Possible Subdural Block during Interscalene Brachial Plexus Block: A Case Report

Abstract

The interscalene block technique is widely used for a variety of procedures on the upper extremities. In general, this technique is safe and effective but is not without hazards. A case of subdural block resulting from an interscalene block is presented. In a 60-year-old female patient with carpal tunnel syndrome, sudden bradycardia, unconsciousness and respiratory arrest developed within several minutes after performing the block. At the end of the postinjectional third hour, she regained full consciousness, spontaneous breathing and full sensorial functions, except for profound anesthesia in the right arm. Within 6.5 hours of injection, right arm strength and sensation returned to normal; however, analgesia remained until the postinjectional ninth hour.

Keywords: Complications, Interscalene block, Subdural block

İnterskalen Brakiyal Pleksus Bloğu Sırasında Gelişen Muhtemel Subdural Blok: Olgu Sunumu

Özet


Anahtar Kelimeler: Subdural blok, İnterskalen blok, Komplikasyonlar
Introduction

The interscalene block (ISB) is a well-established anesthesia technique for a variety of procedures on the upper limbs. In general, this technique is safe and effective but is not without hazards. Minor complications (such as Horner’s syndrome, phrenic nerve block and hoarseness) and severe complications (involving extensive neuroaxial blocks, intravascular injection and systemic toxicity, which can result in respiratory arrest and loss of consciousness) may rarely occur [1]. In particular, performing this technique under general anesthesia could result in the injection of local anesthetic solution into the spinal cord with permanent loss of spinal cord function [2]. In this paper, we present a possible sudden subdural block, unlike the slow subdural block, which has been reported before [3], as a complication of ISB.

Case Report

A 164 cm, 67 kg, 60-year-old female who was ASA physical status I was scheduled for right carpal tunnel release (CTR). In our hospital, neurosurgeons perform CTR with a tourniquet to obtain a bloodless surgical field. For this reason, CTR is performed with ISB. ISB is carried out with 40 mL local anesthetic solution to obtain lower trunk anesthesia. If the lower trunk block is inadequate, supplementary ulnar nerve blockade or sedation might be applied. The patient’s medical history and preoperative evaluation were unremarkable, and she had no previous history of surgery or anesthesia. After written informed consent was obtained and the patient performed an overnight fast, she was premedicated with midazolam 3 mg IM and atropine 0.5 mg IM. Standard monitoring included non-invasive blood pressure, ECG and pulse oximetry. Her baseline blood pressure, heart rate and SpO2 were 100/60 mmHg, 80 beats/min and 96%, respectively.

The patient was placed in the supine position with her head turned to the left. After the injection of a skin wheal, a 22-G, 5 cm needle (Stimuplex Needle A 50, B. Braun Melsungen AG) was inserted between the bellies of the anterior and middle scalene muscles at the level of the cricoid cartilage (C6) and advanced in the medial, dorsal and slightly caudal direction, as described by Winnie [4]. Then, elbow flexion was obtained with 0.5 mA current at a 3 cm depth. After negative aspirations and a confirmed negative response to a 4 mL test dose of local anesthetic solution, a total dose of 40 mL of a mixture of equal parts bupivacaine 0.5% and prilocaine 2% was administered. At 4 mL increments, the same local anesthetic solution was injected at 30 second intervals until 20 mL had been injected. After approximately 2 minutes and 20 mL total injection, the patient was unable to lift her right arm. Slightly thereafter, she developed a sudden bradycardia (38 beats/min) and decrease in SpO2 (87%), and the injection was stopped. The patient was immediately administered 100% O2 by face mask. Then 0.5 mg atropine was administered IV to treat the bradycardia, since the heart rate did not increase. This dose was repeated, and the heart rate reached 58-60 beats/min. Five minutes later, the patient complained of difficulty breathing, and positive pressure ventilation was begun via face mask with 100% oxygen. Two minutes later, the patient lost consciousness and ceased spontaneous breathing. Meanwhile, her blood pressure was 100/70 mmHg. Her pupils were fixed and widely dilated, and there was no reaction to light. In discussion with the surgeons, we all agreed that whatever happened, the event was reversible. Because her vital signs were stable, we decided to carry on the operation after obtaining written informed consent from her husband. Approximately 20 minutes after the first injection, she was intubated with the administration of intravenous thiopental 200 mg and vecuronium 5 mg. Anesthesia was maintained with nitrous oxide 60% in oxygen with isoflurane 0.25% to avoid any potential for awareness. Vital signs remained stable throughout the operation. At the end of the surgery, the patient was transferred to the intensive care unit (ICU). After three hours she regained full consciousness. Her respiratory rate was 16 breaths/min, her SpO2 was 98% and she had a normal tidal volume; therefore, she was extubated. She did not have any deficit of her cranial nerves. On close questioning, she reported that she could not recall anything except difficulty breathing. She could not move her right arm, and there was only sensory anesthesia from C3 to T6 on her right side and from C5 to T4 on her left side. Forty minutes later, all sensory functions fully recovered, except for profound anesthesia of the right arm. Six and a half hours following injection, right arm strength and sensation returned to normal. But analgesia remained until the ninth postinjectional hour. She remained in the ICU overnight. She was subsequently transferred to the neurosurgery department and was discharged after three days without any neurological deficit.

