Enrique J. Carrero MD,
Mercè Agustí MD,
Neus Fàbregas MD PhD,
Francesc Valldeoriola MD,*
Carmen Fernández MD PhD

Unilateral trigeminal and facial nerve palsies associated with epidural analgesia in labour

Purpose: To report a rare complication related to epidural analgesia for obstetrics: the association of unilateral trigeminal and facial nerve palsies in a patient with the clinical syndrome of intracranial hypotension.

Clinical features: A 38-yr-old woman was admitted in pre-term labour (at 35 wk gestation). She was receiving 40 mg methadone *po* daily for opioid addiction. Epidural analgesia for labour was established with 9 ml bupivacaine 0.25%. The patient underwent normal vaginal delivery of a 2500 g female infant. She developed post-dural puncture headache (PDPH) on the third postpartum day which was managed by palliative measures: bed rest (patient's position of choice), increased hydration (water: 3 litres *po* per day), lysine acetyl salicylate (5.4 g *po* per day) and caffeine (600 mg *po* per day). She developed paraesthesiae and numbness of the right side of the face one day after the onset of PDPH and unilateral facial nerve palsy two days later. There was no evidence of dural puncture and no cause was found. Treatment of the nerve palsies with epidural blood patch or epidural dextran 40 was not considered to be indicated. Headache disappeared on the 10th postpartum day and trigeminal palsy regressed. At the time of discharge, on the 17th postpartum day, neurological examination showed minimal facial asymmetry. The patient refused further exploration and follow-up.

Conclusion: Post delivery trigeminal and facial nerve palsy in a 38 yr old woman recovered spontaneously with conservative therapy.

Objectif : Rapporter une complication rare liée à l'analgésie péridurale obstétricale : l'association de paralysie unilatérale du nerf trijumeau et du nerf facial chez une patiente qui présentait le syndrome d'hypotension intracrânienne.

Aspects cliniques : Une femme de 38 ans, en travail prématuré, a été admise à l'hôpital à 35 semaines de grossesse. Elle recevait quotidiennement 40 mg de méthadone *po* pour contrer une dépendance aux opioïdes. L'analgésie péridurale pour le travail a été induite avec 9 ml de bupivacaine 0,25 %. La patiente a accouché normalement par voie vaginale et a donné naissance à une fille de 2 500 g. Elle a présenté, le troisième jour du postpartum, une céphalée post-ponction durale (CPPD) qui a été traitée par des mesures palliatives : repos au lit (position préférée de la patiente), hydratation accrue (eau : 3 litres *po* par jour), lysine salicylate d'acétyle (5,4 g *po* par jour) et caféine (600 mg *po* par jour). Elle a éprouvé une paresthésie et des engourdissements du côté droit du visage le jour suivant le début de la CPPD et, deux jours plus tard, une paralysie unilatérale du nerf facial. Il n'y avait pas d'évidence de ponction de la dure-mère et aucune cause n'a été identifiée. On n'a pas jugé que le traitement de la paralysie des nerfs au moyen d'un bloodpatch péridural ou de dextran 40 péridural était indiqué. La céphalée a disparu au 10^e jour postpartum et la paralysie du trijumeau a aussi régressé. Au moment du congé, le 17^e jour postpartum, l'examen neurologique démontrait une asymétrie faciale minimale. La patiente a refusé tout examen supplémentaire et toute relance.

Conclusion : Une femme de 38 ans a récupéré spontanément d'une paralysie trigémiale et faciale postpartum après une thérapie conservatrice.

From the Departments of Anaesthesiology and Neurology,* Hospital Clínic i Provincial de Barcelona, Barcelona University, Spain.

Address correspondence to: Enrique J Carrero MD, Departamento de Anesthesiologia, Hospital Clínic i Provincial de Barcelona, Villarroel 170, 08036, Barcelona, Spain. Phone: 07-34-3-2275400; Fax: 07-34-3-2275454; E-mail: fabregas@medicina.ub.es

Accepted for publication June 25, 1998.

