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Case report

Horner syndrome after epidural analgesia for labor. Report on three cases☆

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ABSTRACT

Epidural analgesia is assumed to be the technique of choice for the relief of pain in labor. Multiple adverse neurological effects have been reported, one of which is the so-called Horner syndrome (ptosis, myosis, anhidrosis). Its evolution is usually benign and does not require specific management, except clinical monitoring for the more than probable cephalic spread of local anesthetic. Most of the cases that exist in the literature are isolated; in our work we present a series of 3 clinical cases and review the pathogenesis and management in the obstetric patient.

Sindrome de Horner tras analgesia epidural para el parto. Informe de 3 casos

Palabras clave:
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RESUMEN

La analgesia epidural supone la técnica de elección para el alivio del dolor del parto. Se han descrito múltiples efectos adversos a nivel neurológico, uno de ellos es el llamado Síndrome de Horner (ptosis,miosis, anhidrosis), suele presentar evolución benigna y no requiere manejo especifico, salvo vigilancia clínica por la más que probable difusión cefálica del anestésico local. La mayor parte de los casos existentes en la literatura son aislados, en nuestro trabajo presentamos una serie de 3 casos clínico y repasamos su etiopatogenía y manejo en la paciente obstétrica.

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Introduction

Horner syndrome was first described in 1879 by Swiss ophthalmologist Johann Friedrich Horner. It is characterized by the presence of myosis, ptosis, and anhidrosis, with or without enophthalmos.[1] Its primary cause is the ipsilateral interruption of the sympathetic nerve fibers that innervate the pupil, the upper eyelid lifter muscle, and the facial region.[2]

Any obstacle that affects this neuronal region, from the origin to the last synapse, can lead to this clinical picture. Acquired causes are the most frequent, as are iatrogenic causes due to neuraxial anesthesia, and, in certain populations (such as the obstetric population), the incidence increases considerably due to anatomical and physiological changes that occur. Epidural analgesia is considered the analgesic technique of choice for labor.[3] Horner syndrome associated with epidural analgesia for labor was described by Kepes in 1972. Its incidence is estimated at between 0.4 and 4%.[4–7]

In our study, we present a series of three clinical cases of Horner syndrome in pregnant patients that received epidural analgesia for labor. We also review the physiopathology, implications, and management of the labor.

Clinical case

The technique used in the three cases is described as follows: we use an 18 gauge Touhy needle. The space chosen was L3–L4 with an intervertebral approach. Once the epidural space was located through the loss of resistance to saline technique, a multi-perforated epidural catheter was place 4 cm within the space. The technique was applied in all cases without incident. After the administration of one bolus of 0.16% ropivacaine with 1 μm/ml of fentanyl, the perfusion of anesthetic at the same concentration was initiated. As a test dose, we used 4 ml of bupivacaine at 0.25% with 1/200,000 epinephrine.

Case 1

27-Year-old patient in her first gestation in spontaneous labor at 38 weeks of gestation, 172 cm tall and 75 kg body weight, without medical antecedents of interest. A significant characteristic to highlight was a noticeable lumbar hyperlordosis. The epidural catheter was placed at 4 cm dilation, after which 11 ml of ropivacaine with fentanyl at the concentration described above was administered. Continuous perfusion of 10 ml h^{-1} of the same anesthetic solution was initiated. She did not receive any supplementary boluses. After the dilation phase and 95 min after the initiation of the perfusion, the patient complained of symptomatology compatible with brachial palsy. After neurological exploration, a motor deficit was observed (level 3 on the Medical Research Council scale) that included the entire upper limb, as well as unspecific soreness at the ipsilateral ocular level with evidence of ptosis, myosis, and anhidrosis compatible with Horner syndrome. The level of sensory block reached T2. After the perfusion was detained, motor and ocular clinical presentation reversed after 115 min.

Case 2

28-Year-old patient in her first gestation in spontaneous labor at 37 weeks of gestation. 160 cm tall and 55 kg body weight without personal antecedents of interest. 8 ml of ropivacaine and fentanyl were administered in the initial bolus followed by continuous perfusion at 8 ml h^{-1}. The patient received two supplementary boluses, first 30 min after the start of perfusion, and the second 45 min after the previous bolus. During the dilation phase and 80 min after the last bolus, the patient described ptosis, myosis, and enophthalmos, without manifested anhidrosis. Motor deficit was not present; the sensory deficit rose to T3. The clinical presentation disappeared 130 min after detaining the perfusion.

Case 3

32-Year-old patient in her first gestation in induced labor at 41 weeks of gestation. 155 cm tall and 60 kg body weight. She had chronic arterial hypertension as an antecedent of interest. An initial bolus of 9 ml of ropivacain and fentanyl was administered, followed by a continuous perfusion of 8 ml h^{-1}. After 45 min, and due to risk of loss of fetal wellness, the decision was made to initiate an emergency cesarean section. For this, 9 ml of 2% lidocaine was administered. After the beginning of the cesarean section, a clinical presentation suggesting Horner syndrome was observed (see Fig. 1) only 15 min after the administration of the anesthetic bolus of lidocaine. No motor symptoms were reported. The sensory level reached metamere T2. After 95 min of observation, the clinical presentation disappeared without further measures.

After the clinical diagnosis from evidence of ptosis, myosis, enophthalmos, and anhidrosis, an neurological (sensory level and motor function) and cardiorespiratory (continuous monitoring of blood oxygen saturation with pulse oximetry, electrocardiography, and non-invasive blood pressure) exploration was initiated.

The perfusion of local anesthetic was detained in all the cases. None of the patients presented with cardiorespiratory complication, maintaining a heart rate of around 70 beats per minute, average blood pressure above 65 mm/hg, and blood oxygen saturation above 95% with no need for supplementary oxygen. The clinical presentation reversed in a variable time after perfusion was stopped.

There were no neonatal repercussions in any of the cases presented.

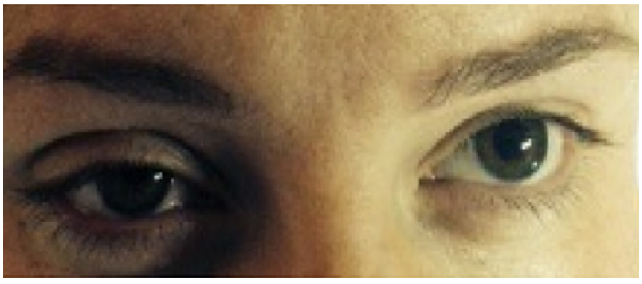

Fig. 1 – Horner syndrome in one of our patients. *Source*: Authors.