## Case Report

# Subdural hygroma accompanied by parenchymal and subarachnoid haemorrhage after epidural analgesia in an obstetric patient

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**Background:** Anaesthetists need to know the different causes of persistent headache or a change in level of consciousness following epidural analgesia for labour. Failure to recognise these neurological complications can lead to delayed diagnoses, with subsequent serious implications.

**Methods:** We present a patient who was re-admitted for postural headache resulting from an unrecognised dural puncture during an epidural for pain relief while in labour. During the interview, the patient confirmed drug use (cocaine), so she was evaluated by a psychiatrist with possible post-partum psychosis or drug withdrawal syndrome. Afterwards, the patient deteriorated neurologically, showing impaired consciousness and seizures.

**T**<sub>HE</sub> loss of cerebrospinal fluid (CSF) volume is typically compensated by subdural fluid collections (subdural hygromas and subdural hematomas) and by increase in intracranial venous blood. The occurrence of subdural hygromas is a known complication, but it is unusual in spinal anaesthesia.1-5 Most subdural hygromas resolve when brain expansion or fluid absorption exceeds effusion.<sup>6</sup> However, if a hygroma persists for a long time, its interior is covered with a neomembrane that becomes vascularised and it may cause micro-haemorrhages that turn into a subdural hematoma.<sup>7-9</sup> Furthermore, the caudal displacement of the brain produces the stretching and tearing of intracranial subdural veins.<sup>10</sup> When the expansive force exceeds absorption, as a result of haemorrhaging or continued effusion, it can cause clinical deterioration.

Magnetic resonance imaging (MRI) of hygromas is highly variable, depends on their composition. They are frequently a mixture of blood and CSF; **Results:** The cranial computed tomography showed bilateral frontoparietal subdural collections with intraparenchymal and subarachnoid haemorrhaging. She improved by burr hole drainage of subdural hygroma and a blood patch.

**Conclusions:** Neurological signs should alert the clinician to the possibility of subdural collection and other possible complications such as sinking of the brain in order not to delay the request for imaging tests for diagnoses and effective treatments.

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therefore, it is sometimes difficult to distinguish them from subdural hematomas.

Another consequence of CSF hypovolemia is sinking of the brain, with descent of the cerebellar tonsils and brainstem.

Early recognition of these complications with spinal anaesthesia is important as they are associated with significant morbidity.<sup>11</sup>

We describe a patient with a probable dural puncture after epidural analgesia who was treated for psychiatry, such as post-partum psychosis or drug withdrawal syndrome, who was later diagnosed with a subdural hygroma accompanied by parenchymal and subarachnoid haemorrhaging. We believe this is the first patient of its kind described in the literature.

### Case report

A healthy, 34-year-old nulliparous Czech woman, at gestation week 35, requested epidural analgesia for

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pain relief while in active labour. Her past history included penicillin allergy. Furthermore, the patient posed an important language problem. However, her husband, who spoke Spanish and Czech well, helped us to translate the different aspects about the procedure. The epidural space was identified without complications with the loss of resistance to air technique at the L3-L4 level, with the patient in a sitting position, using an 18-gauge Tuohy epidural needle. Then a 20-gauge epidural catheter was successfully passed 4 cm into epidural space. A test dose of 3 ml of 0.25% bupivacaine with 50 µg of fentanyl, and then a bolus of 10 ml of 0.0625% bupivacaine, were administered and proved to be a satisfactory analgesia. An infusion of 0.0625% bupivacaine with fentanyl 1.5  $\mu$ g/ml at 12 ml/h was then commenced and continued until a healthy baby was delivered, weighing 3.2 kg with 1 and 5 min Apgar scores of 9 and 10. The patient was discharged the following day.

On day 2 after delivery, the patient arrived at the emergency department for postural bifrontal headache, which worsened when she was in an upright position. Therefore, she was diagnosed with probable postdural puncture headache (PDPH). The neurological examination was normal. The patient improved with bed rest, with the intake of fluids orally and caffeine-containing drinks, IV 100 mg every 8 h of hydrocortisone for 24 h, and oral analgesia of 1 g of paracetamol and 30 mg of codeine every 8 h. She was discharged without symptoms after a 3-day hospital stay.

