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#### CASE REPORT

### Intracraneal complications after spinal surgery



J.J. Sierra<sup>a,\*</sup>, M. Malillos<sup>b</sup>

- <sup>a</sup> Cirugía Ortopédica y Traumatología, Hospital San Pedro, Logroño, Spain
- <sup>b</sup> Cirugía Ortopédica y Traumatología, Unidad de Columna, Hospital San Pedro, Logroño, Spain

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#### **KEYWORDS**

Dural tear; Lumbar surgery; Post-surgery complication; Intracranial haemorrhage

# Complicación intracraneal tras cirugía de raquis lumbar

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#### PALABRAS CLAVE

Lesión dural; Cirugía raquis lumbar; Complicación posquirúrgica; Hemorragia intracraneal

Resumen La hemorragia intracraneal es una complicación poco frecuente tras la cirugía de raquis lumbar. Se cree que esta complicación se produce por una caída en la presión intracraneal tras una pérdida de líquido cefalorraquídeo a través de una brecha dural iatrogénica. Presentamos el caso clínico de una paciente que presentó una hemorragia subaracnoidea, un hematoma intraparenquimatoso y un hematoma subdural tras una intervención de estenosis de canal lumbar. No hemos encontrado en la literatura revisada sobre el tema ningún caso que presente tal asociación hemorrágica tras una cirugía de este tipo.

Abstract Intracraneal bleeding is a rare complication after raquis surgery. It is believed to

occur as a drop in the intracraneal pressure after a loss of CSF secondary to an iatrogenic

dural tear. We report a patient who after surgery for lumbar stenosis presented a subarachnoid

haemorrhage, an intraparenchymal haematoma, and a subdural haematoma. To our knowledge,

this is the first report in the literature with such complications after this type of surgery.

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<sup>\*</sup> Corresponding author.

E-mail address: jorgejuansierra@gmail.com (J.J. Sierra).

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#### Objective

The most frequent complications of spinal surgery are local. They include injury to a nerve root, a visceral injury or durotomies that may be associated with the loss of cerebrospinal fluid (CSF). Another complication described as the result of the loss of CSF is intracranial bleeding. We present a case of intracerebral, subdural and subarachnoid bleeding in a patient operated for lumbar stenosis. To date this would be the first published case of the said association between haemorrhaging and lumber surgery.

#### Material and methods

A 67 year-old female patient presented 9/10 analogue visual scale lumbar pain and bilateral radicular pain when resting in the area of L4-L5 accompanied by neurogenic claudication at 10 m. Diagnosis by magnetic resonance imaging was lumbar stenosis at the level of L3-L4, L4-L5 and L5-S1, with grade 1 spondylolisthesis at L4-L5. Following months of conservative treatment that included invasive pain therapy it was decided to perform surgery. A laminectomy was performed at levels L3-L4 and L4-L5, with posterolateral arthrodesis at L3-L4, L4-L5, leaving suction drainage in the subfascial plane. Surgery was carried out apparently without complications, although after the operation the patient had a prolonged waking from the anaesthesia. During her stay in the post-anaesthesia recovery ward the tendency to sleep continued and she only responded to a painful stimulus. 2h after surgery she suffered a generalised grand mal seizure, due to which an urgent cranial CAT was performed. This showed a left temporal intracerebral haemorrhage and 2 foci of apparently subarachnoid bleeding in the right side, one in the occipital zone and the other in the frontal zone (Fig. 1a and b). A vascular study was undertaken by computed tomography angiography to rule out an arterial malformation in the base of the cranium as the cause of the bleeding (Fig. 1c). At this moment the drainage was withdrawn with a loss of approximately 300 cc of haematic appearance.

Due to this complication the patient remained in the intensive care unit for the following days with intracranial pressure monitoring, spontaneous respiration and normal blood pressure. Although she was in a stupor for the first 24h after the operation she gradually recovered her level of consciousness without any further complications. In neurological examination 2 weeks after surgery the patient had recovered her habitual level of consciousness and presented the symptoms of predominantly right tetraparesia and speech alterations. Imaging tests were conducted at this moment, confirming that the frontal focus of haemorrhaging corresponded to a small frontal subdural haematoma with slight pneumocephalus (Fig. 2a). The patient was seen regularly as an outpatient after the first month to continue with postoperative check-ups. At 6 months a new lumbar magnetic resonance imaging study was performed, showing a small pseudomeningocele at the level of the surgery (Fig. 2b and c).

#### Results

The patient is currently undergoing rehabilitation, and the neurological deficits she suffered are evolving favourably. Her lumbar pain has improved and now scores 2/10 on the visual analogue scale. The irradiating pain when resting has disappeared, as has the neurogenic claudication. Except for the neurological complication the patient is satisfied with the result of the surgery.

#### Discussion

Intracranial haemorrhage following lumbar spinal surgery is not a frequent event. In a review Pham et al.¹ found an incidence of 0.4% for complications of this type. Similarly Khalatbari et al.² found 4 cases among 1077 patients. The most widely accepted cause to explain this severe complication is the production of intracranial hypertension as the result of loss of CSF through a dural injury, giving rise to herniation of the cephalic mass and bleeding from vessels due to traction and breakage.³-6 The majority of cases described presented cerebral and subdural bleeding, while intracerebral bleeding was rare. The typical pattern of cerebral bleeding visible in tests has been denominated the zebra sign.⁵-7

No injury to the dura mater was detected in our patient during surgery, although magnetic resonance imaging carried out 6 months after surgery showed the existence of a pseudomeningocele at the level of the operation. This leads to the suspicion that there was accidental durotomy with secondary loss of CSF that went unnoticed during the operation. Accidental durotomies are one of the complications described in lumbar spinal surgery. It is possible that a slight and unnoticed loss of CSF during surgery becomes larger following the positioning of suction drainage, thereby causing significant intracranial hypotension. We believe that this may have occurred in our patient, giving rise to the described complication.

The habitual symptom following intracranial hypotension is intense cephalea which starts 24–48 h after the injury occurs, and which worsens when standing. It is often accompanied by nausea and vomiting. The clinical symptoms are usually self-limiting and cease after a few days of bed rest.

After an episode of intracranial bleeding the clinical signs usually commence swiftly, during the first 10 h, with signs of neurological deficits, <sup>2,6,7</sup> as we saw in our case. In a patient who has been subjected to surgery the symptoms of a neurological deficit may go unnoticed in the initial phase. Instead of these symptoms the patient may display difficulty in waking up from the anaesthetic, cephalea, dizziness and vomiting. In agreement with Kaloostian et al. <sup>8</sup> we believe that the anaesthesia itself together with opiate analgesia, if used in an operation of this type, may cause similar adverse effects. Due to this, special attention must be paid in the differential diagnosis of patients subjected to spinal surgery who display the said symptoms, more so if any injury to the dura mater was noticed during the operation.

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