Choreiform Movement on the Same Side after Supraclavicular Brachial Plexus Block under Ultrasonography

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Abstract
A healthy woman without any medical history, developed an irregular involuntary movement disorder of the ipsilateral upper extremity about two hours after injection of mepivacaine (480 mg) for supraclavicular brachial plexus block under ultrasound guidance. Midazolam 2 mg and fentanyl 50 µg were intravenously injected at the time of local infiltration just before the block. After neurological consultation on the movement disorder, it was assessed as a choreiform movement. The physical signs subsided for some time with the administration of midazolam or lorazepam but recurred again. The movement abnormality continued without diminution during voluntary action following verbal command. This involuntary movement decreased gradually and disappeared spontaneously twelve hours after the brachial plexus block without further treatment. Brain magnetic resonance imaging showed no abnormal results. She recovered completely without sequelae. This movement disorder seems to be caused by mechanical injury from using a long-beveled sharp needle. We report on this rare case with some presumable mechanisms.

Keywords: Brachial plexus block, Choreiform movement, Mechanical injury.

Introduction
The etiologies of neurological complications associated with nerve block include toxicity of local anesthetics or mechanical injury by a needle [1]. The former, manifesting as perioral numbness, confusion, tremor or seizure, is caused by an incidental intravascular injection of local anesthetics or a formation of a relatively toxic concentration after normal vascular absorption of local anesthetics. The former is characterized by an immediate to acute time course after injection [1]. The latter can manifest as prolonged neurologic deficits [2] or movement disorders, including myoclonus [3] or a choreiform movement disorder, as in this case. Movement disorders could be caused by a reorganization process within the spinal cord or brain triggered by peripheral nerve injury [4]. We observed a choreiform movement disorder after brachial plexus block and report on it, together with some mechanistic considerations derived from related literature in the field of neurology.

Case Report
A 22-year-old woman (160 cm, 46 kg) with postoperative contracture of the metacarpophalangeal joint of her left thumb was scheduled for a capsular release operation. A supraclavicular block was planned for anesthesia. There were no histories of systemic diseases, neurologic disorder, or familial inherited disorder. She didn’t have any special medications. The laboratory results were also normal. Monitoring involved electrocardiography, noninvasive blood pressure, and pulse oximetry.

Just before the local infiltration, midazolam 2 mg and fentanyl 50 µg were injected for sedation. At the beginning of the nerve block, her blood pressure and heart rate were 120/75 mm Hg and 65 beats/ min. A supraclavicular brachial plexus block was performed, using 40 ml of 1.2 % mepivacaine (total 480 mg) under ultrasonographic guidance. We used a 25 G, 5-cm long-beveled, sharp-ended needle (Profi needle™, Shinchang Medical Co., Seoul, Korea, Figure 1) and approached the brachial plexus with in-plane guidance. Starting from the lower trunk level, we repeatedly injected 10-15 ml of local anesthetic solutions along three points, each time after confirming no aspiration of blood. Before giving local anesthetic, we were cautious about placing the needle near the nerve bundle as much as possible without entering the epineurium. She
was communicant during the procedure and did not report any paresthesia or electric shock. Within 15 minutes after injections, it was confirmed that the block was successful by checking the motor functions or sensory areas related to four major branches (musculocutaneous, radial, median, ulnar). About 20 minutes after the block, the patient’s blood pressure and heart rate increased to 150/90 mm Hg and 110 beats/min, respectively, but she denied any dizziness or tingling sensations. The blood pressure also decreased to a normal range when the surgery began. Additional midazolam 1 mg was given intravenously to provide adequate sedation in the course of the surgery. The duration of the surgical procedure was about 40 minutes.

After the surgical procedure, the patient was transferred to the recovery room. Her mental status was a little drowsy. After ten minutes in the recovery room, she said “My body is shaking, though I am not cold.” Her body temperature was 36.7°C. Meperidine 25 mg was given intravenously to relieve the apparent shivering. However, her shivering continued, and she also showed agitation, which was treated with an intravenous injection of propofol 30 mg. After being sedated about 15 minutes, she had involuntary movements of her left arm, while the other body parts were normal. The movement was best characterized as choreiform movement following the neurologic consultation. The left arm moved back and forth quickly and irregularly with repetitive flexion-extension and rotations of the elbow. The abnormal movement continued during voluntary action following the command of the examiner (Video). She did not complain of pain or any other discomfort, and there were no other neurological symptoms except for the involuntary movement. The neurologist successively ordered intravenous midazolam 5 mg and lorazepam 4 mg, but the abnormal movement recurred again after some time of sedation.

No exacerbating or relieving factors were found. Arterial blood gas result was unremarkable (pH 7.39, PaO₂ 192 mm Hg, PaCO₂ 41 mm Hg, HCO₃⁻ 24.8 mmol/L). Also, postoperative laboratory tests, which included a complete blood cell count, chemistry, and urinalysis, showed no abnormal results. Normal sinus rhythm was seen on the electrocardiogram and the change of vital signs was not remarkable. After we observed the patient in the recovery room for two hours, the abnormal movement weakened but did not disappear completely. She became awake, fully alert and responsive, and was transferred to the general ward. Her movement abnormality disappeared while she was sleeping.
Brain magnetic imaging was taken the day after the surgery. It showed no significant abnormal lesions in brain parenchyma, including the subcortical areas. The next day, the patient experienced the same type of involuntary movement three times, lasting for about five minutes each. The symptoms disappeared gradually over 12 hours after the operation without any medication. The patient was discharged from hospital on the fifth postoperative day with complete absence of choreiform movement. A telephone interview one month later revealed that no more episodes of movement abnormality had occurred.

