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Case Report

Horner's Syndrome Caused by Ultrasound-Guided Supraclavicular Nerve Block

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Abstract

Horner's syndrome, caused by supraclavicular nerve block, is a rare case. It is mostly expected after interscalene nerve block, caused by anatomic reasons. Horner's syndrome results from neuronal paralysis of the post-ganglionic cervical sympathetic chain. For anatomic reasons, interscalene nerve block is very common but very uncommon in the case of supraclavicular nerve block. Horner's syndrome results from the paralysis of the ipsilateral sympathetic cervical chain. One common cause is interscalene nerve block. This effect occurs frequently due to anatomical proximity – the brachial plexus nerves in the interscalene region are situated very close to the sympathetic cervical chain. When a local anesthetic is injected near the interscalene nerves, it can spread to surrounding tissues, including the sympathetic chain. In contrast, with a supraclavicular nerve block, this effect is extremely rare. The rarity is due to the anatomical distance between the supraclavicular nerves and the cervical sympathetic chain, as well as the presence of a thick fascial layer surrounding the supraclavicular nerves, which prevents the spread of local anesthetic to the upper tissues.

In this case, the unusual effect of supraclavicular nerve block was revealed as a Horner's syndrome soon after injection of local anesthetic. There are a few reasons explaining this outcome. In one case, an anatomic-short neck can cause rapid distribution of local anesthetic through surrounding tissues. Another reason might be fat tissue, as local anesthetics are fat-soluble agents, and rapid injection of local anesthetics can be a reason for the upward distribution of medication. This case is important to understand what might be expected, even in cases when it is unusual, and inform the patient in advance to avoid any incomprehension after an operation.

Keywords: Horner`s syndrome, supraclavicular nerve block, ultrasound

Introduction

Horner's syndrome is a result of a lesion of an oculosympathetic pathway. Lesions might be central, pre-ganglionic, and post-ganglionic (1). Horner's syndrome might be caused by different reasons. During brachial plexus, nerve block voltage-gated sodium channels are reversibly blocked by local anesthetics. By this mechanism, sodium entrance is intracellularly being stopped, and nerve excitation cannot happen (2). Of course, local anesthetics are not sensitive only to target nerves, and if their spread happens through surrounding tissues, other nerves are also being blocked. This happens in some cases of interscalene nerve block, and surrounding nerves, such as recurrent laryngeal nerve or stellate ganglion, might be blocked. Horner's syndrome, caused by post-ganglionic neuron damage, occurs after brachial plexus nerve block, which begins from the superior cervical ganglion and reaches the ophthalmic branch of the trigeminal nerve. On this pathway, it also passes through the carotid plexus (2).

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Otherwise healthy, a 32-year-old male (BW 96 kg, height 183, ASA class) was prepared for elective operation and ulnar nerve decompression (Fig. 1). The operation was performed under tourniquet compression. For this operation, the brachial plexus block was selected by supraclavicular approach under ultrasound guidance.

Preoperatively, an 18-gauge intravenous catheter was inserted and started natural saline infusion. Thirty minutes before the operation, oral midazolam 7.5 mg (manufacturer: La Roche, Basel, Switzerland) was given orally to the patient. The patient was transferred to the operative room, where standard non-invasive monitoring was started: blood pressure 137/85 mmHg, heart rate 87 bpm, SpO2 98, and respiratory rate 16. The patient was in the supine position, and his head was turned to the contra-lateral side of the needle injection. By linear US probe, supraclavicular brachial plexus was visualized, and local anesthetic 1% lidocaine 2.0 mL (manufacturer: Dentafill plus, Tashkent, Uzbekistan) was injected into the skin and subcutaneous tissue, after local anesthetic 50 mm stimulator needle was inserted under US guidance. When the needle was in perineural space, 1 mL of saline was injected through negative aspirate. Once the needle was in the correct place, 0.5% ropivacaine 15 mL (manufacturer: Astra-Zeneca, AB Sweden, code: 22772) was injected, and the single shot block was performed.

After 15–20 min, complete sensory and approximately full motor blocks were achieved. The operation was provided without any discomfort for the patient, but 30 min later, after a single shot injection, the patient developed Horner's syndrome on the ipsilateral site of injection, revealing ptosis, meiosis, conjunctival hyperemia, and anhidrosis (Fig. 1).

Figure 1 Horner's syndrome of the left eye demonstrated ptosis, miosis, and anhidrosis. Horner's syndrome was resolved after 7 h of single shot injection.

Discussion

The brachial plexus nerve block is a type of regional anesthesia that allows better post-operative pain control, reduces risks of nausea and vomiting (3,4), and has negligible effects on organ system function (5), especially in patients with very low ejection fraction (6), pulmonary fibrosis (7), renal (8,9) or hepatic failure (10). Less side effects allow early post-operative recovery and discharge of the patient (11-13).

The location of the lesion along the oculosympathetic pathway determines whether Horner's syndrome is classified as central, pre-ganglionic, or post-ganglionic. Central Horner's syndrome is caused by damage to the first-order neuron within the central nervous system and is typically associated with other neurological signs and symptoms. Pre-ganglionic Horner's syndrome results from a lesion of the second-order neuron, which exits the spinal cord and ascends the cervical sympathetic chain to synapse in the superior cervical ganglion. Post-ganglionic Horner's syndrome occurs due to damage to the third-order neuron, which exits the superior cervical ganglion, travels along the carotid plexus into the skull, and enters the orbit through the ophthalmic division of the trigeminal nerve.

Horner's syndrome is clinically characterized by ptosis, miosis, and anhidrosis on the ipsilateral side, resulting from the blockade of post-ganglionic neurons in the superior sympathetic cervical chain of the stellate ganglion. Paralysis might be induced by different reasons, such as surgery, tumors, and hematoma (14,15).

The supraclavicular nerve block is used for the advantages of less side effects, such as phrenic nerve block or Horner's syndrome (16,17). Developing Horner's syndrome after a supraclavicular nerve block is extremely rare. Injection of local anesthetic into the nerve sheath prohibits the spreading of solution up through the stellate ganglion (18).

This case is a rare exception in which Horner's syndrome is caused by the supraclavicular nerve block, not by inter-

Figure 1. Horner's syndrome of the left eye..

scalene. There are a few reasons explaining this outcome. In one case, an anatomic-short neck can cause rapid distribution of local anesthetic through surrounding tissues. Another reason might be fat tissue, as local anesthetics are fat-soluble agents and rapid injection of local anesthetic can be the reason for more upward distribution of medication.

Conclusion

Horner's syndrome after brachial plexus nerve blocks is a benign side effect of anesthesia as it is caused by the blockade of nerves from local anesthesia. It will resolve completely after several hours. Horner's syndrome is a common outcome of brachial plexus nerve block by interscalene approach or after cervical epidural analgesia, caused by transient paralysis of the sympathetic cervical chain, so-called stellate ganglion. Important to understand that Horner's syndrome might be induced in rare cases by supraclavicular nerve block and prevented by gentle injection of medication to avoid the rapid spread of substance through fat tissue, especially in obese patients.

Disclosures

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