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CLINICAL INFORMATION

# Horner's syndrome and paresthesia in the trigeminal nerve territory secondary to epidural analgesia for labor

**Céline Ferreira***, **Ana Luísa Macedo**, **Valentina Almeida**

*Centro Hospitalar e Universitário de Coimbra, Serviço de Anestesiologia, Coimbra, Portugal*

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**KEYWORDS**
Obstetric analgesia;
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**Abstract** Currently, epidural analgesia is a common procedure for labor analgesia. Although it is considered a safe technique, it is not without complications. Horner's syndrome and paresthesia within the trigeminal nerve distribution are rare complications of epidural analgesia. We report a case of a pregnant woman who developed Horner's syndrome and paresthesia within the distribution of the trigeminal nerve following epidural analgesia for the relief of labor pain.


**PALAVRAS-CHAVE**
Analgesia obstétrica;
Anestesia peridural;
Síndrome de Horner;
Parestesia do território do nervo trigêmeo

**Síndrome de Horner e parestesia do território do nervo trigêmeo secundário a analgesia peridural para trabalho de parto**

**Resumo** A analgesia peridural é hoje em dia um procedimento comum para analgesia do trabalho de parto. Embora seja considerada uma técnica segura, não está isenta de complicações. A síndrome de Horner e a parestesia do território do nervo trigêmeo são complicações raras da analgesia peridural. Relatamos um caso de uma grávida que desenvolveu a síndrome de Horner e parestesia do território do nervo trigêmeo após analgesia peridural para o alívio da dor do trabalho de parto.


* Corresponding author.
*E-mail:* celine.ferreira@gmail.com (C. Ferreira).

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## Introduction

Epidural analgesia is currently a common procedure for pain relief during labor. Although considered safe, this analgesic technique is not free of complications.[1]

Horner syndrome is a rare and benign complication of epidural analgesia for labor[1,2] that was first reported by Kepes in 1972.[3] It may occur in any patient undergoing anesthesia or epidural analgesia. The incidence is higher in the obstetric population and ranges from 0.4% to 4%, being more prevalent in pregnant women undergoing cesarean sections.[1,2,4] It is believed that this incidence is undervalued, as in some cases the clinical manifestations are clearly evident.[1–3]

Horner syndrome clinical signs includes ptosis, miosis, and anhidrosis, as well as enophthalmia and hemifacial flushing.[1,3–5] It is a rapidly evolving condition that disappears in a few hours, mostly without sequelae.[2] Paresthesia in the trigeminal nerve territory as a concomitant symptom is extremely rare and is associated with a high sensory blockade.[3]

We report a case of a pregnant woman who developed Horner syndrome associated with ipsilateral paresthesia in the trigeminal nerve territory after lumbar epidural analgesia to relieve labor pain. We also discuss the possible reported causes that may explain the onset of Horner syndrome.

The patient gave written informed consent for publication of the case details.

## Case report

Pregnant woman, aged 25 years, 74 kg, 163 cm, BMI 28.6 $kg.m^{-2}$, GIIP0, no personal medical history, was admitted to the delivery room at the onset of labor, at 39 weeks of gestation.

The risks and benefits of the neuraxial blockade technique with epidural catheter placement were explained to the patient. Written informed consent was obtained.

After hydration with 500 mL of Ringer's lactate, the patient was placed in the left lateral decubitus position and a single puncture was performed to investigate the epidural space at the L3–L4 level, using a Tuohy needle and loss of resistance to air.

The catheter was introduced 4.5 cm into the epidural space in the cephalad direction. Aspiration was negative for blood or cerebrospinal fluid; thus, a test dose of 2 mL lidocaine was administered.

Five minutes after dose-test administration via the epidural catheter, the patient showed no sensory or motor change. Therefore, a manual bolus of 0.2% ropivacaine (8 mL) was administered, and a Patient Controlled Epidural Analgesia with Programmed Intermittent Peridural Bolus (PCEA-PIEB) from our institution was initiated (solution of: 20 mL 0.75% ropivacaine, 20 μg sufentanil, and NaCl 0.9% to make up 100 mL of the solution; compulsory bolus: 8 mL every 60 min; rescue bolus: 5 mL; lockout time: 15 min; safety limit: 3 boluses/h; without perfusion).

The baseline visual analog scale score was 7/10 and 30 minutes (min) after epidural analgesia it decreased to 3/10 with sensory block up to T7.

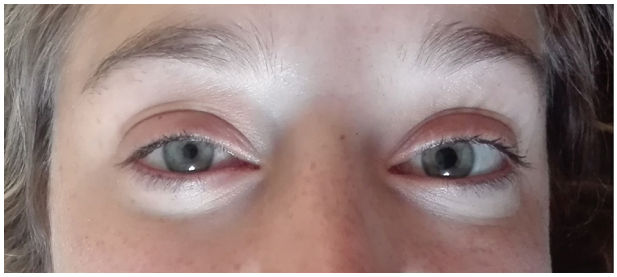

**Figure 1** Ptosis and miosis of the right eye.

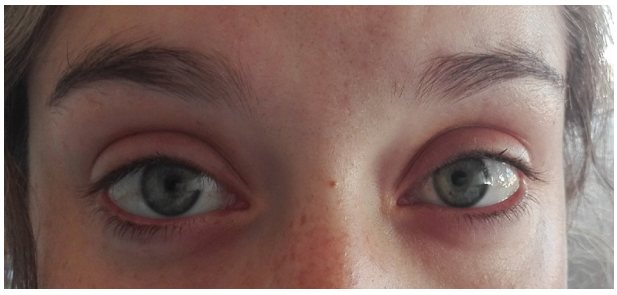

**Figure 2** Horner syndrome totally reversed after three hours of diagnosis.

The patient was instructed to change the position between left and right lateral decubitus every 30 min in order to obtain a better anesthetic distribution in the epidural space.

