



# Recurrent Horner's Syndrome after Epidural Analgesia for Labor

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## Case Report

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## Abstract

**Background:** We describe a case of recurrent Horner's Syndrome (HS) after epidural analgesia twice in the same delivery.

**Case Report:** A 35-year-old woman requesting epidural analgesia for labor. Epidural technique was carried out without complications but resulted in a Horner's Syndrome with incomplete and asymmetric sensory block. Due to the high suspicion of a subdural block no further local anesthetics (LA) were administered. HS resolved spontaneously. Another attempted epidural catheter placement was performed. Horner's syndrome appeared again. No further epidural attempts were performed.

**Conclusions:** There are no guidelines for epidural catheter management when HS is developed. Some authors consider it safe to continue LA infusion in patients with isolated HS. However, it seems prudent to discontinue LA administration to avoid high spinal block. It is important to make decisions on a case-by-case basis. This case adds the experience of management of recurrent HS in a patient in the same delivery.

**Keywords:** Physiological Changes; Hemodynamic Changes; Epidurography

**Abbreviations:** HS: Horner's Syndrome; LA: Local Anaesthetics; VAS: Visual Analogue Scale.

case, despite removal and placement of another epidural catheter for labor analgesia.

## Introduction

Epidural analgesia is commonly performed for labor pain management. Generally, it is a very reliable technique but complications may result, such as Horner's syndrome (HS), a consequence of pre-ganglionic sympathetic fibres of the stellate ganglion (C8-T2) blocked by local anesthetics (LA). Clinical features include ipsilateral ptosis and miosis, with normal response to light, enophthalmos, vasodilatation and facial anhidrosis [1]. HS is a rare complication, with an incidence from 0,9% in parturients undergoing epidural analgesia, to 4% for those with an epidural anaesthesia for a caesarean [2].

It is essential to exclude LA spread to the subdural space to avoid serious complications. We report a recurrent HS

## Case Report

A 35-year-old pregnant woman with no medical history came to our hospital at week 39 of gestation for spontaneous labor. She requested epidural analgesia and as no contraindications were found, this technique was performed. Tuohy needle was inserted with the patient in a sitting position at the L3-L4 space, using loss of resistance to saline technique. The epidural space was located at the first attempt at 5 cm. A catheter was placed without difficulty and we inserted 4 cm into the epidural space. The aspiration test and the test dose (3ml of bupivacaine 0,25% with epinephrine 5 ug/ml) were both negative. Five minutes after the first LA bolus (lidocaine 1% 5ml and levobupivacaine 0, 125% 8ml) the patient experienced a reduction in pain (Visual Analogue Scale -VAS-1-2).

Twenty minutes after the first bolus, the patient developed a left Horner's syndrome with a right side T2 level superficial sensory block and a left side T7 level without motor blockade. There were no significant hemodynamic changes and the rest of the clinical explorations were normal. Despite the block level, the patient started complaining of abdominal pain on the right side. Since a subdural catheter position was suspected, no more LA was administered. HS was fully resolved within two hours after the first bolus.

The patient accepted a new epidural technique, which proceeded without incident. Space was modified to L2-L3. A catheter was inserted and a low- volume fractionated bolus (lidocaine 1% 4 ml and levobupivacaine 0,125% 4 ml) was administered. Five minutes after the bolus, the patient presented a clear symmetrically and bilaterally reduction in pain.

The patient suffered a systemic hypotension episode (non-invasive blood pressure 90/50 mmHg) which was successfully managed with intravenous fluids (Plasmalyte® 250ml) and vasopressors (ephedrine 6 mg).

Fifteen minutes after the bolus administration, the patient suddenly developed HS again with a superficial bilateral sensory block (T2 level) without motor blockade. Neurological and hemodynamical exploration was normal. Due to the potential risk of a high spinal block resulting from excessive cephalad spread, we decided to discontinue LA administration after informing the patient. No further epidural attempts were performed.

The delivery was uneventful and the HS disappeared ninety minutes after the onset of symptoms. She was discharged after 48h with no complications during admission.

## Discussion

Horner's Syndrome after epidural analgesia is due to pre-ganglionic sympathetic fibres of the stellate -cervico-thoracic- ganglion (lower cervical ganglion +/- first thoracic sympathetic ganglion) blockade by local anesthetics. The stellate ganglion is located behind the transverse process of C7 [3,4].

Two main mechanisms have been proposed that may explain this syndrome. The first hypothesis is based on the existence of abnormalities in the epidural space, such as those due to anatomical and physiological changes inherent in pregnancy (epidural venous plexus ingurgitation secondary to inferior vena cava compression, increased pressure resulting from uterine contractions, etc). Another possibility is the existence of fibrosis or epidural septae, which make a correct distribution of LA difficult. In all these

cases, the epidural space is narrowed, resulting in increased cephalic diffusion of the local anesthetic [2,5,6].

