Unilateral Horner Syndrome Following Epidural Anaesthesia in a Morbidly Obese Parturient

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Horner's syndrome is rarely observed in epidural anaesthesia; it is characterized by ptosis and enophthalmos on the affected side; miosis, anisocoria, and conjunctival hyperemia in the affected eye and anhydrosis, flushing on the affected side of the face. It is usually a complication spontaneously resolved without permanent neurological deficits. Intraoral anaesthesia, stellate, cervical and brachial plexus block, thoracic, lumbar and caudal epidural anaesthesia and intrapleural analgesia are the main causes associated with Horner's syndrome in anaesthesia. Among the other causes of Horner's syndrome are head and neck surgery, trauma and puncture of the internal jugular vein. We aimed to present a case with unilateral Horner's syndrome, which appeared in the morbidly obese parturient after lumbar epidural anaesthesia.

Keywords: Epidural anaesthesia, pregnant, Horner syndrome

Introduction

Horner's syndrome is a condition that arises because of sympathetic nervous system dysfunction. It is characterized with ptosis, miosis, enophthalmos, conjunctival hyperaemia, flushing on the affected side of the face and anhydrosis. Benign or malignant tumours of the nervous system or of the adjacent structures, trauma or cardiovascular diseases can cause this syndrome, or it can occur as a hereditary or iatrogenic disease (1).

In this case report, we aimed to present Horner's syndrome that developed as a complication of epidural anaesthesia in a pregnant woman with morbid obesity.

Case Presentation

A 31-year-old and 170-cm-tall multigravida parturient weighting 120 kg (body mass index=41.5), who was in her 39th gestational week, was taken into operation for elective caesarean with ASA II risk. In her medical history, she had no feature except gestational diabetes mellitus and postdural puncture headache after prior spinal anaesthesia for caesarean section. Her laboratory values were within normal limits. Before the operation, 500 mL saline solution infusion was administered to the patient. In the operation room, following ECG, pulse oximeter and noninvasive arterial pressure monitorization, the patient was administered cutaneous and subcutaneous local anaesthesia in the sitting position under sterile conditions. Then, epidural space was entered from lumbar 4–5 intervertebral space at a depth of 7 cm using the loss-of-resistance technique with an 18G Tuohy needle and the median approach. The catheter was advanced in the cranially and fixed at a depth of 13 cm in the skin. The length of the catheter in the epidural space was 6 cm. After detection of the absence of intravascular and intrathecal localization with 3 mL 2% lidocaine, 1:200000 epinephrine through epidural catheter, 15 mL fluid (50 µg fentanyl, 12 mL 0.5% isobaric bupivacaine and 2 mL normal saline) was injected. The operation was initiated for the patient whose sensory block level was found to be T6 with the pinprick test performed approximately 20 min later, and the Bromage score was II. After the birth of the baby, it was detected that the patient had first hypokinesia and then motor loss in her left arm, and the sensory block level was T4. The Apgar scores of the new-born at the 1st and 5th minute were 8 and 10, respectively. After the operation was completed in 35 min, the sensory block of the patient, who was followed up in the recovery room, was measured to be T4. Horner's syndrome was detected in the patient who developed ptosis, miosis and conjunctival hyperaemia (Figure 1). No other neurological finding was detected. After the patient was monitored in the recovery room for

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approximately 2 h, her hypokinesia and motor loss in her left arm regressed and then she was transferred to the clinic. At the postoperative 6th hour, the findings of Horner's syndrome regressed. She was discharged with full recovery from the hospital on the postoperative 2nd day.

