

Effect of Low Dose Lignocaine Injection in the Treatment of Post Stroke Pain: A Case Report

Vijay Adabala, Praveen Talawar*, Ajit Kuma

Department of Anesthesiology, All India Institute of Medical Sciences Rishikesh, Rishikesh, India.

ARTICLE INFO

Article history:

Received 25 August 2019

Revised 16 September 2019

Accepted 29 September 2019

Keywords:

Central post-stroke pain;

Low dose lignocaine;

Neuropathic pain

ABSTRACT

Pain experienced after a stroke is one of the worst experience for a given patient. Post stroke pain can present in various forms of which central post stroke pain (CPSP) is a neuropathic pain involving the area affected during the stroke. Till date there were different classes of medication used to treat CPSP without any promising results. This indirectly indicates so many mechanisms were included in these patients resulting in pain.

We would like report a case of CPSP successfully treated in our institute with low dose lignocaine injection peripherally.

We would like to conclude that the afferent sensory input from the painful area plays a role in maintaining the spontaneous (and the evoked) pain in CPSP which are getting blocked by giving low dose lignocaine injection peripherally. Further studies will be required to establish this novel treatment.

© 2020 Tehran University of Medical Sciences. All rights reserved.

Pain experienced after a stroke is one of the worst experience for a given patient. Post stroke pain can present as headaches, shoulder pain, spasticity, and central post stroke pain (CPSP) [1]. Central post-stroke pain (CPSP) is a neuropathic pain condition characterized by pain and sensory abnormalities that manifest in the body parts that correspond to the area of the brain that has been injured by the cerebrovascular lesion [2]. It can present in a single limb or even one half of the body. Although the reported incidence is less than what is expected there will be an increased trend in this condition as the life expectancy of the population is increasing.

Case Report

57-year-old female who had left sided hemiplegia one year back came to our pain clinic with the complaints of pain in the left upper and lower limbs with severity of pain on numerical rating scale (NRS) is 8/10. Patient was being treated with tab. gabapentin 200mg TDS, tab. duloxetine 20mg OD from past 6 months without any significant result in the reduction of pain.

Low dose lignocaine injection was planned in the upper and lower limbs. After explaining the treatment procedure to the patient and relatives written informed consent was taken for three serial injections of low dose lignocaine every 72 hours. Patient was advised to write NRS in a dairy every 12 hours. Patient was contacted on telephone on every second day after block.no adverse reactions occurred after blocks.

Patient in supine position with all the standard monitors attached, 1.5ml of 2% lignocaine diluted to 10ml was injected in the ulnar and radial nerve division dermatome (ulnar crease and anatomical snuff box respectfully) with 26G needle for upper limb and 10ml of the same drug is injected close to sciatic nerve in the upper and outer quadrant of the gluteal region by 22G, 8cm spinal needle after contacting the ileum bone.

Response of the patient:

1. After first injection relief of pain was there for 8 hours. (NRS 4/10).
2. After second injection relief of pain is for 1 day (NRS 4/10).
3. After 3rd injection significant pain relief was appreciated by the patient for 4 days.

The authors declare no conflicts of interest.

*Corresponding author.

E-mail address: praveent64@gmail.com

© 2020 Tehran University of Medical Sciences. All rights reserved.

At present patient NRS has come to 4/10 and patient was advised for physiotherapy for further management.

Discussion

Till date the exact pathogenesis of CPSP is unknown. The probable mechanisms include hyper excitation in the damaged sensory pathways, damage to the central inhibitory pathways, or a combination of the two [3]. The afferent input is critical for maintaining spontaneous pain after peripheral nerve damage. Once the afferent input from the painful area is removed, the spontaneous pain is abolished, regardless of the extent of central sensitization.

The central pain that is identified as CPSP may be described as constant, intermittent, moderate, or severe, and is caused by the brain lesion itself as in this case. Due to the disturbance in signaling pathway and the central compartment, normal sensations are also perceived as pain in these patients. The afferent sensory input from the painful area plays a role in maintaining the spontaneous (and the evoked) pain in CPSP [4].

As in this case low dose lignocaine injected in the periphery was thought to block all the sensory inputs from the periphery. The given dose (0.05%) is much lesser than the analgesic dose of lignocaine. Efficacy reported with antidepressants in reducing CPSP in some patients seems to implicate multiple chemical mechanisms [5]. The effectiveness of anti-epileptics and anti-arrhythmic implicates calcium and sodium channels; the effectiveness of opioids implicates opioid receptors [6]. Although local anaesthetics are not indicated in these cases in view of side effects, low dose local anaesthetics would give promising results. Providing temporary alleviation of neuropathic pain with a variety of treatments may attenuate, or “reset,” certain sensitized neuronal functions for periods well beyond the pharmacologically dictated duration of drug action.

Our results provide evidence that the afferent sensory input has an important role in maintaining pain in patients suffering from CPSP. This approach may open new

therapeutic horizons for targeting central neuropathic pain syndromes.

Till date there were different classes of medication used to treat CPSP without any promising results. This indirectly indicates so many mechanisms were included in these sorts of patients resulting in pain. Drugs such as amitriptyline, pregabalin and lamotrigine are effective treatments for CPSP. Pregabalin may improve pain-related anxiety and sleep disturbances. Non-pharmacologic treatment modalities, such as motor cortex stimulation or deep brain stimulation, are used in some cases; however, they are not proven to be effective. Low dose lignocaine would give promising results in this sort of patients with neuropathic pain. Further studies are necessary to establish this phenomenon.

References

- [1] Jensen TS, Finnerup NB. Central pain. In: McMahon SB, Koltzenburg M, Tracey I, Turk DC, editors. Wall and Melzack's textbook of pain. Philadelphia, PA: Elsevier, 2013.
- [2] Klit H, Finnerup NB, Jensen TS. Central post-stroke pain: clinical characteristics, pathophysiology, and management. *Lancet Neurol.* 2009; 8(9):857–68.
- [3] Nicholson BD. Evaluation and treatment of central pain syndromes. *Neurology* 2004; 62:S30–36.
- [4] Haroutounian S, Nikolajsen L, Bendtsen TF, Finnerup NB, Kristensen AD, Hasselstrom JB, et al. Primary afferent input critical for maintaining spontaneous pain in peripheral neuropathy. *Pain.* 2014; 155(7):1272–9.
- [5] Onouchi K, Koga H, Yokoyama K, Yoshiyama T. An open-label, long-term study examining the safety and tolerability of pregabalin in Japanese patients with central neuropathic pain. *J Pain Res.* 2014; 7:439–44.
- [6] Willoch F, Tolle TR, Wester HJ, Munz F, Petzold A, Schwaiger M, et al. Central pain after pontine infarction is associated with changes in opioid receptor binding: a PET study with ¹¹C-diprenorphine. *AJNR Am J Neuroradiol.* 1999; 20(4):686-90.