CRANIAL nerve palsy is an uncommon neurological complication following epidural anaesthesia.¹ Isolated facial or trigeminal involvement during epidural anaesthesia has been previously described²⁻⁴ but, there are no reports describing involvement of both nerves. We report the case of a woman who presented with paraesthesiae in the right trigeminal territory and right facial nerve palsy on the fourth and fifth days after epidural analgesia for obstetrics had regressed.

Case report

A 38-yr-old white woman was admitted in pre-term labour (at the 35th week). She had had an uncontrolled pregnancy. Her past medical history included heroin addiction, one induced abortion and one pre-term delivery (TPAL: 0,1,1,1). She had negative serology for acquired immunodeficiency syndrome (AIDS). She was receiving methadone, 40 mg *po* daily, as a substitute treatment for her opioid addiction. The patient requested epidural analgesia and an epidural catheter was inserted at the L₃₋₄ interspace through an 18-gauge Tuohy needle. There was no evidence of dural puncture. Analgesia was established with 9 ml bupivacaine 0.25% without evidence of extensive block. The patient underwent a normal vaginal delivery of a 2500 g female infant. The APGAR scores at one and five minutes were nine and ten. One hour later the epidural catheter was withdrawn. On the third postpartum day she complained of headache worsened by upright position and relieved by lying flat. On the fourth postpartum day she developed paraesthesiae and numbness of the right side of the face. The next day the patient showed right-sided facial weakness with drooping of her lip to the left and she was unable to close the eyelid.

The patient was afebrile and general examination was normal. Neurological examination showed a fully conscious patient. Minimal neck stiffness was observed but extension of the knee joint did not produce back pain and flexion of the neck did not result in lower limbs flexion (Kernig and Brudzinski signs absent). Pupils reacted normally to light. Ocular motility was normal. Eyeblink rate was slightly increased. Facial nerve evaluation disclosed weakness of frontalis, orbicularis oculi and orbicularis oris muscles on the right side. The corneal reflex was reduced on the right side. Hypoalgesia and hypoesthesia were observed on the right side of the face following the typical distribution of the trigeminal nerve affecting its three branches. The jaw reflex and the function of the masseter muscle was preserved. The remainder of the neurological examination showed no abnormalities.

Blood analyses disclosed normal hepatic and renal function. Electrolyte, haematological and coagulation

tests were normal. Electrocardiogram and chest X-ray were also normal. A computed tomography (CT) scan of the head disclosed no abnormality.

Treatment of post-dural puncture headache and cranial nerve palsies consisted on palliative measures: bed rest (patient's position of choice), hydration (water: 3 litres *po* per day), lysine acetyl salicylate (5.4 g *po* per day) and caffeine (600 mg *po* per day). The headache progressively improved and disappeared on the 10th postpartum day. The patient was discharged from hospital on the 17th postpartum day. Neurological examination showed only a minimal facial asymmetry. Weakness of the orbicularis oculi was still present and closure of the eyelid was incomplete. We did not obtain permission from the patient to perform a neurophysiological examination and a magnetic resonance of the brain. Patient's follow-up was unsuccessful.

Discussion

This case describes an unusual neurological complication of epidural analgesia in labour that has not been previously reported: the association of unilateral trigeminal and facial nerve palsies. No prophylaxis of the post-dural puncture headache (PDPH) was planned because of the lack of evidence of dural puncture. In our hospital, the protocol for the management of PDPH consists, initially, of palliative measures and epidural dextran 40 is reserved for severe or persistent headache after 48 hr of conservative treatment. Epidural dextran treatment was not performed because symptoms of cranial nerve palsy (CNP) appeared just 24 hr after the onset of PDPH. The course of clinical events suggested the clinical syndrome of intracranial hypotension as the most possible explanation of CNP. In this case, there was a good outcome with conservative treatment.

Cranial nerve palsy, as a complication of epidural analgesia in labour, is rare. Scott¹ reported five cases in 506,000 peridural blocks (84% for relief of pain in labour). Epidural anaesthesia can induce CNP through different mechanisms. High cephalic spread of local anaesthetic, central nervous system infection, idiopathic nerve palsy, exacerbation or unmasking of preexisting disease and changes in cerebrospinal fluid (CSF) pressure are considered in the differential diagnosis.