Discussion

This syndrome characterized by miosis, ptosis, anhidrosis and enophthalmos that developed because of from the blockage of sympathetic fibres coming from the cervical region to the facial region; this syndrome was first identified by Horner in the 19th century (2). The symptoms are from the blockage of sympathetic fibres stemming from C8-T1 level. Sensation disorder is not generally observed because sympathetic fibres are more sensitive to the local anaesthetic. Intraoral anaesthesia, stellate, cervical and brachial plexus blockade, thoracic, lumbar and caudal epidural anaesthetic methods and intrapleural analgesia are among the most common reasons of Horner syndrome (1). Head and neck surgery, traumas and internal jugular vein puncture are among the other reasons (3). Although there is no exact information regarding the real incidence and prevalence of Horner's syndrome after epidural anaesthesia, it is more often confronted because of interventions undertaken for obstetric reasons (4, 5). In our case, Horner's syndrome was detected after lumbar epidural anaesthesia; this supports our study.

The higher incidence of Horner's syndrome in pregnant women than the normal population is because of anatomical and physiological changes. However, the exact incidence is unknown. In a study conducted with 200 cases, the incidence of Horner's syndrome after epidural anaesthesia performed for painless childbirth for obstetric patients was reported to be 1.33% in normal delivery and 2.5% after caesarean section (6). In contrast, the incidence was found to be 0.13% in the study conducted by Rabinovich et al (7). Facilitation of the anaesthetic and analgesic agent's spreading to the head because of anatomic variation of epidural cavity, improper subdural injection of these agents and increased sensitivity of the nervous system to the injected agents can be counted among the physiopathological reasons (8-10). Pressure of the gravid uterus to the inferior vena cava increases intra-abdominal pressure and thus, enlargement of epidural veins decrease epidural cavity volume. Moreover, obesity on its own is among the reasons to narrow epidural space. Contractions of the uterus contribute to the anaesthetic agents' going further from the entrance region by forming rhythmic waves (11). Therefore, spreading of anaesthetic agents in small quantities to the head is observed (12). In our case, morbid obesity was among the risk factors. Esmer et al. (13) reported that Horner syndrome developed because of epidural blockage with 17 mL 0.5% bupivacaine and 100 µg fentanyl. In the literature, Horner's syndrome, which developed because of epidural blockage with 12 mL 0.5% isobaric bupivacaine and 50 µg

Figure 1. The view of a one-sided ptosis

fentanyl, was reported in a pregnant case having a body mass index of 26.6 (14). Chandrasekhar et al. (15) reported the development of Horner's syndrome after one hour in a pregnant woman who had a body mass index of 33.7 and who underwent caesarean section by applying 15 mL bolus and 15 mL hour⁻¹ infusion from the solution containing a very low concentration of 0.04% bupivacaine and 1.66 mcg mL⁻¹ fentanyl. In our case, Horner's syndrome was observed 20 min after the injection of the solution that contained 12 mL 0.5% bupivacaine, 50 µg fentanyl and 2 mL saline.

In Horner's syndrome, symptoms are generally benign, and they spontaneously regress over time. Signs appear approximately 25 min after the epidural administration (2–100 min), and they disappear in 215 min on average (3 min–24 h) (1). In patients with Horner's syndrome whose symptoms do not regress, further examination should be performed and serious causes, such as pancoast tumour or thoracic aort aneurysm, should be ruled out. In our patient, symptoms occurred approximately 20 min after the administration of the drug into the epidural space. Hypokinesia and motor loss in the arm were resolved 2 h later and the findings of ptosis, miosis and conjunctival hyperaemia completely disappeared 6 h later. Respiratory distress that can develop because of an elevated sympathetic block in Horner's syndrome should be kept in mind. If respiratory problems are suspected, additional doses should be avoided and other analgesia techniques should be considered. Because the findings that can be observed in Horner's syndrome can be precursors of high sympathetic blockade or cardiopulmonary arrest, postoperative close follow-up is necessary from detection until the elimination of block (16). In our case, respiratory distress, hypotension or bradycardia was not observed.

Conclusion

Early diagnosis of Horner syndrome, which is one of the rare complications of epidural anaesthesia in a morbidly obese parturient, prevents the anxiety of the parturient and anaesthetist, who implements the anaesthesia procedure, and unnecessary diagnostic procedures, and it is also important for the prevention of the possible development of cardiopulmonary arrest.

Informed Consent: Written informed consent was obtained from patient who participated in this case.

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