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Case Report

Horner's syndrome after supraclavicular continuous nerve block through catheter - A rare case report

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ABSTRACT:
Horner's syndrome happens due to ipsilateral sympathetic cervical chain paralysis that maybe caused by surgery, drugs specially with high concentrations of local anesthetics, compression on the chain by hematoma or tumor, also inadequate positioning of the patient during the surgery. incidence rate is 100% in patients undergoing brachial plexus interscalene block and can rarely in patients with other types of blocks as supraclavicular blocks.

Keywords: Horner's syndrome, ipsilateral sympathetic, cervical chain

INTRODUCTION:

Horner's syndrome, also known as oculo-sympathetic paresis, is a combination of symptoms that arises when a group of nerves known as the sympathetic trunk is damaged. The signs and symptoms occur on the same side (ipsilateral) as it is a lesion of the sympathetic trunk.
The nerves of the sympathetic trunk arise from the spinal cord in the chest, and from there ascend to the neck and face. The nerves are part of the sympathetic nervous system, a division of the autonomic nervous system.
Signs include the following, ptosis, anhidrosis, miosis, Enophthalmos, inability to completely close or open the eyelid, facial flushing, headaches, loss of Cilio-spinal reflex and bloodshot conjunctiva.

Interruption of sympathetic pathways leads to several implications. It inactivates the dilator muscle and thereby produces miosis. It inactivates the superior tarsal muscle which produces ptosis. It reduces sweat secretion in the face. Patients may have apparent enophthalmos, but this is not always the case. The ptosis from inactivation of the superior tarsal muscle causes the eye to appear sunken in, but when actually measured, enophthalmos is not present.
Sometimes there is flushing on the affected side of the face due to dilation of blood vessels under the skin. The pupil's light reflex is maintained as this is controlled via the parasympathetic nervous system.
Horner's syndrome is usually acquired as a result of disease, but may also can be congenital (inborn, associated with heterochromatic iris) or iatrogenic (caused by medical treatment). In rare cases, Horner's syndrome may be the result of repeated, minor head trauma, such as being hit with a soccer ball. Although most causes are relatively benign, Horner's syndrome may reflect serious disease in the neck or chest (such as a Pancoast tumor (tumor in the apex of the lung) or thyrocervical venous dilatation).
Causes can be divided according to the presence and location of anhidrosis.

Preganglionic (anhidrosis of face) which can be caused by Cervical rib causing traction on stellate, Thyroid carcinoma, Thyroidectomy, Goiter, Bronchogenic carcinoma of the superior fissure (Pancoast tumor) on apex of lung, Klumpke paralysis, Trauma base of neck, sometimes surgery and aneurysm. Postganglionic where no anhidrosis is noticed as in Cluster headache, Cavernous sinus thrombosis, Middle ear infection, Sympathectomy and in this case report Nerve blocks, such as cervical plexus block, stellate ganglion or interscalene block and very rarely supraclavicular block.

Case Presentation:

After his written consent, a 26-year-old healthy man (80 kg), classified as American Society of Anesthesiologists physical status I E, was scheduled for urgent reimplantation of semi amputated left thumb. The patient had a grinder injury resulting in partial amputation of the left thumb. He presented to the emergency

department within 2 hours of the injury and was taken to operating room within 2 hours of admission. He was fasting for 5 hours only. IV cannula was inserted in right hand dorsum from emergency. He was given general anesthesia with rapid sequence intubation as the surgery was urgent. IV metoclopramide 10mg was administered as premedication. He was induced with fentanyl 200mcg, propofol 150mg and succinylcholine 100mg. Airway was secured with oral endotracheal tube size 8 fixed at 21 cm at angle of mouth. Muscle relaxation was maintained with rocuronium bolus 50 mg followed by another dose of 10mg. Analgesia was topped with intermittent doses of fentanyl 100mcg, making a total of 500mcg for the procedure that lasted 3 hours. Unfractionated heparin 2000 units were administered intravenously in the third hour of surgery by order of surgeon. Tourniquet time was 97 minutes. For the whole procedure the patient was routinely monitored with electrocardiogram (ECG), noninvasive blood pressure (NIBP) measurement, end tidal CO2 and pulse oximetry (SpO2). The patient was extubated at the end of procedure after reversing neuromuscular blockade with sugammadex 200mg and shifted to post anesthesia care unit. As per unit protocol, it was decided to insert supraclavicular nerve block catheter for postoperative analgesia and vasodilation as the procedure involved revascularization of the thumb.

In the post anesthesia care unit, the need for supraclavicular catheter was explained to the patient once he was fully awake. He agreed for the procedure. The patient was in supine position, with the head facing away from the left side, and the arm were adducted.

