Clinical Reports

Autonomic hyperreflexia during labour

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We present two cases of automatic hyperreflexia (AH) during labour in women with spinal cord damage, in whom AH developed before and after delivery. The AH was successfully controlled using epidural anaesthesia in Case #1, but failed in Case #2. The blood pressure was controlled with nicardipine. However, overdose of nicardipine produces vasodilatation and its side effects include headache, flushing and palpitation similar to AH.[1] Considering these effects, we recommend epidural anaesthesia to control AH, because epidural anaesthesia does not only reduce BP, but also blocks the noxious stimuli and relieves the symptoms of AH. Our experience suggests that the epidural catheter can be placed two to three weeks before the date of predicted childbirth, because the onset of labour in a patient with spinal cord damage is difficult to predict and can proceed very rapidly. Also, the epidural catheter is available after the delivery. We recommended the epidural catheter is maintained for 24–48 hr postpartum.

Les auteurs présentent deux cas d'hyperréflexie autonome (HA) survenue pendant le travail de parturientes souffrant de lésion de la moelle épinière. Dans un cas, l'HA est apparue avant l'accouchement, et dans l'autre, après l'accouchement. Dans le premier cas, l'HA a été contrôlée efficacement avec une anesthésie épidurale. Dans le deuxième, on a réussi à contrôler la pression artérielle (PA) avec de la nicardipine. Cependant, une surdose de nicardipine a produit, comme il survient au cours de l'HA, une vasodilatation et des effets secondaires dont de la céphalée, des rougeurs et des palpitations. A cause des effets secondaires de la nicardipine, les auteurs recommandent l'anesthésie épidurale pour le contrôle de l'HA, parce que l'anesthésie épidurale ne contrôle pas seulement la PA, mais bloque aussi les stimuli nocifs et atténue les symptômes de l'HA. Les auteurs suggèrent d'insérer le cathéter épidural deux ou trois semaines avant la date prédite de l'accouchement, parce que le début du travail est difficile à prédire chez les patientes qui souffrent d'une lésion de la moelle épinière et parce qu'il peut se dérouler très rapidement. De plus le cathéter épidural reste disponible après l'accouchement. Les auteurs recommandent le maintien du cathéter pendant les 24 à 48 heures du postpartum.

Key words
ANAESTHESIA: obstetrical;
ANAESTHETIC TECHNIQUES: epidural;
COMPLICATIONS: autonomic hyperreflexia.

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Autonomic hyperreflexia occurs in about 85% of patients with spinal lesion above the level of T_6.[2] This can be caused by noxious stimuli such as distention of the bladder, cervix, or rectum, which evoke a mass triggering of sympathetic and parasympathetic afferents below the level of the cord lesion that are not inhibited by supraspinal centres. Autonomic hyperreflexia is a serious complication during pregnancy, labour and delivery in the patient with spinal cord injury.[3] We present two cases of AH during labour in women with spinal cord damage and comment upon the efficacy of epidural anaesthesia in managing the syndrome.

Case reports

Case #1

The 33-yr-old white woman was admitted at the 37th week of gestation. She had become quadriplegic at the age of 16 yr following a spinal cord injury at the C_{3-5} level. Neurological examination revealed loss of sensory, motor, and reflex function below the C_6 level and partial motor function at the C_6 level. Although there was no evidence of preterm labour, an epidural catheter was placed in the L_2–L_3 epidural space, because she exhibited signs suggestive of AH such as facial flushing, sweating, and piloerection during abdominal distension or vaginal examination. The epidural catheter was placed at the 37th week of gestation and was kept *in situ* until its removal after delivery. No AH was observed with the epidural injection of 5 to 10 ml lidocaine 1%, given before each vaginal examination. At the 40th week of gestation, symptoms of AH such as pounding headache, hypertension,

marked diaphoresis with piloerection, and flushing above the level of the cord lesion without epidural blockade heralded the beginning of labour. However, epidural administration of 10 ml lidocaine 2% and meperidine 15 mg produced an immediate reduction in the arterial blood pressure (BP) from 160/110 to 110/62 mmHg, and relieved the symptoms of AH. Thirty minutes later, the patient was transferred to the delivery room and a female infant weighing 2704 g, Apgar score 10 was delivered spontaneously. The labour was 21 min. Epidural blockade using 10 ml lidocaine 2% was administered twice for mild AH after delivery. The catheter was removed 48 hr later. No symptoms of AH were observed thereafter.

