

- por el enfermo crónico que acude a urgencias. *Rev Clin Esp*. 2010;210:379–88.
2. Roca M, Escanilla A, Monje A, Baño V, Planchat LM, Costa J, et al. Banco de tejidos neurológicos de Sant Joan de Déu-Serveis de Salut Mental para la investigación de las enfermedades mentales. La importancia de un programa de donación en vida. *Psiquiatr Biol*. 2008;15:73–9.
 3. Rivas E, Teijeira S, Tardio A, Fachal C, Quintáns B, Navarro C. A brain tissue bank in a neuropathology laboratory. Basic methodology. *Neurología*. 2003;18:709–15.
 4. Balasa M, Gelpi E, Antonell A, Rey MJ, Sánchez-Valle R, Molinuevo JL, et al. Neurological tissue bank/University of Barcelona/Hospital Clínic NTB/UB/HC Collaborative Group. Clinical features and APOE genotype of pathologically proven early-onset Alzheimer disease. *Neurology*. 2011;76:1720–5.
 5. Ferrer I. Bancos de tejidos neurológicos. *Rev Esp Patol*. 2004;37:57–64.
- M. Lozano Espinosa, S. Bueno Macias, F. Morato Montaña, R. Saura Llamas, C. Alfonso Cano, M. Leal Hernández*
- Centro de Salud Docente de San Andrés, Unidad Docente de Medicina Familiar y Comunitaria de Murcia, Murcia, Spain*
- * Corresponding author.
E-mail address: arboleja@yahoo.es (M. Leal Hernández).

Declaration of conflicts of interest[☆]

Declaración sobre el conflicto de intereses

Dear Editor:

Your recent editorial on conflicts of interest and scientific publications was very interesting. I would like to share an idea regarding the statement ‘‘A declaration of relationships should be required in the communication of research, but its existence should not prejudge inappropriate conduct’’.¹ Essentially, publishing conflict of interest statements is common practice for all journals. The problem resides in how to verify authors’ declarations. Generally speaking, those involved in the journal assume that the information provided is true, and the issue therefore arises in cases where statements are incomplete. I completely agree that other types of misconduct may exist, and that other issues related to conflict of interest may also come up. Despite complicated declaration forms, it is impossible to prevent authors from hiding their conflicts. How then can the problem be

solved? Firstly, an additional system for detecting conflicts of interest must be in place, such as using an online tool to search for potential conflicts of interest related to the author (for example, mentioning specific products, working as a consultant, etc.). Secondly, providing referees with some information about the author may be useful for identifying possible conflicts of interest. Some may argue that this practice could create reviewer bias and affect the decision-making process for the document submitted. However, this step could be taken after the article has been accepted, for the purpose of checking for undeclared conflicts of interest. Lastly, the process used to check for undeclared conflicts of interest should be comparable to processes for detecting other types of inappropriate conduct, such as plagiarism.

Reference

1. Matías-Guiu J, García-Ramos R. Conflicto de intereses y publicaciones científicas. *Neurología*. 2012;27:1–3.

V. Wiwanitkit^{a,b}

^a Hainan Medical University, China

^b Joseph Ayobabalola University, Nigeria

E-mail address: wviroj@yahoo.com

[☆]Please cite this article as: Wiwanitkit V. Declaración sobre el conflicto de intereses. *Neurología*. 2013;28:380.

Subdural haematoma secondary to epidural anaesthesia. A rare complication[☆]

Haematoma subdural secundario a anestesia epidural. Una complicación infrecuente

Dear Editor:

Intracranial subdural haematoma (SH) rarely presents as a complication of epidural anaesthesia, although we do find cases in the literature. If the dura mater is punctured during

this procedure, there is a risk that SH will occur, and that risk may be related to cerebrospinal fluid (CSF) hypotension syndrome.

Symptoms of SH are linked to the mass effect and displacement of structures, and they depend on the patient’s age; haematoma location, size, and speed of onset; the patient’s prior clinical condition; and the compression of intracranial structures. Distinguishing CSF hypotension syndrome from SH due to intracranial hypertension may be difficult in differential diagnosis, and this can be an obstacle to diagnosing the condition early.

