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Brain edema after ultrasound-guided supraclavicular block

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Brain edema after ultrasound-guided supraclavicular block

Supraclavicular nerve block is the most popular regional anesthesia approach for upper limb surgery. It also predispose some complications because of the adjacent neurovascular structures such as pleura, recurrent laryngeal and phrenic nerve, supraclavicular vessels, and spinal cord structures. The widespread use of ultrasound for peripheral nerve blocks has reduced the incidence of these complications. We aimed to present a case of brain edema developed after ultrasound-guided supraclavicular block which was not reported in the literature before.
A 25 years old, American Society of Anesthesia (ASA) score I, male patient was admitted to our clinic to perform the replacement of external fixator with an internal fixator. The ultrasound guidance supraclavicular nerve block was performed. After the block, we observed disorientation, blurring in consciousness, paleness, tachycardia, hypertension and tachypnea. Upon this, we performed induction for general anesthesia and intubation. After the onset of myoclonic epileptic seizure, he was re-intubated in the recovery room. Bilateral cerebral parenchyma was edematous, the sulci were faint and venous structures were prominent on the brain computerized tomography. Because of magnetic resonance imaging (MRI) finding that restricted diffusion which was compatible with cortical-subcortical acute ischemia, mannitol, dexamethasone and furosamid were started as anti-edema treatment. On the third day in intensive care unit, the patient was extubated because of improvement in consciousness and marked decrease in brain swelling on the control radiologic imaging.
It should be kept in mind that brain edema secondary to local anesthetic systemic toxicity (LAST) may develop as a complication after brachial plexus blocks.

KEY WORDS: Brain Edema, Epileptic Seizure, Supraclavicular Nerve Block, Ultrasound

Introduction

Supraclavicular block is the most frequently used regional anesthesia technique for upper extremity surgery[1]. It is superior to other brachial plexus blocks and analgesic modalities because it allows to perform nearly all upper extremity surgical procedures with a single injection and provides excellent postoperative analgesia [1,2]. However, it also predispose some complications because of the adjacent neurovascular structures such as pleura, recurrent laryngeal and phrenic nerve, supraclavicular vessels, and spinal cord structures. It may lead to laryngeal and phrenic nerve block, toxicity after intravascular injection, myotoxicity after intramuscular injection, block formation on the contralateral side and total spinal block due to epidural and intrathecal spread [2]. Using of ultrasound helps the practitioner during peripheral nerve blockades in terms of diminishing the amount of local anesthetic agent and also the risk for unwanted circumstances [1,2]. We aimed to present a case of brain edema developed after ultrasound-guided supraclavicular block which was not reported in the literature before.

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Case Report

A 25 years old ASA I male patient who was operated on under general anesthesia one week ago due to right humerus fracture was admitted to our clinic to perform the replacement of external fixator with an internal fixator. Preoperative laboratory findings were normal. After the patient signed the informed consent, we administered 2 mg midazolam for premedication.

We performed supraclavicular blockade by the help of a linear transducer over the supraclavicular fossa at the coronal oblique plane and just superior to the midclavicular point. The induction of blockade was in the semi-sitting position and the patient's head turned from the blocked side A 22-gauge insulated block needle was inserted in-plane (lateral to medial) to the ultrasound probe. The brachial plexus was visualized as compact group of nerves which were hypo-echoic, circular or oval, located lateral and superficial to the subclavian artery and superior to the first rib. A local anaesthetic of 30 ml (bupivacaine 0.5%, 20 ml and lidocaine 2%, 10ml) was injected. During the blockade, negative aspiration was performed before each 3 ml of drug administration and perineural hypoechoic image of the local anesthesic was observed on ultrasonography. After obtaining adequate level of anesthesia after the block, the patient was given left lateral decubitus position for surgery. Then, we observed disorientation, blurring in consciousness, paleness, cold sweating, tachycardia (138 beats/min.), hypertensiob (175/89 mmHg) and tachypnea (33/min.); just before the operation. Upon this, we performed induction for general anesthesia and intubation. Surgery was postponed because of the change in patient's general condition. Then, the intubated patient was taken to the post-anesthesia care unit. After detecting blood gas and hemodynamic parameters as normal, we decided to recover and awake the patient.

