer-controlled infusion pump to target and maintain stable plasma lidocaine concentrations and study their effects on pain scores in patients with neuropathic pain after peripheral nerve injury. They found that there was significant plasma concentration-dependent decrease in the pain scores starting at 1.5 \(\mu\)g ml\(^{-1}\). This effect corresponded to the decrease in the size of the receptive field to which the pain was referred.

The lidoderm 5% patch has been approved by the food and drug administration for the treatment of post-herpetic neuralgia. We present here a use of lidoderm patches, outside of their UK licence, in the management of a refractory case of CRPS type II. The long-term use of lidoderm patch therapy is currently very expensive. However, facilitating the functional rehabilitation as painlessly as possible using various interventions is pivotal in the management of CRPS. We suggest that lidoderm patches may have a role in the management of difficult cases of CRPS and deserves further investigation.

References