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Spinal Myoclonus Developed during Cervical Epidural Drug Infusion in Postherpetic Neuralgia Patient

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Postherpetic neuralgia is the most frequent complication of herpes zoster. Treatment of this neuropathic pain syndrome is difficult and often disappointing. Although postherpetic neuralgia is generally a self-limited condition, it can last indefinitely. Continuous epidural blockade for patients with acute zoster can shorten the duration of treatment. However, continuous epidural block has some complications such as infection, dural puncture, and total spinal and nerve damages. We report a case of myoclonus during continuous epidural block with ropivacaine, morphine, and ketamine in an acute zoster patient. (Korean J Pain 2011; 24: 169-171)

Key Words:
analgesia, complication, epidural, myoclonus.

Postherpetic neuralgia is the most frequent complication of herpes zoster. Treatment of this neuropathic pain syndrome is difficult and often disappointing. Continuous epidural blockade for patients with acute zoster can shorten the duration of the treatment [1]. Continuous epidural blockade has some complications such as infection, vascular injection, dural puncture, total spinal anesthesia, and nerve damage. Spinal myoclonus after neuraxial blockade is very rare complication. We report a case of bilateral myoclonus during continuous cervical epidural block with ropivacaine, morphine, and ketamine and reviewthe related literature.

CASE REPORT

A 61-year-old female came to our pain clinic and presented with erythematous vesicles and pain on the right side of her arm for 2 weeks. She had no significant past or family medical history. Initial physical examination of the patient showed grouped vesicles and crusts on an erythematous skin lesion, scattered along the right C5 dermatome. The intensity of pain using the visual analogue scale (VAS) from 0 to 10 (0 = no pain, 10 = worst pain imaginable) was 8. She complained of burning and lancinating pain, hyperesthesia, and sleep disturbance due to night pain on the affected dermatome.

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She had been treated with famciclovir (FAMVIR®, Novartis, USA) 250 mg 3 times a day and pregabalin (Lyrica®, Pfizer Korea, Korea) 150 mg twice a day and a tramadol/acetaminophen tablet (Ultracet®,Yansen Korea, Korea) 3 times a day orally for 7 days. Complete blood count, urine analysis, blood chemistry, electrocardiogram, and chest and spine radiographs were all within normal limits. In medicalhistory, she was free of neurological disorders such as cerebral infarction, epilepsy, or Parkinson's disease.

After discussing with the patient, continuous epidural analgesia was selected for treatment of the herpes-zoster-induced pain. In theoutpatient department of our pain clinic, an 18-G epidural catheter was inserted 3 cm cephalad through a 17-GTuohy needle to the C6-7 interspace. The epidural space was detected by the hanging drop technique. Catheterization of the epidural space was done, and no blood or cerebrospinal fluid was aspirated through the catheter. The patient did not complain of anyparesthesia during the procedure. An epidural injection of 5 ml of 0.2% plain ropivacaine produced bilateral sensory block of the C5-7 dermatomes determined by the pinprick method. Continuous epidural infusion was started with 0.2% ropivacaine 4 ml/h and morphine sulfate 0.2 mg/hwith a portable disposable pump.

After 7 days, she visited our outpatient pain clinic and her VAS score haddecreased to 5, but she still complained of burning sensations and lancinating pain. Therefore, we administered 0.2% ropivacaine 4 ml/h, morphine sulfate 0.4 mg/h, and ketamine 1 mg/hepidurally.

On the thirdday after infusion of this analgesic regimen, she showed bilateral involuntary movement of both hands. Brief, involuntary twitching of the muscle inboth hands persisted for about 2-3 minutes intermittently, and it occurred several times an hour. An MRI on the headand cervical spine was doneand aneurologist assessed the patient's neurological deficits. However, there were no neurological deficits,no abnormal findingson the MRI, and no electrolyte imbalances in the blood sample. Therefore, we diagnosed spinal myoclonus. Therefore, we stopped epidural infusion and removed the epidural catheter. Thirty-six hours after stopping the infusion, her involuntary movement ceased. We decided to maintain her previous oral medication to control for pain and discharged her without any complications. At that time, her VAS score was 5.