We present the case of a patient with no relevant personal history who presented a SH secondary to the epidural anaesthesia received during childbirth.

A 27-year-old woman came to our hospital’s emergency department on 2 consecutive occasions due to a frontal and occipital headache that increased while standing and

[☆]Please cite this article as: López Soler EC, et al. Hematoma subdural secundario a anestesia epidural. Una complicación infrecuente. *Neurología*. 2013;28:380–2.

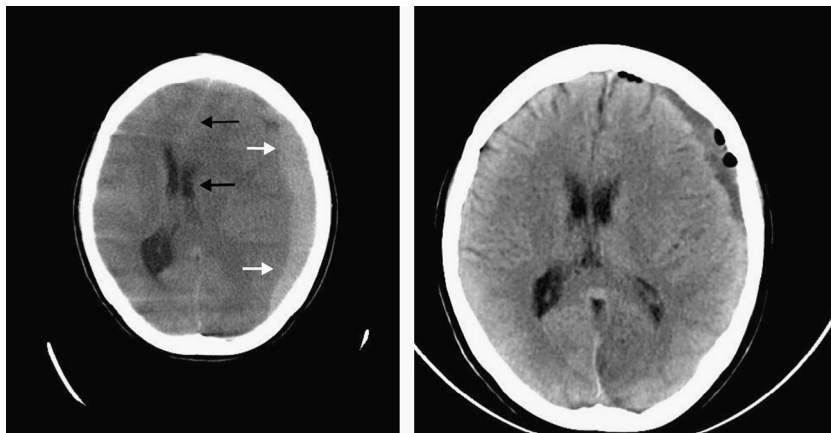


Figure 1 CT Left: acute phase image showing the subdural haematoma (white arrows) and the midline shift (black arrows). Right: haematoma after surgery.

improved upon lying down. These visits took place 4 days after a normal vaginal delivery without complications. Epidural anaesthesia had been administered during child-birth by means of an 18 gauge Weiss needle used to inject levobupivacaine into the L2–L3 intervertebral disc space. Blood pressure (BP) was 148/73 mm Hg and heart rate (HR) was 70 bpm. Physical examination did not reveal any pathological signs, marfanoid/leptosomatic habitus, or articular hypermobility/skin hyperlaxity. Neurological examination showed no focal signs. The patient was discharged with analgesic and anti-inflammatory treatment. In the following weeks, she experienced a postural headache that did not prevent her from performing her daily activities and that resolved or lessened with the analgesic treatment prescribed. Approximately one month later, she returned to our department due to an intense headache which did not respond to postural change or improve with habitual analgesics. It was accompanied by vomiting and a state of anxiety and agitation. BP was 136/86 mm Hg and HR was 37 bpm. Neurological examination revealed no pathological findings. The blood count, biochemical and coagulation study, venous blood gas values, and urine analyses provided no significant findings. The ECG detected sinus bradycardia with no other findings. The chest radiography was normal. The brain CT showed an extensive subacute left fronto-temporo-parietal SH with significant mass effect shifting the midline and ventricular system 14 mm to the right (Fig. 1). After the case was discussed with the neurology department at the referral hospital, the patient was transferred to that centre. While in the emergency room prior to being transferred, she experienced an abrupt decrease in level of consciousness (GCS score of 3) and anisocoria. Doctors therefore decided to administer sedatives, relaxants, and anti-oedema drugs, as well as orotracheal intubation and mechanical ventilation.

Upon her arrival at the referral hospital, doctors performed emergency evacuation of the haematoma using 2 burr holes (one in the parietal and frontal lobe and the other in the left parietal and posterior lobe). Fluid exited under considerable pressure. The patient made good clinical progress; fluid output was abundant and her

level of consciousness increased (GCS 15). Brain CT was then performed, revealing re-expansion of the cerebral parenchyma and resolution of the midline shift (Fig. 1). The patient was subsequently discharged and monitored by her primary care doctor and local neurologist; she will also require check-ups in the neurosurgery outpatient unit.