The patient was confused but had normal respiratory parameters. After the onset of myoclonic epileptic seizure, he was re-intubated. Bilateral cerebral parenchyma was edematous, the sulci were faint and venous structures were prominent on the brain computerized tomography, therefore clexane 0,4 and 2x500 mg levetiracetam was started for possible venous sinus thrombosis. After removal of the external fixator, magnetic resonance imaging (MRI) (Fig. 1) and MR angiography were performed. Flow phenomena, configurations, and calibrations of the cerebral venous sinuses were reported as normal on MR angiography. Because of the finding of restricted diffusion which was compatible with cortical-subcortical acute ischemia, mannitol, dexamethasone and furosamid were started as anti-edema treatment. On the third day in intensive care unit, the patient was extubated because of improvement in consciousness and marked decrease in brain swelling on the control radiologic imaging (Fig. 2). The patient whose hemodynamic parameters and neurological examination were normal was discharged on the seventh day. The surgery was performed two months later under general anesthesia without any problem.

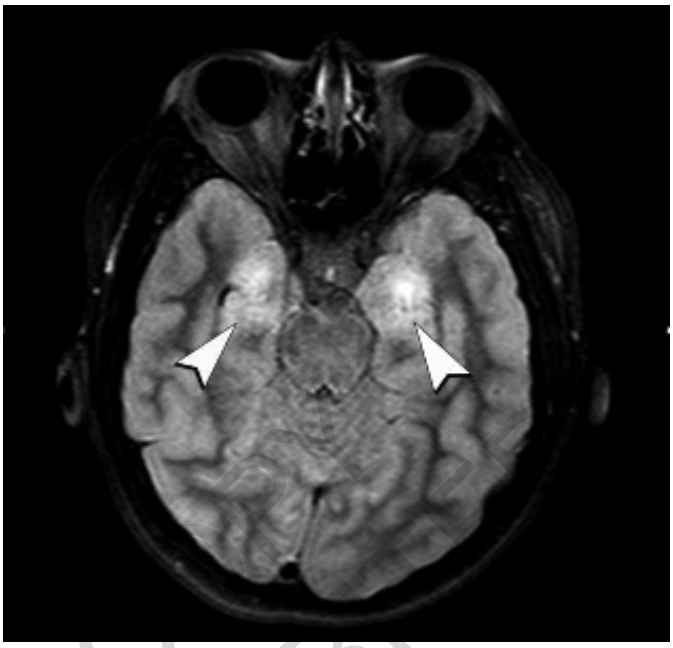

Fig. 1: MRI performed one day after the procedure revealed patchy cortical - subcortical hyperintensity (arrows) in bilateral hippocampal regions and restriction of diffusion consistent with acute ischemia in T2-FLAIR sequences.

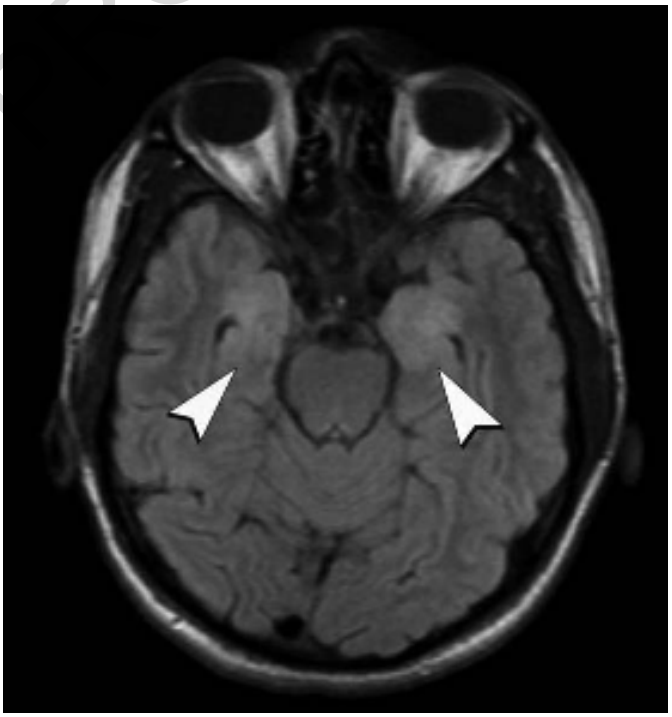

Fig. 2: MRI performed On the third days later showed that the findings regressed significantly.

