

HORNER'S SYNDROME AFTER LUMBAR EPIDURAL ANALGESIA DURING LABOR

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Abstract

Horner's syndrome is rarely reported after epidural analgesia during labor. The use of Top-Up local anesthetic for controlling labor pain in the first stage of labor, or to dense the block in caesarean deliveries can result in this complication.

We reported a cases of Horner's syndrome during epidural analgesia in labor in spite of not giving any Top-Up dose. The case was clinically evident and was successfully managed by stopping the epidural infusion, and reassuring the parturient as well as the family; until the disappearance of the signs and symptoms. The infusion was then restarted, delivery was uneventful and no consequent neurological or psychological problems were noticed after a one-month follow-up.

Introduction

Horner's syndrome is a triad of miosis, ptosis, and enophthalmos, commonly associated with vasodilatation (facial flushing, anhidrosis and nasal stuffiness). Lumbar epidural analgesia is widely used to control pain during labor and is still the superior technique and preferred by parturient and anesthetist¹. It was found previously, that the incidence of Horner's syndrome as a complication following epidural anesthesia is rare, but occurs mainly in obstetric patients and especially in caesarean deliveries after a (top-up) injection of local anesthesia through an epidural catheter. With the use of small doses of continuous infusion in the epidural mixtures; the incidence is decreasing. We reported a case of Horner's syndrome after starting lumbar epidural analgesia for parturient during the second stage of labor.

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Case Report

A 21-year-old healthy woman, primigravida, ASA I, weighing 65 Kg, and 168 cm tall, was admitted to the labor ward in active labor at 38⁺² weeks gestation. The parturient requested epidural analgesia for labor. The epidural catheter was inserted with ease via a 16-gauge Tohy needle through the L3-L4 interspace. The epidural space was 4 cm from the skin and catheter was advanced 4 cm in the epidural space. Following a negative aspiration test, 10 ml of epidural infusion mixture (bupivacaine 0.1% and fentanyl 2 µg ml⁻¹) was injected slowly over 5 minutes. An epidural infusion mixture of bupivacaine 0.1% and fentanyl 2 µg ml⁻¹ was started at a rate of 15 ml h⁻¹. The upper sensory level to cold thirty minutes later was T9 and T10 on the right and left sides respectively. The parturient and fetal vital signs were stable all through the time and the systolic blood pressure was always above 115 mmHg. The parturient was very satisfied with the epidural analgesia.

Six hours after starting the epidural infusion the parturient started to complain of redness on the right side of her face and heaviness on the right eye. On examination, the blood pressure was 95/70 mmHg, the heart rate (HR) was 95 per minute, the respiratory rate was 15 breaths per minute and the fetal heart rate was normal. There was obviously right side Horner's syndrome. The facial skin was flushed, the conjunctiva was red, and miosis with ptosis of the eyelid were present. There was no shortness of breath, hoarseness, weakness or numbness in her arms. The upper sensory level to cold was T3 on the right side and T4 on the left side.

The epidural infusion was stopped, the parturient was reassured and ephedrine 6 mg was given to raise the blood pressure. Sixty minutes later, the Horner's syndrome signs and symptoms were completely resolved and the decision to restart the epidural infusion rate at 8-ml h⁻¹ was made by two consultant anesthesiologists. The infusion continued for another 6 hours without recurrence of any signs or symptoms of Horner's syndrome. The woman was satisfied with pain control through the delivery of her baby. A healthy male baby, 3,250 gm was delivered vaginally, with 1- and 5- min Apgar scores of 9 and 10 respectively. After one month of her discharge the patient was seen in

the Out Patient Clinic. Her physical examination was completely normal, with an excellent psychological status.

Discussion

Horner's syndrome occurs when the sympathetic nerve supply to the pupil, levator palpebrae, conjunctiva and face is interrupted. These sympathetic preganglionic fibers originate from the anterior horn cells of C8 and T1, and occasionally as low as T4. The higher sensitivity of the sympathetic fibers to local anesthesia is due to their smaller diameter.

Most of the reported cases of Horner's syndrome in pregnant women were after lumbar epidural analgesia for labor pain or caesarean section. This was explained by the anatomical and physiological changes during pregnancy and labor which will favor cephalic spread of local anesthetics that are injected in the epidural space. The gravid uterus increases the intra abdominal pressure and results in partial occlusion of the inferior vena cava, this diverts blood through the epidural venous plexus, and will lead to a decrease in the epidural space volume². In the second stage of labor when the women are always asked to push down (Valsalva maneuvers); further reduction in the epidural space will occur. Also during uterine contraction the epidural pressure is transiently increased and can be significantly elevated if injection occurs during uterine contraction².

Several hypotheses explained the high spread of the drugs when given in the epidural space. One of this explanation could be the anatomy of the subdural space, which is described as a "potential cavity" between the dura and arachnoid matter, contains a small volume of serous fluid. The space runs up from the lower border of the second sacral vertebra into the cranial cavity, as high as the floor of the third ventricle. Subdural injections of local anesthetics can cause an unusually high cephalad spread. This finding has been reported in a case in which a high block followed injection of only 2 mL of local anesthetic through a catheter that was probably placed subdurally³.

Reina et al.⁴ threw new light on the ultrastructure of the subdural space. Their work described the arachnoid matter as having a compact laminar portion

covering the inside of the dural sac and a trabeculated portion that spreads like a spider's web to the pia matter coating the spinal cord and the nerve roots. Between the laminar arachnoid portion and the inner surface of the dura they found a cellular junction, where the dura-arachnoid interfaces. This region is composed of neurothelial cells surrounded by an amorphous substance. There was no subdural space in untraumatized tissues, but they hypothesize that a subdural space can appear if the neurothelial cells break up as a result of pressure exerted by mechanical forces, air, or fluid injection, creating fissures within the amorphous substance of the interface. Fissures could readily expand toward weaker areas, particularly laterally where the amorphous substance is more prolific. This finding explains the variability of onset and extension of neural block after subdural injection.

Recently De la Gala and colleagues⁵, investigated the high epidural block in a laboring patient and he found that a usual cause of the high spread is the transverse septum in the epidural space, which is

present in about 2% of their patients.

Horner's syndrome was most commonly unilateral and lateralized on the dependent side. Bilateral Horner's syndrome was seen in 11.5% (6 of 56) of reported cases¹⁶. Nevertheless, neuraxial blockade is generally temporary and benign. However, cases with serious complications such as fetal bradycardia associated with maternal hypotension have been reported.

A review of reported cases described the mean time of onset of Horner's syndrome as 25 minutes after epidural injection (range a few minutes to 100 minutes), and the mean time of resolution as 215 minutes (range a few minutes to 24 hours), depending on the agent used. Both of our cases had a longer onset and shorter resolution times.

Fortunately, our reported case had no sequels after one month of follow up. Reassurance of the patient and monitoring the mother and the fetal vital signs are very important if this problem occurs.

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