

CASE REPORT

A case of unilateral Horner's syndrome after combined spinal epidural anesthesia with ropivacaine 10 mg/mL for cesarean section

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SUMMARY. We report a case of transient unilateral Horner's syndrome during the setting of combined spinal epidural anesthesia for elective cesarean section using ropivacaine 10 mg/mL. The pathophysiology of Horner's syndrome and the possible mechanisms in relation to combined spinal epidural anesthesia are also presented.

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INTRODUCTION

Horner's syndrome is a triad of miosis, ptosis and enophthalmos commonly associated with vasodilatation (facial flushing), anhydrosis and nasal stuffiness. Horner's syndrome has been reported following several regional anaesthetic techniques, including brachial plexus block¹ and thoracic and lumbar epidural anesthesia.²⁻⁴ Several cases have been reported in obstetric patients using bupivacaine and ropivacaine epidurally^{5,6} but there are no reports of Horner's syndrome following spinal injection of a local anesthetic in the obstetric setting. Moreover we could not find any data in the available literature (MEDLINE) regarding the occurrence of Horner's syndrome after subarachnoid injection of ropivacaine for cesarean section.

We report a case in which a patient developed Horner's syndrome after the subarachnoid injection of ropivacaine as a single agent for cesarean section.

CASE REPORT

A 27-year-old, 75 kg, 168-cm primigravida was scheduled for cesarean section because of cephalopelvic dis-

proportion. Ringer's lactate 1000-1300 mL was infused, then, with the patient in the sitting position, a combined spinal-epidural was sited at L3/4 lumbar interspace using the needle through needle technique. The epidural space was located with a 17-gauge Tuohy needle using loss of resistance to saline and 1% ropivacaine 2 mL was given intrathecally via a 25-gauge Whitacre spinal needle, followed by insertion of an epidural catheter. The patient was turned on to the left lateral position. There were no episodes of hypotension. The level of the block to cold sensation was ascertained at T4 bilaterally just before the cesarean section. A healthy baby was born with 1- and 5-min Apgar scores 9 and 10, respectively.

Ten minutes after the delivery, and 20 min after the spinal injection of ropivacaine, the patient complained of heaviness of the left eye lid, swelling of the left eye and loss of strength of the left arm. Examination revealed left miosis, ptosis and conjunctival hyperemia. A diagnosis of left Horner's syndrome was assumed. The sensory block to cold was now T3-4 on the left side and at T4-5 on the right. A consultant neurologist was called and confirmed the initial diagnosis.

The patient was transferred for CT scan and intracranial pathology was excluded, as there were no abnormal findings. She was then transferred to the recovery room for closer observation. As the block started to subside the patient noticed an improvement of her symptoms. Once the block had completely receded, about 4-6 h later, there were no neurological symptoms. Subsequent analgesia was provided using intramuscular morphine sulfate.

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DISCUSSION

To date there have been no reports of Horner's syndrome in association with spinal anesthesia in obstetric practice. In contrast, Horner's syndrome is often reported following epidural blockade, and the incidence has been found to be 1.33% associated with epidural analgesia for labor and 4% with epidural anesthesia for cesarean section.⁷

In our case, Horner's syndrome manifested with a sensory analgesia to the T3-4 level but it appears that sympathetic blockade rose to a higher segmental level. The sympathetic nerve supply for the region arises from the intermediolateral grey column of C6 through T1.

Ray et al.⁸ found a substantial individual variability in the innervation of the dilating fibers of the iris. The sympathetic innervation controlling of the dilator pupillae and opening of the eye was found to travel through one or more spinal roots between the levels of C8 and T4. They also showed that stimulation of the anterior spinal roots between T1 and T4 induced dilation of the homolateral pupil. These fibers, after passing through the white rami communicantes, continue to the cervical sympathetic chain and ascend through the stellate and middle cervical ganglia to terminate in the superior cervical ganglion. Unmyelinated fibers leave this ganglion and form the internal carotid plexus, which further divides, giving rise to the cavernous plexus. Fibers from this plexus enter the orbit and send branches to the superior rectus and the levator palpebrae superioris muscles. Some fibers innervate the dilator pupillae through the long ciliary nerves.

Paralysis of these various sympathetic pathways causes unopposed parasympathetic tone and results in miosis, ptosis and enophthalmos. The occurrence of Horner's syndrome in the absence of sensory blockade may be explained by the high sensitivity of sympathetic nerve fibers to local anesthetics⁹ and the fact that the sympathetic blockade appears to be more cephalad than the sensory. In addition, distension of the epidural veins during pregnancy, which reduces the volume of the epidural space and Cerebrospinal Fluid (CSF), will favor cranial spread of local anesthetics.

It is known that sympathetic responses during spinal block can be depressed at levels well above those of sensory block (two to six spinal segments higher).¹⁰ The preganglionic B sympathetic fibres are anatomically available to spinal blockade and, according to Heavner and de Jong, are more easily blocked than other types of fibers,⁹ although Bengtsson et al. report that B fibers appear to be relatively resistant to spinal blockade.¹⁰

Patients with extensive sympathetic blockade would be expected to have pronounced hypotension which is absent in most of the patients reported.³ A possible explanation could be the adequate fluid administration

(1000-1500 mL) before the subarachnoid injection, which is critical to prevent the disastrous effect of hypotension due to high sympathetic blockade.

The Horner's syndrome in our case was unilateral, probably because of the left lateral position (ropivacaine is slightly hypobaric with the baricity of 0.9998 at 37 °C) and this is in agreement with previous reports of unilateral Horner's syndrome associated with labor epidurals.¹¹ For the same reason, the injection of ropivacaine may result in a higher spread when the patient is kept in the sitting position for at least 2 min after the injection, as it has been demonstrated for bupivacaine.¹² Thus, both ropivacaine and plain bupivacaine may be unreliable for spinal anesthesia if the hyperbaric solution is not used, as they occasionally produce high spinal block.¹³⁻¹⁵ Therefore, as the optimal dosage of ropivacaine is unknown, a hyperbaric solution for spinal anesthesia, especially for cesarean delivery, is considered superior to an isobaric solution.¹⁶

Associated symptoms and signs are usually benign and resolve spontaneously. They are most commonly related to the Horner's syndrome itself, e.g. nasal stuffiness, blurred vision, strange feeling over the affected eye or hemiface. Patients are more likely to complain of respiratory discomfort because of nasal stuffiness than because of diminished chest wall motion.

Although in this case Horner's syndrome was transient, secondary to spinal cephalad spread of local anesthetic (ropivacaine), some cases may result from intracranial pathology and other causes of Horner's syndrome should be ruled out (e.g. stroke).¹⁷

The purpose of this report is to review the theories, to explain the development of this complication of spinal anesthesia and to heighten awareness of this benign condition among labor suite personnel. Last but not least, the symptomatic patient and anxious family members may need appropriate reassurance when a diagnosis of Horner's syndrome is made in the obstetric setting.

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