Discussion

In all regional techniques, an incidental intravenous injection or rapid absorption can result in central nervous system toxicity. Intravascular injection of bupivacaine may lead to bradynrrhythmias. But it is also seen with hypotension, atioventricular heart block and dysrythmias, such as ventricular fibrillation [5]. In our case, there was adequate brachial plexus block and extensive bilateral neuraxial block, meaning that at least a major part of the local anesthetic was administered extravascularly. Furthermore, any sign (such as seizure or hypotension) of systemic toxicity was not observed.

This case does not match a reversible locked-in syndrome, as reported by Durrani and Winnie [6], secondary to braintem toxicity due to unintentional injection of local anesthetic into the vertebral artery. In that syndrome, apnea, hypotension, brady-cardia and seizure are seen, and consciousness is maintained despite the presence of tetraplegia and aphonia. Since in our case there was neither seizure nor hypotension and the patient was unconscious, we did not take into consideration reversible locked-in syndrome.

A sudden bradycardia after the placement of interscalene block has been attributed to overactivity of the autonomic nerv-
ous system that activates the Bezold-Jarish reflex, which results in a vasovagal response [7]. In the present case, this mechanism is out of the question because this problem is generally seen in the sitting position and with hypotension. This patient was supine position, and there was no hypotension.

Clearly this case looks like an extensive neuraxial block. Because the epidural space does not extend intracranially, only extensive spread of a large amount of local anesthetic can cause central neural blockade [8]. As mentioned above, only a little amount of anesthetic might have gone outside of the interscalene space, since adequate brachial plexus anesthesia had been obtained. In addition, anesthesia, loss of consciousness and respiratory arrest occurred very rapidly in this case, while epidural anesthesia has a slow onset. Therefore, the possibility of epidural injection is very unlikely in our case.

In high subarachnoid or subdural blocks, local anesthetics can spread intracranially; as such, sudden unconsciousness and apnea are expected to occur. Nonreactive dilated pupils can be explained with a loss of parasympathetic efferent activity from the Edinger-Westphal nucleus as a result of intracranial spread of local anesthetics [9]. Also in both blocks, bradycardia can be seen due to cervicothoracic anesthesia with blockade of the cardiac accelerator fibers (T1-T4) [4].

In high spinal anesthesia, severe hypotension occurs, which was obviously not seen in our case. A more reassuring finding to exclude subarachnoid block is that there were no bilateral motor signs. As a result, the last alternative remained in this case is subdural block.

The subdural space is a potential space between the dura and arachnoid membranes. It is narrower than the epidural space and extends intracranially, just like the subarachnoid space. The subdural space extends laterally over the nerve roots, and it is larger in the cervical region than elsewhere [10]. Subdural injections have occurred inadvertently while performing epidural [4], interscalene [1] and stellate ganglion blocks [10]. The onset of the block has been described as both fast and delayed (5-45 min after injection). In all of these cases, an unexpectedly high level of sensory block was described with the loss of consciousness lasting from one to several hours. Since the potential space is widest over the dorsal root ganglion, solution injected into this space spreads and pools posterolaterally. This situation results in sparing of motor and preganglionic sympathetic fibers, which is why sensory block is more profound than sympathetic and motor blocks [12]. This can be a good explanation for the phenomenon seen in our case in which there was profound sensory blockade with remarkable stability of the arterial blood pressure and absence of motor blockade except for in the right arm. This case differs from the characteristic features of subdural blockade, described by several authors, only in that the onset of the block was very fast. However, some atypical cases, in which rapid onset occur, have been reported by other authors [3, 4, 13-15].

Several mechanisms have been postulated to explain inadvertent neuraxial blockade occurring during interscalene brachial plexus block. The first of these is direct injection into these spaces by using a long needle directed toward the intervertebral foramen. Secondly, intraneural and perineural injection of a drug may spread into the subdural and subarachnoid spaces.
Conflict interest statement  The authors declare that they have no conflict of interest to the publication of this article.

References