SH is defined as a collection of blood in the cranial cavity between the dura and arachnoid mater. Its most common aetiology is trauma. Chronic SH was first described by Wepfer (1658) and Morgani (1761). In 1857, Virchow wrote that the aetiology of what he called 'pachymeningitis hemorrhagica interna' was not traumatic.^{1,2} In 1914, Trotter^{1–3} considered the possibility of SH being caused by the rupture of small veins in the arachnoid mater. SH as a result of epidural anaesthesia is rare, with a prevalence ranging from 1/500 000 to 1/1 000 000.⁴

Post-dural puncture headache (PDPH) is the most frequent complication of epidural anaesthesia. It is associated with CSF hypotension syndrome, since CSF extravasation by lumbar puncture decreases the intracranial pressure. According to the International Headache Society's diagnostic criteria (2004, ICHD-II),^{5,6} the characteristic feature of PDPH is postural headache that appears or intensifies after 15 minutes of standing and improves upon lying down for a similar time period. Studies show that symptoms last no more than 5 days in most cases.⁵ When CSF pressure decreases suddenly, the displacement of brain structures may cause intracranial subdural veins to tear, giving rise to SH.^{7–10} Many authors have linked the appearance of SH to the technique and material used in lumbar puncture, stating that larger needle diameter, pencil-point type needle tips, and the angle of the bevel are associated with a higher probability of vascular lesion.¹ However, the solution to the problem does not seem to reside in the type of needle, since there are also documented cases of SH secondary to epidural anaesthesia with fine gauge needles.^{1–5}

We should highlight that during the acute phase of SH, intracranial pressure (ICP) increases due to the larger brain volume. In advanced stages, this phenomenon leads to hypoperfusion and ischaemia of the brainstem, which

increases the activity of the sympathetic and parasympathetic autonomous nervous system in an attempt to increase the stroke volume and the BP to a level exceeding the pressure on the brainstem. The purpose of this process is to overcome the vascular resistance to cerebral blood flow caused by increased ICP.^{11–15} This physiological response to elevated ICP is called the Cushing reflex and it is described clinically by the triad of arterial hypertension, bradycardia, and irregular breathing, indicators of poor clinical prognosis. In the case we describe, doctors detected bradycardia, but no arterial hypertension or irregular breathing.

Headaches that last more than one week after lumbar administration of epidural anaesthesia, stop responding to postural change, or appear with focal neurological signs should alert us to the possibility of an acute intracranial process. Symptoms of such processes no longer reflect CSF hypotension – the typical feature of PDPH – but rather intracranial hypertension, mass effect, and displacement of intracranial structures caused by SH.

Reference

1. Reina M, López A, Benito-León J, Pulido P, María F. Hematoma subdural intracraneal y espinal, una complicación poco común de la anestesia epidural y subaracnoidea. *Rev Esp Anestesiología Reanimación*. 2004;51:28–39.
2. Guenot M. Chronic subdural hematoma: historical studies. *Neurochirurgie*. 2001;47:461–3.
3. Trotter W. Chronic subdural haemorrhage of traumatic origin, and its relation to pachymeningitis hemorrhagica interna. *Br J Surg*. 1914;2:271–91.
4. Machurot PY, Vergnion M, Fraipont V, Bonhomme V, Damas F. Intracranial subdural hematoma following spinal anaesthesia: case report and review literature. *Acta Anaesthesiologica Belgica*. 2010;61:63–6.
5. Amorin JA, Canuto dos Anjos Remígio DS, Damázio Filho O, Guerra de Barros MA, Nicole Carvalho V, Moraes Valença M, et al. Hematoma subdural intracraneal postanestesia subaracnoidea: relato de dos casos y revisión de 33 casos de la literatura. *Rev Bras Anestesiología*. 2010;60:344–9.
6. International Headache Society. International Classification of Headache Disorders (ICHD-II). 2nd ed. Cephalalgia. 2004;24 Suppl. 1:1–160.