Altered anatomy and physiology of the epidural space related to pregnancy in combination with body positioning and catheter migration (subdural, subarachnoid or asymmetric position) may explain the eventual extensive cephalic spread of local anaesthetic. Furthermore, there is hormonal enhancement of local anaesthetic nerve-blocking action in pregnancy which is maximal just before term, and carries the risk of relative overdose and a high segmental blockade.² Unilateral trigeminal nerve block, associated with Horner's syndrome after epidural analge-

sia in labour, has been reported by Gazmuri³ and Sprung.⁴ The neurological symptoms developed 90 min after the last dose of local anaesthetic in the former report and during the onset of a high level of sensory anaesthesia in the latter. Symptoms disappeared within four hours. The authors suggested excessive upward extension of blockade reaching the cervical sympathetic fibres and the spinal tract, nucleus and/or the exit from the pons of the trigeminal nerve as a more plausible explanation. Accidental misplaced injections and extensive spread of block may be delayed for hours or days if continuous epidural analgesia is employed (catheter migration),² but this technique was not employed in this case. The last dose of local anaesthetic was administered before delivery and the epidural catheter was withdrawn one hour later. The onset of symptoms during the fourth and fifth postpartum days ruled out then this mechanism.

Inadvertent intravascular injection was not considered in differential diagnosis because toxic plasma levels of local anaesthetic usually result in seizures and cardiovascular depression.

Central nervous system infection is a rare complication of spinal and epidural anaesthesia in obstetrics⁵ and is not supported by the clinical course and spontaneous remission of symptoms presented by this patient. A history of fever, non-positional headache, meningismus and some degree of impairment of higher integrative functions was not seen in our patient. Although CNPs are not uncommon late in the course of meningitis, focal neurological deficits at the time of presentation should prompt CT scan to exclude brain abscess, cerebral tumour or focal encephalitis.⁶ The course of clinical events and normal CT argue against these aetiologies.

Acute idiopathic polyneuritis (Landry-Guillain Barré syndrome) were excluded because of clinical criteria: there was no ascending paralysis, the limbs were not affected and neurological symptoms did not progress.

Idiopathic facial nerve palsy (Bell's palsy) can develop in the early postpartum period⁷ and must be considered in the differential diagnosis. In our patient, association of facial and trigeminal nerve palsies made this diagnosis improbable.

Cranial nerve palsies can also occur from an exacerbation or unmasking of preexisting neurological disease. Nevertheless, clinical examination, normal brain scan and the presence of previous postural headache suggest other pathophysiological mechanism.

Cranial nerve palsies after epidural analgesia for obstetrics usually appear after dural puncture and are associated with PDPH. Both are thought to result from low CSF pressure caused by leakage of CSF through the dural hole (intracranial hypotensive syndrome).¹ Accidental dural puncture is a rare complication of

obstetric epidural analgesia and occurs in about 1% of cases.¹ In one-third of the accidental punctures no signs of dural puncture can be seen at the time of catheter insertion² and retrospective diagnosis is made after PDPH appears. This case supports this hypothesis.

Direct anatomical connections between the cochlea and the subarachnoid space and the anatomical pathways in the skull of the third, fourth, sixth and eighth cranial nerves have been used to explain why these nerves are more frequently affected when CSF loss occur. We have been unable to find previous reports involving both the trigeminal and facial nerves. Magnetic resonance imaging studies have demonstrated a downward displacement of intracranial contents in patients with PDPH and intracranial hypotension.⁸ Stretching of both nerves cannot be excluded. However, it would be expected that other cranial nerves would also have been involved.

Predictable clinical factors for developing CNP are difficult to determine. Review of case reports of CNP related to low CSF pressure shows that previous severe and prolonged PDPH is a common symptom.^{9,10} We agree with Bromage² in considering that the severity of PDPH is related to the rate of CSF loss. If the decrease in CSF volume is excessive, life-threatening subdural haematoma can occur.^{1,11} We think the presence of persistent PDPH must be considered a potentially premonitory symptom of more severe complications and requires prompt treatment and strict patient follow-up. The course of clinical events, however, cannot be always anticipated and, in consequence, prevented. Cranial nerve palsy or intracranial subdural haematoma have been described without premonitory symptoms.¹²