Discussion

The rate of local anesthetic systemic toxicity (LAST) in peripheral nerve blockades has been announced as 0,025% [3]. Groban et. al.[4] have stated that local anesthetics are not safe molecules and that closer follow-up is crucial. Agitation after brachial plexus block usually occurs secondary to pain and anxiety. Occasionally, it may constitute the preliminaey findings of LAST. Administering 2 mg midazolam for premedication and sedation may have overshadowed the emergence of these preliminary findings.

While performing the blockade, we tried to confirm whether we were not in any vessel by negative aspiration at every 3-4 ml of injection. However, Ichikawa et al.[5] presented a case with generalized convulsion which occured after 3 minutes after the interscalene block despite negative aspiration and they stated that negative aspiration did not prevent this complication. In addition, the perineural hypoechoic appearance of local anesthetic and the formation of sensory block after the blockade suggested us that local anesthetics did not spread intravascularly in our patient and caused us to get away from the diagnosis of LAST.

Plasma concentration of bupivacaine which causes cardiovascular toxicity is generally assumed to be 2-4 µg/mL [6,7]. Jong et al.[8] observed convulsions in cats at level of 3.6±0.7 µg/mL of bupivacaine infused for 5.3±2.1 minutes. In a study with volunteers, it was reported that the threshold plasma concentration of bupivacaine leading to central nervous system (CNS) toxicity was 2.1 µg/mL[9,10]. Although the changes in consciousness in these patients are likely to be due to intravascular injections, absorption by the surrounding vascular structures may have contributed to the increase in plasma concentration of bupivacaine. Plasma concentration of the local anesthetic was not evaluated in our hospital.

Sytemic effects of local anesthetic toxicity such as prolonged bradycardia and hypotension which were reported by Nelson et al.[11] were not observed in our patient. On the contrary, we observed tachycardia and hypertension. We suppose that cardiovascular effects such as bradycardia and hypotension may not have occurred due to our patient's young age and that hypertension was secondary to his agitation. Kiuchi et al.[12] reported that the risk of systemic toxicity is associated with age and low in younger rats. In a retrospective evaluation of 659 interscalene blocks, risk for CNS toxicity was reported as 0.76% whereas cardiovascular toxicity was rare[13]. Apart from bupivacaine, other potent local anesthetics in amide group such as ropivacaine and levobupivacaine have been reported to cause convulsions in the literature[14,15].

Some studies also demonstrated that propofol may have protective effect in bupivacaine-related cardiotoxicity [16-19]. Ohmura et al.[20] suggested that propofol may be protective against cardiodepressive effects of bupivacaine. In a similar study, pre-treatment with propofol containing 10% intralipid has been reported to delay the cardiodepressive effect of bupivacaine and increase the threshold for asystole development[21]. There are publications reporting the beneficial effects of propofol administration when CNS symptoms or convulsions occured [22,23]. In our case, we think that symptoms of LAST were masked by administering midazolam before the procedure for sedation, propofol against the agitations and propofol ultiva in the post-anesthesia care unit for sedation. Therefore, this may have prevented us from giving the decision of 20% lipid solution treatment.