7. Peiró CM, Caballer N, Errando CL, Moliner S. Hematoma espinal y cerebral postpunción lumbar diagnóstica con evolución letal. *Rev Esp Anestesiología Reanimación*. 2003;50:481–5.
8. Acharya R, Chhabra SS, Ratra M, Sehgal AD. Cranial subdural haematoma after espinal anaesthesia. *Br J Anaesthesia*. 2001;86:893–5.
9. Reynolds F. Dural puncture and headache: avoid the first but treat the second. *Br Med J*. 1993;306:874–6.
10. Van de Kelft E, Bosmans J, Parizel PM, Van Vyve M, Selosse P. Intracerebral hemorrhage after lumbar myelography with iohexol: report of a case and review of the literature. *Neurosurgery*. 1991;28:570–4.
11. Gamal Hamdan Suleiman MD. Trauma craneoencefálico severo. *Medicrit*. 2005;2:107–48.
12. Jeremitsky E, Omert L, Dunham CM, Protetch J, Rodríguez A. Harbingers of poor outcome the day after severe brain injury: hypothermia, hypoxia and hypoperfusion. *J Trauma*. 2003;54:312–9.
13. Marion DW, Obrist WD, Carlier PM, Penrod LE, Darby JM. The use of moderate hypothermia for patients with severe head injury: a preliminary report. *Neurosurgery*. 1993;79:354–62.
14. Busto R, Dietrich WD, Globus MY, Valdés I, Scheinberg P, Ginsberg MD. Small differences in intrasystemic brain temperature critically determine the extent of ischemic neuronal injury. *J Cereb Blood Flow Metab*. 1987;7:729–38.
15. Gilo Arrojo F, Herrera Muñoz A, Anciones B. Hipertensión intracraneal aguda. *Neurología*. 2010;25 Suppl. 1:3–10.

E.C. López Soler^{a,*}, J.M. Villalba García^b, R. Fernández Madruga^b, M. Fernández Rodrigo^c, M. Mirallas Mezquita^d, P. Mínguez Sorio^d

^a *Medicina Familiar y Comunitaria, Servicio de Urgencias, Hospital Obispo Polanco, Teruel, Spain*

^b *Servicio de Urgencias, Hospital Obispo Polanco, Teruel, Spain*

^c *Medicina Familiar y Comunitaria y Anestesiología y Reanimación, Servicio de Anestesiología y Reanimación, Fundación Hospital de Calahorra, La Rioja, Spain*

^d *Unidad Docente de Medicina Familiar y Comunitaria de Teruel, Teruel, Spain*

* Corresponding author.

E-mail address: emiliocls@yahoo.es (E.C. López Soler).

Opalski syndrome: A variant of lateral-medullary syndrome[☆]

Síndrome de Opalski: una variante del síndrome medular lateral

Dear Editor:

Lateral-medullary syndrome is often found in patients with vertebrobasilar vascular lesions. On rare occasions, the syn-

drome is associated with ipsilateral hemiparesis; this is known as Opalski syndrome.¹

We present the case of a male smoker aged 67 with a history of arterial hypertension, peripheral artery disease, and trigeminal neuralgia. He was undergoing treatment with enalapril, carbamazepine, and baclofen. He was examined following a 12-hour episode of dizziness, nausea, vomiting, and difficulty walking. The initial assessment showed right-sided facial paralysis, mild paresis, dysmetria of the right upper limb, and ataxic gait with lateropulsion. Blood tests (including serum levels of carbamazepine) and cranial CT yielded normal results. The patient was admitted with a diagnosis of cerebral infarct in the vertebrobasilar region which was probably atherothrombotic in origin. Cardioembolic origin was ruled out, and the patient began treatment with antiplatelet drugs. The patient's clinical

[☆] Please cite this article as: Gil Polo C., et al. Síndrome de Opalski: una variante del síndrome medular lateral. *Neurología*. 2013;28:382–4