Pain Management after Traumatic Spinal Cord Injury

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ABSTRACT

Pain is experienced by people suffering from spinal cord injury leading to disability and affecting the person's functional ability, independence, psychological well - being, ability to return to work and quality of life. Pain management in these patients is notoriously difficult due to multiple factors and varied mechanism leading to pain. Only few treatments have been assessed in randomized, controlled trials and management is based on different case reports. We are reporting our experience of managing 2 patients with neuropathic pain following traumatic spinal cord injury. Both the patients were refractory to conventional pain medications but there was significant reduction in pain following lignocaine infusion in the first patient and with ketamine in the second patient.

Key words: Ketamine. Lignocaine. Pharmacological therapy. Spinal cord Injury. Neuropathic pain.

INTRODUCTION

Neuropathic pain is a major cause of disability following traumatic spinal cord injury (SCI) and have greater impact on patient's quality of life, psychological well-being than the extent of injury itself.¹

Mechanism that lead to this neuropathic pain is unclear therefore, the effective treatment of pain is notoriously difficult and poses a therapeutic challenge. Only few treatments have been assessed in randomized controlled trials and management is based on different case reports.² In this case report we present 2 cases of traumatic spinal cord injury with continuous neuropathic pain managed at the Aga Khan University, Karachi, Pakistan.

CASE REPORT

Case 1: A 57 years old male after gun shot injury in the neck had C7-T1 vertebral fracture and SCI. He was managed with cervicodorsal decompression and fixation. After that he developed paraplegia, paresis of both upper limb and pain in both forearms and hand. Neurosurgeons tried to manage his pain for 1 year but there was no response and he was therefore, referred to pain clinic.

Pain was diffusely located over both forearms and hand and was constant and debilitating. Pain score was 8-10/10 on numerical rating scale (NRS). His sleep was significantly disturbed and mood was irritable. His bladder and bowel controls were normal. On physical examination, he had both motor and sensory loss of

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lower limb. There was weakness of upper limb and it was difficult to examine because of allodynia. At the time of assessment his medications were tramadol, nonsteroidal anti-inflammatory drugs (NSAID) gabapentin and amitryptyline. We planned a diagnostic lignocaine injection (100 mg i/v loading dose followed by 100 mg i/v infusion over one hour) with monitoring of haemodynamics and lignocaine toxicity. He showed a very good response and pain score decreased to 3-5/10. Allodynia and hyperalgesia was also reduced. He was then prescribed capsule mexiletine in addition to fluoxetine, amitriptyline and pregabalin. Mexiletine was given in a dose of 100 mg at night for 3 days and was then increased to 100 mg twice daily. Lignocaine infusion was repeated twice at 4 weeks interval. He was also referred for physiotherapy. He came for follow-up at 3-4 monthly intervals for 3 years and his pain score ranged between 2-3/10 and was manageable.

Case 2: A 42 years old male was referred to chronic pain management service with severe pain in both lower limbs from thigh to feet. He was admitted in hospital one month ago after gunshot injury of lower back with fracture of first and second lumbar vertebrae and spinal cord injury and was managed conservatively. Neurosurgeon failed to control his pain by opioids and NSAIDs.

Pain radiated from thigh to feet bilaterally with score of 9-10/10 on 10 point NRS. Pain was paroxysmal, crampy, shooting burning like sensation with marked allodynia. His sleep was disturbed and was depressed. He also had faecal and urinary incontinence. Initial attempt to manage his pain with gabapentin (upto 3600 mg/day) and amitriptyline (25 mg twice daily) could not cause significant pain relief. Lamotrigine was added and later replaced by carbamazepine but to no beneficial effect. Patient refused different interventional therapies (ankle block, lumbar sympathectomy and continuous epidural infusion).

On the 40th day of management lignocaine injection was given at the rate of 150 mg i/v followed by i/v infusion of 150 mg over one hour with monitoring of haemodynamics and lignocaine toxicity. The patient's pain score remained 8-9/10. After 2 days, ketamine infusion was started at the rate of 3 mg hr⁻¹ subcutaneously along with other drugs. For the first time patient reported that his pain scores reduced to 2-3/10 and had a very good sleep. Ketamine infusion was continued for 3 days and tablet amantadine was started in a dose of 100 mg twice daily. Patient was managed in the ward for next 20 days on oral amantadine, gabapentin and amitryptyline. During this 2 months period he was also seen by a psychologist and provided physiotherapy and rehabilitation services. Patient was discharged on the 60th day with pain scores of 0-2/10. Patient had come from a remote place and later was lost to follow-up.

DISCUSSION

After SCI adequate pain treatment is an integral part in the rehabilitation process. The cases presented here were suffering from continuous neuropathic pain below the level of injury which could not be managed by analgesics and conventional antidepressants and anticonvulsants. The first case had incomplete spinal cord lesion in cervical region, while second case had complete lesion of the spinal cord in the lumbar region. Quality of life was deteriorating in the first and it was difficult to rehabilitate the second patient.

The evidence for treatment algorithm, duration of adequate drug trials for SCI pain is lacking.³

When chronic neuropathic pain is resistant to conventional therapy than other drugs like N-methyl Daspartate (NMDA) receptors antagonists, lignocaine or clonidine are tried.

Lignocaine was used in the first case as different studies and case reports have shown a clear analgesic effect in patients with neuropathic pain of peripheral origin.^{4,5} Mexiletine is an oral congener of lignocaine and has been reported to be an effective analgesic for neuropathic pain in patients with diabetic neuropathy and central poststroke pain. Lignocaine is only available for i/v use, therefore, an infusion is administered for diagnostic purpose to predict the response to oral mexiletine.^{6,7} As seen in this case, the patient had remarkable improvement as evident by change in pain scores from 8-10/10 to 2-4/10, therefore, tablet mexiletine was added to the medication which the patient was already taking. There are clinical studies which demonstrated that systemic administration of the NMDA receptor antagonist may be used most effectively to reduce the symptoms of neuropathic rather than acting as a traditional analgesic. Ketamine and amantadine (non-competitive NMDA receptor antagonit) has also been used for the management of neuropathic pain with variable patterns of response.⁸⁻¹⁰ As the second case had no pain relief with lignocaine we planned to use ketamine by subcutaneous infusion. As oral ketamine was not available, amantadine was then started along with other medications which carried on with adequate pain control.

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