Postpartum Unilateral Horner's Syndrome Following Lumbar Epidural Anesthesia after a Cesarean Delivery

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We report on a case demonstrating unilateral Horner's syndrome (HS) after lumbar epidural obstetric anesthesia. A healthy, 32-year-old woman with a breech presentation was scheduled for an elective Cesarean section. The patient had normal vital signs throughout the surgical procedure. The operation lasted for 50 min. In the recovery room, she complained of left nasal stuffiness, left cheek numbness, and heaviness in her left eye. Meanwhile, left nipple sensory loss was noted during baby suckling training. On physical examination, her left eyelid was droopy along with left-side ptosis and facial flushing. Reduced sensation over the left hemifacial region and upper arm was also noted, which resolved completely over the next 110 min. A diagnosis of unilateral HS was then made. Although typically a benign side effect which often spontaneously resolves, HS is likely to cause anxiety in both the patient and the doctor. Prompt recognition of this syndrome and determination of its cause from lumbar epidural anesthesia can prevent unnecessary and potentially dangerous diagnostic workup and can reassure both patients and clinicians. The patient was discharged from the hospital 5 days after onset with a good outcome. *(Chang Gung Med J 2004; 27:624-8)*

Key words: postpartum, Horner's syndrome, ptosis, lumbar epidural anesthesia.

Horner's syndrome (HS) (consisting of ptosis, miosis, anhydrosis, enophthalmus, and conjunctival and facial congestion) is an uncommon consequence of lumbar epidural anesthesia, and was first reported by Kepes.[1] Epidural anesthesia and analgesia in obstetric patients have been reported to produce the majority of cases of HS.[2-9] In our current delivery room practice, lumbar epidural anesthesia is widely used for all cesarean sections (CSs). During the past 25 years, we have noted several cases of mild and transient HS with no morbidity or neurologic deficits.

We report a case of unilateral HS that was caused by lumbar epidural anesthesia, resulting in typical symptoms and signs. This article highlights the importance of early recognition of this transient symptom and discusses the various mechanisms of its process. We also wish to raise awareness of this syndrome as a potential complication of epidural anesthesia.

CASE REPORT

A 32-year-old woman (weighing 78 kg and 167 cm tall) at 38 weeks' gestation was scheduled for a CS. In the preanesthetic evaluation, this was her second pregnancy. Her first, a breech presentation resulted in a CS without morbidity. Her second pregnancy again was found to have a breech presentation, and the antenatal course had been uneventful.

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Informed written consent was obtained after a full explanation of the anesthetic procedures and risks to the patient. The patient was slightly anemic (with hemoglobin of 10.9 g/dl and hematocrit of 32.4%) with normal serum electrolytes, prothrombin time (10.9 s), activated partial thromboplastin time (30.7 s) values, and platelet count (172,000/mm^3). She was sent to the operating room after an intravenous bolus volume preload of 500 ml Ringer's lactate solution. The patient was monitored with routine standard electrocardiography (ECG), pulse oximetry, and an automated noninvasive blood pressure cuff. Six liters of oxygen was applied through a facemask during the operation.

Epidural block was performed at the L3-4 interspaces with the patient in a left lateral decubitus position. An epidural catheter was gently inserted 5.5 cm upwards inside the epidural space. The patient was immediately placed in a supine position with a left lateral tilt. A total volume of 21 ml of local anesthetic/opioid mixture, containing 2% lidocaine with 50 µg fentanyl and 100 µg epinephrine was incrementally injected (5 ml) following a test dose (3 ml), without evidence of subarachnoid block or intravascular injection. Ten minutes following the epidural top-up dose, cutaneous analgesia was obtained to the T3/T4 sensory level by the pinprick test. The patient complained of no discomfort such as nausea, vomiting, or surgical pain during the operation. The vital signs of the mother remained stable throughout the procedure. A female baby (3770 g) was delivered with Apgar scores of 9 at 1 and 5 min.