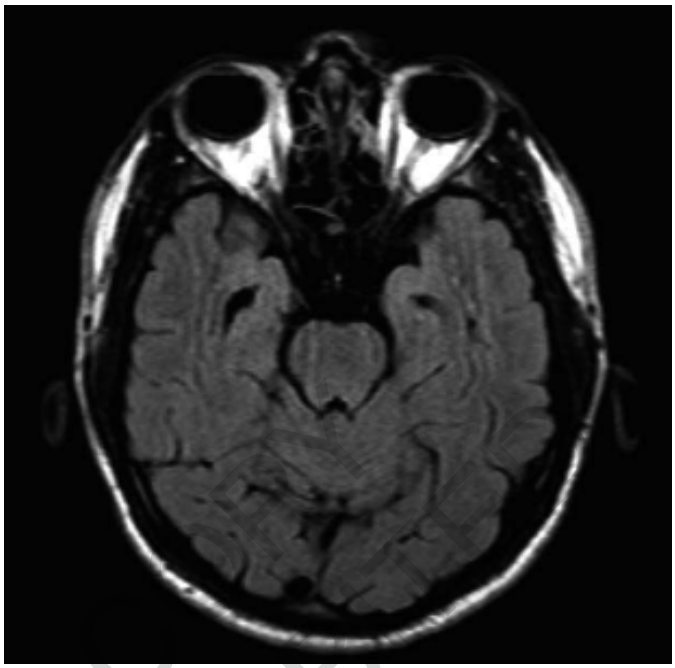

Fig. 3: MRI performed after six months later revealed complete disappearance of the findings.

In some cadaveric studies, it has been shown that there was volume-independent spread to the epidural space during ultrasound-guided brachial plexus block and this may result in bilateral loss of neural functions [24,25]. In our patient, giving lateral decubitus position after the block may have caused epidural or intrathecal spread of the local anesthetic and secondary cerebral edema. However, the blockage was restricted only to the arm on the surgical site and hypotension or bradycardia due to epidural or spinal block did not develop.

We suppose that spread of local anesthetics to the intravascular space in the later period resulted in local anesthetic toxicity and secondary brain edema and convulsions.

MRI study performed after six months (Fig. 3) revealed no pathological finding. During this period, the patient had no complaints or epilepthical attacs.

Conclusion

Brain edema secondary to LAST may develop as a complication after brachial plexus blocks should be kept in mind. During or after blocking, deep sedation or general anesthesia may mask the clinical signs in patients with altered consciousness. Detecting plasma concentration of local anesthetic can be useful in differential diagnosis. If there is any suspicion of LAST, 20% lipid solution should be administered first before any effort on differential diagnosis in terms of avoiding complications.

Riassunto

Il blocco nervoso sopraclavicolare è la più diffusa tipologia di anestesia locale nella chirurgia dell'arto superiore. Può anche indurre alcune complicazioni a causa della vicinanza con strutture neurovascolari quali la pleura, il nervo laringeo e frenico ricorrente, i vasi sopraclavicolari e le strutture del midollo spinale. La diffusione nell'uso degli ultrasuoni per effettuare il blocco dei nervi periferici ha ridotto l'incidenza di queste complicanze. Il nostro obiettivo è presentare un caso di edema cerebrale sviluppatosi a seguito di un blocco sopraclavicolare ecoguidato, caso non ancora riportato in letteratura. Un paziente maschio di 25 anni (categoria di rischio 1, secondo l'American Society of Anesthesia, ASA) è stato ricoverato nella nostra clinica per eseguire la sostituzione del fissatore esterno con un fissatore interno. È stato effettuato il blocco nervoso sopraclavicolare ecoguidato. Dopo il blocco, abbiamo osservato disorientamento, alterazione della conoscenza, pallore, tachicardia, ipertensione e tachipnea. A quel punto, abbiamo eseguito un'anestesia generale ed intubato il paziente. Dopo l'insorgenza di una crisi epilettica mioclonica è stato reintubato nella stanza di risveglio. Il parenchima cerebrale bilaterale era edematoso, con solchi indistinti e strutture venose che risultavano prominenti nella tomografia computerizzata cerebrale. Nella risonanza magnetica (RMN) si evidenziava una diffusione limitata compatibile con ischemia acuta corticale-subcorticale, ed è stato dunque avviato un trattamento antiedema a base di mannitolo, dexametasone e furosemide. Al terzo giorno di terapia intensiva il paziente è stato estubato, grazie ad una ripresa di conoscenza ed alla marcata riduzione dell'edema cerebrale sull'imaging radiologico di controllo. Va tenuta presente la possibilità che si sviluppi un edema cerebrale causato da tossicità sistemica da anestetici locali (LAST), come complicazione in seguito ai blocchi del plesso brachiale.

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