Discussion

The patient showed an involuntary, jerky, irregular, and random flow of muscle contractions with overall flowing dance-like movements. This hyperkinetic variety of movement disorder is compatible with chorea [5]. The rather delayed onset of this complication, its repetitive and prolonged nature, and its occurrence on the same side of the body as the nerve block all suggest that it was an etiologic process triggered by mechanical nerve injury [4,6]. If we had injected additional local anesthetic into the plexus to confirm the origin of the movement disorder, as in the articles of Assal, et al. and Shin, et al. [7,8], we could have found more concrete evidence. Also, if we had performed tests, such as nerve conduction velocity and electromyography, we could have seen other concrete evidence.

By the way, the possibility of systemic toxicity by the administered local anesthetic is quite low, considering the above characteristics of this complication and the nonexistence of this kind of problem in the literatures [1]. Regarding the possibility of drug toxicity by other drugs, there were reports about antipsychotics, dopaminergics, anticholinergics or anticonvulsants in the settings of Parkinsonism or epilepsy. But the drugs used in our case such as opioids and small doses of midazolam or propofol were not reported as possible agents [9].

Even though we have used the same type of needle without any specific complications for several years, there were literature reporting the risk of using a long-beveled, sharp-ended needle [10,11] and thus it might be a better idea to use a short-beveled and blunt-tipped needle. If the difficulty of the initial skin penetration is a problem, one might try using skin-nicking with a cutting-type needle. The use of ultrasonography with a nerve block has been welcomed recently, acknowledging the benefit of an increasing success rate. However, one systematic review concludes that the rate of neurological complications has not decreased compared to techniques not using ultrasonography [2]. Because the reasons for such results could not be detailed now, practicing physicians should know about the possibility of nerve damage while using ultrasonography. We could not find any case report for this kind of neurological complication associated with peripheral nerve block like in this case. However, though not related to nerve block, historically there have been cases of chorea describing that even small lesion or simple touch in the peripheral or central nervous system could cause choreiform movement [12,13].

Although movement disorders caused by peripheral trauma have been frequently argued from many case series in the field of neurology, little research has been done for understanding the underlying mechanisms [4,14]. Considering the rarity of this movement disorder, disproportionate to the frequency of peripheral trauma, it may be that predisposing factors like genetic susceptibility, preexisting brain dysfunction, or prior use of drugs or toxins could play roles [4,13]. In this case, there were no known suspicious factors at the preoperative stage. However, there remains the question about whether the effects of a little higher dose of mepivacaïne (480 mg) [15], midazolam (3 mg), fentanyl (50 μg), meperidine (25 mg) and propofol (30 mg), given before development of the movement disorder, could be predisposing factors. Furthermore the exact time of onset of the complication could be uncertain, because the abnormal movement developed just when the effect of the nerve block was wearing off. These questions in our case should await explanations until better understanding of this kind of problem could be obtained.

We could find some mechanistic information in the related literature in the field of neurology under the assumption of some similarity between peripheral trauma and possible direct nerve injury in our case. Regarding pathophysiology, the suggested key concepts are the aberrant input from the site of nerve injury and the ensuing central functional changes secondary to a peripheral injury, such as a reorganization process within the spinal cord, cortex, and subcortex [16]. For example, de Ceballos, et al. [17] showed that thermal injury to a hind limb of rats causes bilateral reduction of neuronal activity in the basal ganglia. Also, recent evidences show that a variety of movement disorders are characterized by defects in the sensorimotor integration involving connections between the sensorimotor cortex, thalamus, and basal ganglia, closely coupled with abnormal sensory input in the cases of peripheral trauma-related disorders [18-20]. This concept of disordered sensorimotor coordination is quite different from previous theories about movement disorders and also throws light to such a puzzled problem as in our case. Traditionally, movement disorders were explained as abnormalities of the motor systems only, but the recent theories about defects of sensorimotor integration are giving more reasonable explanations for this kind of case, which is suspicious of a movement disorder secondary to peripheral nerve injury.

The fact that the patient’s movement abnormality disappeared during sleep or with the administration of sedative drugs (midazolam and lorazepam in this case, haloperidol [21], sodium valproate [22]) does not give any direct evidence as treatment agents, because they could be regarded as being given based on empirical knowledge without a detailed mechanistic understanding. When patients recover from the effects of sedatives, they tend to repeat their previous movements, and we don’t yet have clinical evidence on whether the drugs could shorten the natural course of those complications. Furthermore, the natural course of this complication in this case was a rather benign course, so it may be a reasonable choice to observe the patient closely without any treatment.

Conclusion

In conclusion, we observed a choreiform movement disorder after an apparently successful brachial plexus block. The abnormal movement decreased over 12 hours without any treatment. For etiology, mechanical injury by a long-beveled and sharp-ended needle is suspected. Those clinical features might have originated from higher neuronal centers, but it will take further research to understand this interesting phenomenon. For adequate treatment, more research should be done on the neuropathological changes. For prevention, we recommend that a short and blunt-tipped needle be used.
References