CASE REPORT

Treatment of an acute severe central neuropathic pain syndrome by topical application of lidocaine 5% patch: a case report

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Study design: Case report.

Objective: Metastatic epidural spinal cord compression frequently leads to central neuropathic pain symptoms, which are often difficult to treat. A new treatment option is being presented.

Setting: Antwerp University Hospital, Belgium.

Clinical presentation: We present the case of a 54-year-old male patient suffering from metastatic pancreatic cancer. During the course of his disease the patient developed a paraplegic syndrome due to metastatic compression of the spinal cord. After surgical decompression was performed, patient developed a severe at-level neuropathic pain syndrome, consisting of dysesthesias and allodynia. After all other pharmacological approaches had failed, treatment with lidocaine 5% patches was initiated. Within 4 h after the first patch application, neuropathic symptoms started to disappear. After 12 h, patient had become completely pain free. This topical treatment was continued during several weeks with lasting analgesic efficacy.

Conclusion: This case report indicates that transdermal administration of lidocaine could be potentially useful in the treatment of acute central painful neuropathies. We hypothesize that the transdermal administration of lidocaine induces suppression of ectopic impulses in sensitized cutaneous afferents, leading to some kind of 'dewinding syndrome' in the dorsal horn, which in turn leads to a decrease of painful symptoms.

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Introduction

Metastatic epidural spinal cord compression (MESCC) will develop in 5–10% of cancer patients, often leading to excruciating pain syndromes.¹ Treatment consists of a combination of surgery, radiation treatment, chemotherapy and pharmacotherapy. Little is known about pain symptoms that can develop after surgical decompression of MESCC. As in peripheral compressive neuropathies patients with MESCC will often display an increase in neuropathic symptoms at or below the level of injury after decompression. Dysesthesias are a frequent clinical problem after release of spinal cord compression.² Management of such pain symptoms remains difficult due to poor understanding of the underlying mechanisms.

Case report

A 54-year-old male patient was diagnosed with metastatic pancreatic cancer. Chemotherapy was initiated using cisplatin and gemcitabine. Four months later, patient was admitted to the emergency ward displaying a paraplegic syndrome. Two days earlier he noticed weakness in his legs, which became progressively worse. Later on he lost sensation to void and became unable to urinate. Emergency magnetic resonance imaging was performed which showed expansive metastasis at the level Th11 (Figure 1). Invasion of the posterior epidural space with severe compression of the thoracic myelum was visible (Figure 2). Surgical decompression was performed in an effort to restore neurological function.

Three days after surgery, patient developed excruciating pain, situated within the Th11 dermatome (Figure 3). He described spontaneous pain symptoms as if needles were stuck into his skin. In addition, presence of mechanical allodynia was revealed within the same dermatome. Treatment with oral morphine was rapidly initiated. Despite titration to a daily dose of 60 mg it failed to produce

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Figure 1 Sagittal magnetic resonance imaging (MRI) with contrast demonstrating a mass at the level of Th 11 resulting in a significant cord compression at this level.

Figure 2 Axial T1-weighted magnetic resonance imaging (MRI) revealing an expansile mass at the level of Th 11 resulting in a significant compression of the spinal cord.

sufficient analgesia. Opioid rotation to hydromorphone (up to 16 mg daily) was then performed but discontinued after a few days due to nausea and vomiting. Concomitant administration of amitriptyline had to be stopped because of dysrhythmias. Owing to failing analgesic responses, all therapies were stopped and a treatment using lidocaine patches 5% (Neurodol, Ciba, CH) was initiated. One entire patch was applied to the allodynic skin area in a 12 h on/off schedule (Figure 3). Within 4 h of initiation of patch therapy dysesthetic symptoms started to resolve. Twelve hours later, spontaneous and evoked pain symptoms had almost completely disappeared. Patch treatment was continued on a

Figure 3 Photograph of the patient, indicating the precise placement of the lidocaine patch. The drawing delineates the contours of the allodynic region that was observed before the start of the patch therapy.

daily basis with lasting analgesic efficacy. During patch placement, patient would neither report dysesthetic nor allodynic neuropathic symptoms. After removal of the patch patient would remain pain free during many hours, but towards the end of every patch-free period he would report minor non-evoked nociceptive symptoms (such as paresthesias and dysesthesias). However, patient never complained of allodynia-related symptoms any more. Over the next weeks, neurological status of the patient deteriorated to a status of spastic paraplegia. After 4 weeks, he developed a septic syndrome from which he eventually expired.

Discussion

We describe the case of a MESCC leading to spinal cord injury (SCI). Shortly after undergoing decompressive surgery patient developed at-level neuropathic pain symptoms. To our knowledge, this case report cites for the first time the usefulness of lidocaine patches in the treatment of acute central neuropathic pain syndromes.

Pain following SCI may be related to the activity in the neuronal pools located adjacent to the site of injury. Wide dynamic range (WDR) neurons close to the lesion turn into a status of hyperexcitability, characterized by robust increase in spontaneous activities, more action potentials in response to C-fiber input and facilitated wind-up response.³ This hyperactivity may contribute to the development of spontaneous and evoked neuropathic symptoms. Human evidence supports the fact that sodium channel blockers can confer significant neuroprotection after SCI and result in behavioral recovery.⁴ Although the anatomic target of sodium channel blockers in central pain remains unknown, decreasing ectopic discharges from injured neurons in the spinothalamic-cortical pathway has been proposed to account for the effect. In the present case, lidocaine was administered through the skin targeting the afferent fibers present in the allodynic skin area.

The observations that spontaneous nociception always reoccurred towards the end of the patch-free period, to disappear again soon after application of a new patch, indicate a lidocaine-dependent analgesic effect. It is unlikely that the patch by itself (acting as a protective barrier) inhibited the allodynia, because such an effect would never last during the 12 patch-free hours. Moreover, a previous study has shown the efficacy of lidocaine patches on ongoing pain and allodynia in peripheral neuropathies, with no analgesic effect from placebo patches.⁵ Since the neurological status of the patient never recovered after surgery, the observed analgesia cannot be explained through natural remission. It is therefore our hypothesis that transdermal lidocaine resulted in an inhibition of cutaneous C-fiber activity, leading to a significant decrease in afferent input to the WDR neurons. This inhibition of C-fiber input to the spinal cord decreased action potential generation, hereby preventing the development of a central wind-up phenomenon and leading to behavioral recovery. Further

research is warranted into the neurophysiologic actions of this new treatment option.

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