Management of dural taps and PDPH occurring after epidural analgesia for pain relief in labour varies widely among institutions.^{13,14} Our unit has a protocol for both prophylaxis and treatment of PDPH. In the case reported no prophylactic measures were taken because a dural tap was not suspected. Our protocol for management of PDPH consists, initially, of palliative measures (bed rest, hydration, analgesics and caffeine). With conservative treatment many spinal headaches resolve spontaneously in two or three days.^{15,23} We do not have experience in performing epidural blood patches although it is an effective method.^{16,17} Instead, we administer epidural dextran¹⁸ which is reserved for severe or persistent headache (after 48 hr conservative treatment). In the present case, trigeminal nerve palsy appeared just one day after PDPH before the efficacy of conservative treatment was assessed and before considering the epidural alternative.

Development of CNP is rare before the fourth day after dural puncture (mean time of presentation is at 10 days) and extremely rare after resolution of PDPH.¹⁹ To

prevent the development of CNP after PDPH, Dunbar and Katz¹⁹ suggested conservative therapy for three days and, if ineffective on relieving PDPH on the fourth day, performing an epidural blood patch.

There is controversy about the efficacy of epidural blood patch in the treatment of CNP. Reversible neurological changes have been reported with epidural blood patch in two patients in whom CSF was drained intentionally for a neurological procedure.²⁰ Most often, this treatment is ineffective in reversing established CNP.¹⁹ On the other hand, transient cranial nerves palsies have been described as severe complications of epidural blood patches.^{16,17,21,22} An increase in epidural and subarachnoid pressures secondary to the injection of blood has been postulated in previous reports. Perez *et al.*²² suggested that increased pressure could damage neural fibres both directly and indirectly compromising neural blood flow. Physiological modifications during pregnancy or oedema or nerve stretching secondary to previous loss of CSF could contribute to such injury if a blood patch had been done. Cranial nerves with narrow courses, such as the seventh nerve, seem to be more liable to stretching. In this case, we excluded epidural treatments with blood or dextran 40 because of the possibility of worsening the symptomatology.

The prognosis of CNP associated with dural puncture is usually good with resolution of palsies within a week. However, neurological symptoms may persist for months and incapacitate patients in their normal activities.²³ In the case reported, only minimal facial asymmetry persisted on the 17th postpartum day. Patient follow-up was unsuccessful and final CNP evolution unknown. Our experience shows that persistent neurological deficits, when they occur, are notified. In agreement with the literature patient recovery is the most likely evolution.^{1,23}

As initially suggested by Costigan and Sprigge,²⁴ we also believe that all pregnant women requesting epidural analgesia in labour must be aware of the risk of PDPH. Severe neurological complications associated with the clinical syndrome of intracranial hypotension are usually preceded by PDPH. It must be noted that such complications can appear or recur after discharge from hospital. In our opinion, although the risk of CNP associated with PDPH is low, mothers should also be warned of such a possibility and quickly re-admitted to hospital, if necessary, before serious sequelae occur.

Conclusion

Follow-up of the patient is essential for early diagnosis of cranial nerve palsies. Nerve palsies develop quickly after the onset of post-dural puncture headache, probably as a consequence of a rapid decrease in cerebrospinal fluid pressure. In this case, unilateral trigeminal

and facial nerve palsies had a benign course with only conservative treatment.

Acknowledgment

The authors wish to thank Professor Carmen Gomar Sancho MD PhD, Head, Department of Anaesthesiology, Hospital Clínic y Provincial, Barcelona, for her valuable support.