Following the positioning, the area on the supraclavicular region was disinfected. A 6 to 15 MHz linear probe (Sonosite Xporte USA) was placed in the supraclavicular area. Under sterile conditions and after subcutaneous infiltration, an 18 G insulated needle was inserted and advanced using in-plane needle-probe alignment. Once needle was in proper position, injection of 25% dextrose selectively surrounded each sonographically imaged brachial plexus cord. An 18 G nerve block catheter (Sonolong 18 G * 50 mm) was inserted through the needle and 20ml of 0.2% ropivacaine was injected. The nerve catheter was secured in place (6cm at skin) with glue and dressing applied. Block onset time was 10 minutes. There was no change in vital parameters. Ropivacaine infusion was started. 0.2% ropivacaine 200ml bag was used, in a micrel pump with settings of 5ml/hour basal infusion, bolus 5ml, lockout time 30 minutes and a 4-hour dose limit of 30ml. The infusion was continued, and the patient was discharged from PACU to ward with infusion ongoing

Anesthesia follow up on postoperative day 1 morning revealed no abnormality. He had mild sensory deficit of left-hand dorsum which was considered normal as the bolus local anesthetic was administered @ 20 00 hours. Call was received from ward nurse @ 18 00 hours on POD 1 that the patient was complaining of stuffiness of left side of face and ptosis. Patient was attended immediately. Examination findings included left eye ptosis, left pupil constricted 2mm, right pupil 4mm, both reacting to light. He had enophthalmos of the left eye also. Immediate actions included stopping the ropivacaine infusion and reassuring the patient. The patient was reassessed after 2 hours for relief of symptoms. He was able to open the left eye and had still not started experiencing any pain from the stoppage of the ropivacaine infusion. Pupils were left 2 mm, right 4mm. After another 2 hours, the patient was reassessed again. He was able to open the left eye completely. Pupils were left 3mm and right 4 mm. He started complaining of gradual onset of pain in the operative site with pain score of 1-2/10. Under ultrasonographic guidance, normal saline 10 ml was injected through the supraclavicular catheter to assess the spread of the solution beneath the supraclavicular plexus and no hematoma was seen. Pain was managed with systemic analgesics. Paracetamol 1g intravenously 8 hourly and parecoxib 40mg intravenously 12 hourly. The ropivacaine infusion was not restarted to allow for complete resolution of the Horner's syndrome and the nurse was instructed to look for resolution of symptoms.

On POD 2 morning rounds, the patient was seen to be completely free of symptoms of Horner's syndrome and ropivacaine infusion was restarted with settings of 3 ml/hr basal infusion, bolus 3 ml, lockout time 30 minutes and 4 hours dose limit of 22 ml. Nurse was instructed to look for symptoms of Horner's syndrome. Rest of the postoperative period was uneventful. He consumed a total of 445ml of ropivacaine 0.2% over a period of 6 days. Supraclavicular catheter was removed on the 6th postoperative day and he was discharged on the 7th postoperative with no residual sensorimotor deficit or Horner's syndrome.

DISCUSSION:

This is a rare case report of Horner's syndrome occurring almost 24 hours after placement of supraclavicular nerve block catheter. Incidence of Horner's syndrome after brachial plexus block (supraclavicular and infraclavicular) has been reported to vary from 4% Kilka et al. [1] 7%, Rettig et al. [3] 12%, and Neuburger et al. [4] 1%

It's believed that Horner's syndrome is happening due to prevertebral spread of local anesthetics in the paravertebral spaces with involvement of the sympathetic nerves and the communicating cervical nerve trunks. Ipsilateral sympathetic cervical chain paralysis happens because of blockage or compression of these nerves. Horner's syndrome can be classified as central, preganglionic or postganglionic according to the location

of the lesion along the sympathetic pathway which supplies the ocular system. Central Horner's syndrome is usually associated with other neurological signs and symptoms as this is caused by damage to the first order neuron within the central nervous system. A lesion on the second order neuron as in injury is at the level of spinal cord and the ascending cervical sympathetic chain this results on Preganglionic Horner's syndrome. Finally, A lesion on third order neuron at the level of superior cervical ganglion or at the level of carotid plexus carrying the trigeminal nerve supply to the face results into postganglionic Horner's syndrome.