The surgery lasted 50 min. The patient was sent to the postanesthesia care unit (PACU) where she complained of left nasal obstruction and left upper arm and facial numbness with a sensation of heaviness in her left eye. On physical examination, the patient was awake, alert, and in mild discomfort. The neck was supple with a full range of motion. Left-sided blepharoptosis, miosis, and facial and conjunctival congestion were evident when compared with the right side (Fig. 1). There was no headache, diplopia, nystagmus, blurred vision, or photophobia. She also complained of sensory loss (with the left nipple affected, but with intact feeling in the right one) during baby-suckling stimulus. She had left-side numbness over the shoulder and left hemifacial region within the distribution of the ophthalmic and maxillary divisions of the trigeminal nerve and reduced sensation to touch in these distributions. However, there were no motor deficits, and upon examination, the remaining cranial nerve was unremarkable. The grip strength on the right side was normal with a slight decrease in the left. Her vital signs revealed no hypotension, bradycardia, or desaturation. The patient's condition gradually improved, and the symptoms and signs had completely resolved 110 min after onset (Fig. 2). She was discharged from the hospital 5 days after the CS, and had no neurological deficits.

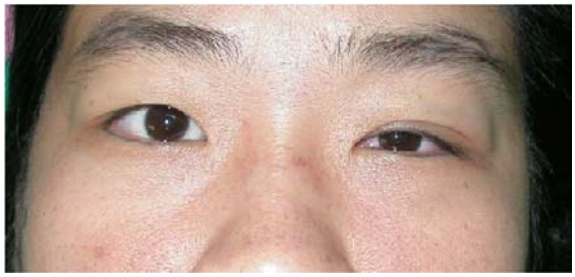

Fig. 1 Left blepharoptosis, miosis, and facial and conjunctival congestion secondary to Horner's syndrome.

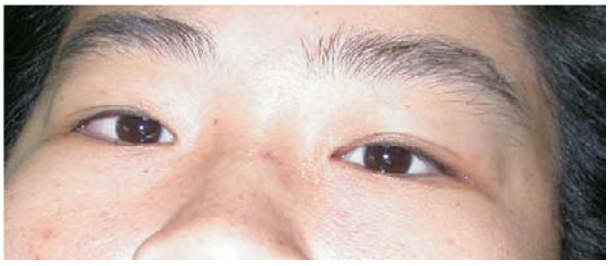

Fig. 2 Patient's symptoms and signs completely resolved 110 min after onset.

DISCUSSION

HS has been recognized as a complication of lumbar epidural anesthesia, the occurrence of which is unpredictable among large series of patients. HS due to the effect of a local anesthetic is a relatively benign and transient condition that usually does not warrant further extensive investigation. Persistent ocular and severe neurologic deficits can cause anxiety for both the patient and the doctor, which may prompt inappropriate emergent and costly diagnostic film-taking and pharmacological workup.[3] However,

it can sometimes precede a high sympathetic blockade and cardiovascular collapse. Consequently, a diagnosis must be made by a proper and thorough evaluation.

Regional anesthesia (intraoral anesthesia, brachial plexus block, stellate ganglion and cervical plexus block, or epidural anesthesia using either a thoracic,[10] lumbar,[3-9] or caudal[11] approach, as well as interpleural analgesia) is the main anesthetic cause of HS. Other causes of HS include face and neck surgery, or trauma, which may also present with this 1-sided effect. Our case, with unilateral HS occurred after a CS, and may have been related to the lumbar epidural anesthesia.

It is likely that the HS occurring after epidural anesthesia is caused by disruption of the occulosympathetic pathway (preganglionic neurons).[2,12] Most anesthesia is performed for obstetric procedures, such as labor pain control or with CSs.[2-9] Various mechanisms have been proposed to explain the pregnancy-associated occurrence of HS after epidural anesthesia. Among them epidural-venous engorgement during pregnancy, resulting in a decreased epidural space and increased epidural pressure caused by each uterine contraction, may thus result in a high, upward, cephalad spread of the anesthetic agent. Other contributory factors include patient positioning and a greater sensitivity of sympathetic preganglionic B fibers to the action of local anesthetic agents.[3] Local anesthetics including bupivacaine, chloroprocaine, lidocaine, or ropivacaine are often used in epidural anesthesia and analgesia. Even a diluted local anesthetic,[9] with a relatively low sensory level (T8) of epidural labor analgesia[5] or a low-dose regimen,[13,14] may cause HS. Recurrent HS has also been reported.[7,8,15] HS begins shortly after the last epidural injection of local anesthetic (mean, 25 min; range, a few minutes to 100 min). The symptoms and signs often spontaneously regress, within a mean of 215 min.[3] The highest level of cutaneous sensory loss achieved after anesthesia is highly variable, ranging from L1 to C4 or C5.[3] Loss of suckling sensation (T4) along with partial numbness over the patient's left upper arm and brachial region (C4-6) was found in our case.