References

- 1 Scott DB, Hibbard BM. Serious non-fatal complications associated with extradural block in obstetric practice. *Br J Anaesth* 1990; 64: 537-41.
- 2 Bromage PR. Neurologic complications of regional anesthesia for obstetrics. In: Shnider SM, Levinson G (Eds.). *Anesthesia for Obstetrics*, 3rd ed. Baltimore: Williams & Wilkins Publishers Inc., 1993: 433-53.
- 3 Gazmuri RR, Riche CA, Dagnino JA. Trigeminal nerve block as a complication of epidural anesthesia. *Reg Anesth* 1992; 17: 50-1.
- 4 Sprung J, Haddox JD, Maitra-D'Cruze AM. Horner's syndrome and trigeminal nerve palsy following epidural anaesthesia for obstetrics. *Can J Anaesth* 1991; 38: 767-71.
- 5 Cascio M, Heath G. Meningitis following a combined spinal-epidural technique in a labouring term parturient. *Can J Anaesth* 1996; 43: 399-402.
- 6 Haas DW, Kaiser AB. Infecciones del sistema nervioso central. In: Shoemaker WC, Ayres S, Grenvik A, Holbrook PR (Eds.). *Tratado de Medicina Crítica y Terapia Intensiva*, 3rd ed. Buenos Aires: Editorial Médica Panamericana S.A., 1996: 1291-304.
- 7 Mc Gregor JA, Guberman A, Amer J, Goodlin R. Idiopathic facial nerve paralysis (Bell's palsy) in the late pregnancy and the early puerperium. *Obstet Gynecol* 1987; 69: 435-8.
- 8 Pannullo SC, Reich JB, Krol G, Deck MDF, Posner JB. MRI changes in intracranial hypotension. *Neurology* 1993; 43: 919-26.
- 9 Vandam LD, Dripps RD. Long-term follow-up of patients who received 10,098 spinal anesthetics. Syndrome of decreased intracranial pressure (headache and ocular and auditory difficulties). *JAMA* 1956; 16: 586-91.
- 10 Lee JJ, Roberts RB. Paresis of the fifth cranial nerve following spinal anesthesia. *Anesthesiology* 1978; 49: 217-8.
- 11 Bjärnhall M, Ekseth K, Boström S, Vegfors M. Intracranial subdural haematoma - a rare complication following spinal anaesthesia. *Acta Anaesthesiol Scand* 1996; 40: 1249-51.
- 12 Wildsmith JA, Lee LA. Neurological sequelae of spinal anaesthesia (Editorial). *Br J Anaesth* 1989; 63: 505-8.

- 13 *Sajjad T, Ryan TDR.* Current management of inadvertent dural taps occurring during the siting of epidurals for pain relief in labour. A survey of maternity units in the United Kingdom. *Anaesthesia* 1995; 50: 156–9.
- 14 *Souron V, Simon L, Sacquin P, Hamza J.* Current management of inadvertent dural taps occurring during epidural analgesia for pain relief in labour: a French postal survey. *Br J Anaesth* 1998; 80(Suppl): 153.
- 15 *Reid JA, Thorburn J.* Headache after spinal anaesthesia (Editorial). *Br J Anaesth* 1991; 67: 674–7.
- 16 *Abouleish E, de la Vega S, Blendinger I, Tio T-O.* Long-term follow-up of epidural blood patch. *Anesth Analg* 1975; 54: 459–63.
- 17 *Ostheimer GW, Palahnuk RJ, Shnider SM.* Epidural blood patch for post-lumbar-puncture headache (Letter). *Anesthesiology* 1974; 41: 307–8.
- 18 *Salvador L, Carrero E, Castillo J, Villalonga A, Nalda MA.* Prevention of post dural puncture headache with epidural-administered dextran 40 (Letter). *Reg Anesth* 1992; 17: 357–8.
- 19 *Dunbar SA, Katz NP.* Failure of delayed epidural blood patching to correct persistent cranial nerve palsies. *Anesth Analg* 1994; 79: 806–7.
- 20 *Guido LJ, Patterson RH Jr.* Focal neurological deficits secondary to intraoperative CSF drainage: successful resolution with an epidural blood patch. *J Neurosurg* 1976; 45: 348–51.
- 21 *Lowe DM, McCullough AM.* 7th nerve palsy after extradural blood patch. *Br J Anaesth* 1990; 65: 721–2.
- 22 *Perez M, Olmos M, Garrido FJ.* Facial nerve paralysis after epidural blood patch. *Reg Anesth* 1993; 18: 196–8.
- 23 *Thorsén G.* Neurological complications after spinal anaesthesia. *Acta Chir Scand Suppl* 1947; 121: 1–272.
- 24 *Costigan SN, Sprigge JS.* Dural puncture: the patient's perspective. A patient survey of cases at a DGH maternity unit 1983–1993. *Acta Anaesthesiol Scand* 1996; 40: 710–4.