For instance, Winnie [2] believes that the brachial plexus is completely encased in a continuous tubular sheath from the interscalene groove to the axilla. On the other hand, Beck et al [5] showed, in an anatomical study, that connective tissue thickening could divide the neurovascular space of the brachial plexus into two compartments, possibly explaining the unidirectional spreading from the supraclavicular space to the infraclavicular space, which would prevent such passage in the opposite direction. Another possible explanation suggests that the infraclavicular neurovascular space septa forms separate compartments around individual nerves of the plexus [6]

When the rates of complications between the supraclavicular and infraclavicular approaches are compared. Impairment in diaphragmatic movements can apparently be rated as 100% for interscalene, 50% to 77% for supraclavicular, 24% to 26% for proximal infraclavicular and 0% for more distal infraclavicular blocks [3, 7, 8, 9]. According to Rettig et al. [3], Horner's syndrome is a clinically significant sign (100%) that predicts changes in hemi diaphragmatic movement; however, in their patients, changes in hemi diaphragmatic movement were also observed without Horner's syndrome. They suggested that the phrenic nerve and sympathetic trunk are more separated. We did not observe altered respiratory functions or dysphonia in our patient.

Ilfeld et al [10] published a series that examined patients managed with continuous postoperative infraclavicular local anesthetic infusion. They did not mention any signs of Horner's syndrome among the adverse events list in this cohort of 30 patients who benefited from three different regimens of continuous postoperative infraclavicular perineural 0.2% ropivacaine (infusion duration was from 13 hours to 1 hours; 125-500 mL total ropivacaine was administered)

Weisman et all (11) in a study comparing continuous interscalene and supraclavicular blocks for arthroscopic shoulder surgery demonstrated Horner's syndrome in 21% of the interscalene but only 3% of the supraclavicular group on POD1

CONCLUSION:

Although the very low rate of Horner's syndrome is associated with supraclavicular block, but still it can happen.

In medically fit patients, this may not cause significant issues, but in certain patients with co-existing respiratory comorbidity with hemi diaphragmatic involvement because of the block, this could be serious and cause life threatening conditions.

Regular follow up after insertion of the catheter for motor, sensory and any related condition like Horner's syndrome can significantly reduce the complications that might occur.

REFERENCES:

1. Kilka HG, Geiger P, Mehrkens HH. Infraclavicular vertical brachial plexus blockade. A new method for anesthesia of the upper extremity. An anatomical and clinical study. Anesthesist 1995;44:339 - 44.
2. Winnie AP. Interscalene brachial plexus block. Anesth Analg 1970;49:455 - 66.
3. H. C. Rettig, M. J. M. Gielen, E. Boersma, J. Klein, and G. J. Groen, "Vertical infraclavicular block of the brachial plexus: effects on hemidiaphragmatic movement and ventilatory function," Regional Anesthesia and Pain Medicine, vol. 30, no. 6, pp. 529–535, 2005.
4. M. Neuburger, H. Kaiser, I. Rembold-Schuster, and H. Landes, "Vertical infraclavicular block of brachial plexus—a clinical study to the reliability of a new method for plexus anaesthesia of the upper extremity," Anaesthesist, vol. 47, no. 7, pp. 595– 599, 1998.
5. H. Beck, J. S. Esch, A. Dziadzka, and W. Lierse, "The dual compartment construction of the brachial plexus sheath: an anatomical study and its clinical implications," Regional Anesthesia and Pain Medicine, vol. 15, article 47, 1990
6. B. M. Ilfeld, T. E. Morey, and F. K. Enneking, "Infraclavicular perineural local anesthetic infusion, a comparison of three dosing regimens for postoperative analgesia," Anesthesiology , vol. 100, no. 2, pp. 395–402, 2004.
7. W. F. Urmey, K. H. Talts, and N. E. Sharrock, "One hundred percent incidence of hemidiaphragmatic paresis associated with interscalene brachial plexus anesthesia as diagnosed by ultrasonography," Anesthesia and Analgesia, vol. 72, no. 4, pp. 498–503, 1991.

8. J. M. Neal, J. M. Moore, D. J. Kopacz, S. S. Liu, D. J. Kramer, and J. J. Plorde, "Quantitative analysis of respiratory, motor, and sensory function after supraclavicular block," Anesthesia and Analgesia, vol. 86, no. 6, pp. 1239–1244, 1998.
9. J. Rodr´ıguez, M. Barcena, and J. ´ Alvarez, "Restricted infra-clavicular distribution of the local anesthetic solution after infraclavicular brachial plexus block," Regional Anesthesia and Pain Medicine, vol. 28, no. 1, pp. 33–36, 2003.
10. Ilfeld BM, Morey TE, Enneking FK. Infraclavicular perineural local anesthetic infusion, a comparison of three dosing regimens for postoperative analgesia. Anesthesiology 2004;100:395 - 402.
11. T. Wiesmann , C. Feldmann , H. H. Muller Phrenic palsy and analgesic quality of continuous supraclavicular vs. interscalene plexus blocks after shoulder surgery. Acta Anaesthesiologica Scandinavica 60 (2016) 1142–1151