Case #2

This 42-yr-old Japanese woman had developed paraplegia at 18 yr due to meningitis. Neurological examination revealed loss of sensory, reflex, and motor function below the level of T_5 except for minimal flexor reflexes in the lower extremities. No complications were found except for functional disorder of right kidney. When she was hospitalized in labour at the 37th week of gestation, her uterine contractions triggered headache, sweating, facial flushing and hypertension. An epidural catheter was placed in the L_{1-2} epidural space in the delivery room. Ten minutes after the epidural injection of 5 ml lidocaine 1%, her BP decreased from 145/83 to 120/77 mmHg and the symptoms of AH were relieved. As the uterine contractions were too weak for delivery, a Caesarean section was scheduled. On entering the operating room, we suspected catheter misplacement, because epidural injection of 15 ml lidocaine 1% failed to reduce the BP. Anaesthesia was induced with thiopentone, 5 $mg \cdot kg^{-1}$ *iv*, and tracheal intubation was facilitated with succinylcholine chloride, 60 mg *iv*. Anaesthesia was maintained with sevoflurane, 1.0 ~ 2.5%, and nitrous oxide, 67%. We administered nicardipine, 0.3 ~ 1.0 $\mu g \cdot kg^{-1} \cdot min^{-1}$ *iv*, which reduced the BP from 180/98 to 140/67 mmHg. A female infant, weighing 2880 g and an Apgar score 8 was delivered. Upon the patient's transfer to the intensive care unit, she exhibited hypertension of 202/120 mmHg so that nicardipine, 2.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ *iv*, was repeated. Epidural injection was not administered after delivery. Re-insertion of epidural catheter was not considered, because the BP had decreased to 150/90 mmHg with injection of nicardipine (2.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$). Although the BP decreased with nicardipine, symptoms, such as sweating and flushing, persisted. The patient's consciousness was obtunded without sedation during the night, but resolved by the next morning and trachea was extubated. At this point, the BP was 165/98 mmHg with nicardipine, 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$, and no symptoms of AH were observed. Three hours after extubation, she complained of discomfort. She became gradually incoherent and nonresponsive, and then apnoeic, and the trachea was reintubated. Blood gas analysis showed pH of 7.43, PaO_2 of 167, $PaCO_2$ of 27, BE of −6.1, with a blood glucose of 47 $mg \cdot dl^{-1}$. After 100 ml glucose 50% *iv*, the blood glucose concentration was elevated to 108 $mg \cdot dl^{-1}$. A CT scan of the brain revealed no haemorrhagic or ischaemic lesions. After 48 hr of controlled ventilation and other appropriate support including the administration of glucose, the patient showed a full recovery.

Discussion

Autonomic hyperreflexia can be triggered by uterine contractions during labour in women with a transection of the spinal cord above T_6.[4–7] The disorder is manifested by the sudden onset of marked hypertension, accompanied by bradycardia, cardiac arrhythmias, headache, piloerection, sweating, and flushing of the skin above the level of the cord lesion. The result may be loss of consciousness, seizures, and a fatal cerebral bleeding.[8]

The neuroanatomical pathway of this syndrome was first described by Kurnick[9] in 1956. In a patient with a transection of the spinal cord, afferent impulses enter the isolated spinal cord via the pelvic and pudendal nerves and elicit reflex autonomic output over the splanchnic outflow which is not modulated by higher centres as normally occurs in the neurologically intact individual. The result is generalized vasoconstriction below the level of injury. The resultant hypertension stimulates the baroreceptors, which may lead to bradycardia via the intact pathways to the heart, and to vasodilatation via the intact sympathetic pathways above the level of injury.

Watson,[7] Baraka[10] and Pauzner[11] have suggested that regional anaesthesia is useful in preventing and controlling AH during labour. While the use of spinal anaesthesia for preventing AH has been reported more frequently than that of epidural anaesthesia,[12] the use of spinal anaesthesia in patients with spinal cord damage may be hazardous, because the extensive sympathetic block in these patients can be complicated by severe hypotension, bradycardia, and even, asystole. It is important to note that AH can develop before or after delivery. In our two patients, AH occurred before, during and after delivery. A single administration of spinal anaesthesia was thus impractical in obtaining a prolonged period of reflex control. Baraka[10] recommends epidural meperidine, because opioids produce selective blockade of the opioid receptors in the substantia gelatinosa of the dorsal horn of spinal cord, and selectively antagonize the discharge evoked by peripheral stimulation.

Our experience suggests that an epidural catheter can be placed two to three weeks before the anticipated date

of delivery, because the onset of labour in a patient with spinal cord damage is difficult to predict and can proceed very rapidly, as in Case #1. The epidural catheter can be maintained with care in avoiding catheter displacement and infection. Also, the epidural catheter is available after the delivery. Crosby[13] recommended that the epidural catheter be maintained for 24–48 hr postpartum. We successfully controlled AH after delivery using epidural anaesthesia in Case #1, but failed in Case #2. We presume that the epidural catheter of Case #2 was displaced during transfer to the operating room, because epidural anaesthesia had been effective in the delivery room. Problems in using epidural anaesthesia for preventing AH include (1) the technique for epidural insertion in patients with spinal cord damage, (2) the dose of agent to be administered, (3) evaluating the level of sensory and sympathetic blockade that is achieved (4) the longterm maintenance of the catheter displacement and infection.

We could not determine the cause of the loss of consciousness that followed tracheal extubation in Case #2. It may have been due to hypoglycaemia (glucose level of 47 $mg \cdot dl^{-1}$) and/or hyperventilation ($PaCO_2$ of 27). Emergency CT scan of the brain revealed no haemorrhagic or ischaemic lesion, and she recovered completely by the next day.

If AH is not prevented and controlled by the administration of a regional anaesthesia, a short-acting antihypertensive agent may be given under careful observation. Brunstein recommended nifedipine for prevention of AH.[14] Our experience suggests that the administration of nicardipine lowers the BP without causing adverse effects in the newborn. In Case #2, however the symptoms such as sweating and flushing persisted on the night after surgery, in spite of a reduction of BP to 150/90 mmHg with nicardipine (2.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$). The persistence of these symptoms may have been drug induced rather than from AH. No symptoms were observed the next morning, although the BP remained at 165/98 mmHg because the dose of nicardipine was low (0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$). Calcium channel blockers should be used carefully because overdose of nicardipine produces vasodilatation and side effects of headache, flushing and palpitation that are similar to those of AH.[1] A short acting beta blocker might be suitable in this situation. Considering these side effects of nicardipine, we think the epidural catheter for Case #2 should have been repositioned.

In summary, we have confirmed the efficacy of epidural anaesthesia in controlling AH in labouring patients with chronic spinal cord damage. We recommend this approach for controlling AH, epidural anaesthesia does not only lower the BP, but also blocks the noxious stimuli and relieves the symptoms of AH.

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