After two hours of epidural analgesia, she reported right hemifacial paresthesia. During this interval, two programmed boluses of PCEA-PIEB were administered. Objective examination revealed a rise in sensory level up to T4 and right Horner syndrome characterized by right eye ptosis and miosis (Fig. 1), in addition to right hemifacial paresthesia along the trigeminal nerve territory (its maxillary and mandibular branches). PCEA-PIEB was immediately stopped, and the patient was placed in the left lateral decubitus position.

One hour after the diagnosis, the patient had a VAS score of 5/10 and partial reversion of symptoms (partial reversion of miosis and ptosis, absence of paresthesia, and sensory block at T10), a manual bolus of 2% ropivacaine (5 mL) was given. Childbirth was normal with APGAR scores at the 1st, 5th, and 10th minutes of 9, 9, and 10, respectively. There were no intercurrences.

During labor, the pregnant woman had no change in consciousness or difficulty breathing and was always hemodynamically stable.

The epidural catheter was removed at the end of labor. There was no worsening of the Horner syndrome symptoms, with complete reversion after three hours of diagnosis (Fig. 2).

## Discussion

There are numerous mechanisms that explain the onset of Horner syndrome during epidural anesthesia. One of the most plausible mechanisms is the increase of local anesthetic cephalad dispersion into the epidural space, causing the blockade of the sympathetic ganglionic nerve

fibers (C8-T1 and occasionally up to T4).[1,2] Horner syndrome is characterized mainly by miosis, palpebral ptosis, and anhidrosis. Other neurological manifestations of the local anesthetic inhibitory effect on the high sympathetic chain are hoarseness, respiratory distress, and hemodynamic instability, such as hypotension and bradycardia, which are due to decreased venous return, cardiac output, and arterial pressure. Paresthesia in the trigeminal nerve territory may also be a concomitant symptom, although rarer than hemodynamic instability.[3] Placement of catheter in the cephalad position also favors that dispersion of local anesthetic[2,3] and its excessive introduction into the epidural space may justify the ipsilateral manifestation.[2] By increasing the force of gravity, lateral decubitus position may favor head dispersion, which justifies the syndrome unilateral manifestation.[2]

Another mechanism explaining this syndrome onset is related to catheter migration out of the epidural space. The accidental migration of a multiperforated catheter tip into the subdural space causes spreading of the local anesthetic through the arachnoid membrane up to the cerebrospinal fluid in areas more cephalic than anticipated.[2,3] Accidental migration of catheter into paravertebral space allows, if there is communication between the different vertebral levels in this compartment, that a single dose of local anesthetic administered in the lumbar region spreads laterally in the cephalic direction and can reach the first thoracic levels.[2]

The incidence of Horner syndrome in the obstetric population is higher because there are others that may explain its onset in addition to the mechanisms described above. In pregnant women, the epidural space is decreased due to engorgement of the epidural veins and uterine contractions.[1–4] Sensitivity to local anesthetics[5] is higher due to the action of progesterone on central and peripheral nervous system and the anatomical variability of nerves, that is, smaller nerves are more easily blocked by local anesthetics.

Ultimately, the type of local anesthetic does not seem to influence the incidence of Horner syndrome. We found no literature comparing this syndrome rate of incidence with different types and different concentrations of commercially available local anesthetics.

In our case, the unilateral manifestation can be explained by the lateral decubitus position and excessive introduction of the epidural catheter. Anatomical and physiological changes in pregnancy, patient positioning, and local anesthetic volume and rate of administration can easily explain the cephalad spread of the local anesthetic, which causes a high sympathetic block even when the observed sensory level of our patient was limited up to the T4 level. The absence of hemodynamic instability in our patient throughout labor can be explained by the increased sensitivity to local anesthetics due to the action of progesterone on central and peripheral nervous system. Subdural administration of the anesthetic could also explain the absence of hemodynamic instability, but the absence of a sensory-motor block after test dose administration and the bilateral, symmetric, and rapid onset of sensory block installation, without sparing the segmental distribution of sacral roots after ropivacaine administration confirm the catheter location within the epidural and not subdural space.

In conclusion, Horner syndrome is a rare complication of epidural analgesia,[1] which may be accompanied by respiratory distress, hemodynamic instability and, more rarely, paresthesia in the trigeminal nerve territory. Horner syndrome or even paresthesia in the trigeminal nerve territory may be signs of hemodynamic instability. Therefore, it is important to maintain a more rigorous fetal and maternal surveillance after the diagnoses.

## Conflicts of interest

The authors declare no conflicts of interest.

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