Second hypothesis is explained by LA spreading into the paravertebral space, which would result in a unilateral cephalic distribution, or by spreading into the subdural space.

There are various mechanisms by which an accidental subdural block can occur during an epidural technique: migration of the catheter to the subdural space, puncture of the dura mater with a needle (without arachnoid injury) and use of multi-hole catheters. In the latter case, the distal orifice may remain in the subdural space and the proximal orifice in the epidural space. This would result in slow AL infusions being distributed correctly through the epidural space (by diffusion through proximal orifice), whereas the use of rapid infusions or boluses, which diffuse through the distal orifice, would result in subdural LA spread. Several predisposing factors to the development of subdural block have been described: previous back surgery, recent lumbar puncture, difficulty in performing neuraxial technique and repeated attempts at the same space [7,8].

The anatomy of the subdural space was studied by Reina MA, et al. [9], and they attribute its origin to iatrogenic causes: mechanical forces and administration of air or fluid to the interface between the dura mater and the outer compact laminar portion of the arachnoid (dura-arachnoid interface). This would break up the arachnoid, creating the subdural space. It has a higher dorsal than ventral capacity; this facilitates LA to be deposited mainly over dorsal roots. For this reason, the most typical clinical presentation is characterized by an abnormally high (sometimes inadequate) sensory block of slow onset (between 15 and 20 minutes), with a duration of up to two hours followed by complete recovery [2].

Motor block may be insufficient or absent. Moreover, excessive cephalic spread of LA into the subdural space can even reach intracranial level, affecting the brainstem and thus causing episodes of unconsciousness and apnea. In these cases, another syndrome described is Trigeminal nerve palsy [7].

Anesthetic management of HS during epidural analgesia remains uncertain. Some authors consider it a benign and self-limited side effect, which permits the continued use of the catheter. Other authors recommend a detailed clinical evaluation to diagnose life-threatening side effects as a consequence of high cephalad spread. They even recommend that special consideration should be given to those cases that have previously presented HS related to this technique [5].

Most cases are unilateral and resolve spontaneously after LA discontinuation, although in those cases described in the literature in which the infusion is continued, symptoms also disappear, with no clinical recurrence after further boluses [2].

In those situations, where HS occurs as a consequence of an inadvertent subdural block, the administration of large doses of LA could result in the development of other complications such as arachnoid rupture, leading to the risk of post-dural puncture headache and leakage of LA into the intradural space, producing a subarachnoid block [7].

In this clinical case, after the first technique and evidence of left HS, the patient presented a high asymmetric (right T2 and left T7 level) and inadequate (pain in the right hemiabdomen) sensory block despite the cephalic spread of LA. Due to the fact that a subdural block was suspected, we decided to stop LA administration and wait for the resolution of symptoms.

Subsequently, we performed a new technique, with a similar result: left HS after fifteen minutes of LA administration. In this case, an effective and uniform sensory block was reached, but with a disproportionately high-level block (bilateral T2) in spite of the low- volume of LA administered. Given this situation, we assessed both the possibility of a new subdural block and the possibility of excessive cephalic diffusion in the epidural space (probably due to anatomical alteration caused at the first technique). Considering the uncertainty and unpredictability of the block, we prioritised the maternal-fetal safety. For this reason, we explained the risks to the patient and decided to discontinue LA administration.

Some authors point out that, after previous dilatation of the subdural space, the anatomy may be distorted, which would facilitate insertion of the catheter into the subdural space in further attempts [10]. The patient, therefore, should be closely monitored and avoid LA administration if a subdural block is suspected.

There are no clear guidelines for epidural catheter management in patients who present HS although it seems prudent to discontinue LA infusion, since cases of late clinical deterioration associated with inadvertent subdural block have been reported. Other authors consider it safe to continue in patients with isolated HS [11], as in most cases they resolve spontaneously [2]. Another option appears to be radiological confirmation of the location of the catheter by epidurography. LA administration could, therefore, be continued, but reducing the infusion rate so as not to produce a high spinal block [6,7,10].

There seems to be a consensus that if HS persists beyond 24 hours or with an atypical presentation (associated with headache or neck pain, for example), a neurologist should be consulted [2].

In summary, recurrent HS after epidural analgesia is uncommon. In most cases, when associated with epidural analgesia it is uncomplicated and resolves itself after LA discontinuation. A detailed clinical examination is essential to rule out possible adverse effects.

There is no consensus on the management of these patients. The decision to continue using the catheter must be made case by case, based on maternal-fetal status and clinical findings, being aware about possibility of subdural block. For this reason, it is a priority to develop diagnostic criteria for early recognition of LA subdural spread and to avoid unnoticed use of subdural catheters.

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- **Running Head:** Recurrent Horner's syndrome and epidural analgesia.

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