Symptoms of HS may be unilateral or bilateral and may indicate sympathetic denervation to at least the T4 level. HS complicating epidural anesthesia usually occurs unilaterally, which may be due to a subdural injection, asymmetrical positioning of the catheter in the extradural space, or anatomical changes in the extradural space of pregnant women.[2,5] The most logical explanation is the cephalad spread of the anesthetic drug along the epidural space while placed in a supine position. Gravity and the posture affect the spread, and patient positioning may be the reason for unilateral sympathetic blockade. Lateral positioning tends to increase analgesia concentrations on the side that is lower during injection of the anesthetic solution.[16] Our patient developed left-sided, unilateral HS, which could be explained by the patient positioning (dependent side) during the epidural injection with left uterine displacement. In other cases, it was explained by inadvertent partial dural puncture and subarachnoid injection of some of the local anesthetic solution.[5]

Associated symptoms and signs are usually benign and spontaneously reversible. They are most commonly related to HS itself (e.g., nasal stuffiness, blurred vision, a strange feeling over the affected eye or hemifacial region). Other associated neurologic manifestations are less common, such as upper extremity involvement, trigeminal nerve palsy, or hoarseness of the voice.[5] Maternal hypotension, ventilatory insufficiency, and fetal bradycardia may be associated with more-serious complications. Cranial nerve involvement (mainly trigeminal paresthesia) suggests high sensory blockage extending up to the cervical sensory level. Our case showed reduced sensation to touch over the left arm and with a distribution of the ophthalmic and maxillary divisions of the trigeminal nerve, and sensory loss in the left nipple during baby-suckling stimulus in the PACU. This may indicate high sympathetic blockade, and such a patient should be closely monitored for autonomic complications and hemodynamic change. But fetal bradycardia, maternal hypotension, and dyspnea were not noted during the intraoperative or postoperative period. We think that the neurologic complication of this unilateral HS was mild and transient. Reassurance of the patient was important in helping her control her fear.

In summary, we report this clinical case of typical unilateral HS caused by lumbar epidural anesthesia for a CS operation. It may indicate a high sympathetic blockade caused by the known etiology of epidural anesthesia. Neither fetal bradycardia nor maternal hypotension was noted throughout the

entire course. The importance of early recognition of transient HS may prevent unnecessary anxiety for the patient and potential initiation of unnecessary diagnostic workup.

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剖腹產使用腰椎硬脊膜外腔麻醉後併發單側Horner氏症候群

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我們報告一例產科腰椎硬脊膜外腔麻醉後，引發單側Horner氏症候群。一位32歲健康的產婦因臀位而接受常規剖腹產。整個開刀過程病人生命徵狀正常。手術歷時50分鐘。在恢復室，病人主訴左邊鼻塞，左臉頸麻痺及左眼皮沈重，同時發覺母嬰吸吮訓練時，左乳頭吸奶沒有感覺。理學檢查發現左眼瞼下垂及左臉潮紅；左半臉及上手臂感覺遲鈍，這些症狀於110分鐘後完全解除。因此被診斷爲單側Horner氏症候群。典型良性的副作用常可自發性解除，但卻常造成病人及醫護人員的憂慮。若能提早確立此症侯群是多因硬脊膜外腔麻醉引起，就可避免一些不必要、昂貴或可能危險性的檢查及測試，同時醫生與病人即可較爲安心。病人於發生此症狀後，第5天出院，預後良好。(長庚醫誌 2004;27:624-8)

關鍵字：產後，Horner氏症候群，眼瞼下垂，腰椎硬脊膜外腔麻醉。

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