DISCUSSION

Myoclonus is comprised of sudden, involuntary contractions of a group of muscles, a single muscle, or part of a muscle. Spinal, focal, or segmental myoclonus has specific features that distinguish it from other forms of more generalized myoclonus. It is often restricted to one somatic region due to the pathology at the involved level of the spinal cord. The appearance of myoclonus may take several months oryears. The possible causes are tumor, infection, trauma, and degenerative processes [2]. The pathophysiology of spinal myoclonus includes abnormal loss of inhibition from suprasegmental descending pathways, loss of inhibition from local dorsal horn interneurons, hyperactivity of contiguous anterior horn neurons, and aberrant local axon re-excitations [3].

Neuraxial high dose opioid therapy may cause spinal myoclonus [4,5]. Kloke et al. [4] reported 10 cases of myoclonus among spinal opioid therapy patients. In addition, spinal cord or nerve lesions are risk factorsof myoclonus. Medullar spinalis lesions are associated with increased risk of myoclonus because of combination of spinal-cord/nerve dysfunction and high intrathecal or systemic opioid analgesia requirement [4]. In our case, the patient did not receive a high dose of neuraxial opioid. Additionally, she did not have spinal cord lesions. Therefore, this case of spinal myoclonus is unlikely opioid induced.

Local anesthetic neurotoxicity may cause spinal myoclonus [6]. It was postulated that the inhibitory effects of the local anesthetic might have led to heightened irritability of the α-motor neurons, leading to myoclonus [7]. However, ropivacaine used in this caseis known to be the least neurotoxic local anesthetics [8].

Zoster myelitis can cause spinal myoclonus [9], butit is not a cause of myoclonus in this case sincemyoclonus appeared and disappeared asthe ketamine infusionwas started and stopped, respectively, and there were no myelitis lesions in the MRI.

Indwelling spinal or epidural catheters may cause myoclonus by irritating the spinal cord or nerve roots [10]. This can be treated immediatelyby withdrawing the offending catheter. In this case, myoclonus continued for 36 hours after catheter removal. Therefore, we excluded catheter-induced myoclonus.

Ketamine may induce seizure activity [11,12]. Ketamine may cause seizure activity either in the form of cortical electroencephalograph (EEG) or clinical seizure activity in epileptics in whom the drug activates subcortical activity [11,12]. Additionally, nutritional andenvironmental factors (smoking, alcohol, diet) and drug interactions may influence the metabolic and pharmacokinetic patterns of ketamine contributing to proconvulsant activity. Potter et al. suggested that myoclonus, as a side effect of treatment with morphine, is more likely to occur in patients taking antidepressants, antipsychotic drugs, antiemetics, or nonsteroidal inflammatory drugs for additional analgesia [13]. In this case, we added ketamine for additional analgesia just 2 days before myoclonus developed and myoclonus resolved 36 hours after catheter removal. Therefore, we postulate that the ketamine may be responsible for the spinal myoclonus.

The treatment of spinal myoclonus includesthe detection and abolition of the etiology and symptomatic treatment with benzodiazepines or anticonvulsants [3]. Discontinuation of drugs suspected of causing myoclonus and treatment of metabolic derangements may resolve some cases of myoclonus. When pharmacological treatment is indicated, anticonvulsants are the main line of treatment. Treatment of myoclonus focuses on medications that may help reduce symptoms. The various drugs used include anticonvulsants such as sodium valproate, clonazepam, and benzodiazepines, which include diazepam and midazolam [14].

In conclusion, spinal myoclonus following epidural analgesia is rare, but the clinician should watch out for this complication, especially in patients using morphine and an adjuvant drug.

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