Eight days after the lumbar puncture, the patient was re-admitted for postural fronto-occipital headache, accompanied by nuchal rigidity, apyrexial with a normal blood analysis and neurological examination. This time the patient attended alone, without her husband because he was abroad due to work-related reasons and then the communication was not so fluent. She was re-diagnosed and treated for PDPH and was scheduled for a blood patch the next day. As this day passed, the patient started showing disoriented, confused and excitable behaviour. During the interview, the patient confirmed drug abuse (cocaine), so she was evaluated by a psychiatrist and was diagnosed with possible postpartum psychosis or drug withdrawal syndrome. A toxic analysis was requested, which was positive for opioids. She was treated orally with 5 mg diazepam and 5 mg olanzapine.

The patient was assessed the next day, her general condition worsened. She developed a severe nonpostural headache, nausea, paresthesia of the

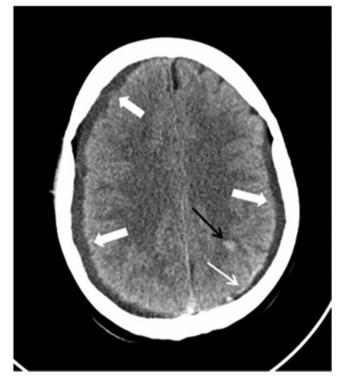
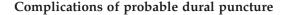


Fig. 1. Cranial computed tomography (CCT). Bilateral hygroma (wide white arrows), and mild intraparenchymal left parietal haemorrhaging (narrow white arrow) with minimal subarachnoid left haemorrhages (black arrow).

left arm and felt drowsy, with a Glasgow Coma Score (GCS) of 13, so a cranial computed tomography (CCT) was urgently performed. Two hours later, the patient deteriorated neurologically with impaired consciousness (GCS, 7), and seizures, proceeding to sedation, intubation and assisted ventilation. The CCT showed bilateral frontoparietal subdural collections and mild intraparenchymal left parietal haemorrhaging with minimal subarachnoid left haemorrhages (Fig. 1). She was admitted to the intensive care unit (ICU) and sedation was reversed. After a poor response to stimuli, surgical intervention was decided. Then, subdural collections were performed, which proved to be hygromas. Subsequently, the patient was taken to the ICU, was agitated and showed less mobility in her left arm after withdrawing sedation. A cranial MRI was performed 48 h after surgery, which ruled out thrombosis of the cerebral venous sinuses, and revealed small bilateral subdural hygromas and a mild intraparenchymal left parietal haemorrhaging with minimal subarachnoid left haemorrhaging (Fig. 2). The patient regained consciousness and was extubated on day 4 post-surgery. However, a blood patch at the L3-L4 level with 20 ml of autologous



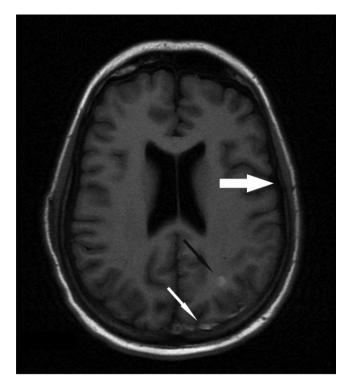


Fig. 2. Cranial magnetic resonance imaging (MRI). Small bilateral subdural hygromas (wide white arrows) and a mild intraparenchymal left parietal haemorrhaging (narrow white arrow) with minimal subarachnoid left haemorrhaging (black arrow).

blood was performed for persistent fronto-occipital headache. Adequate intravenous analgesia and hydration were administered. The next day, the patient was discharged to a ward with normal neurological examination and a mild frontal headache, which was controlled with mild oral analgesics. The patient denied prior history of traumatic events, and her blood pressure remained normal throughout her pregnancy. Seven days after the blood patch, a spinal MRI was performed to check the absence of a CSF leak or intradural and extradural collections. Finally, the patient was asymptomatic, and given the absence of pathological findings in the spinal MRI and neurological focality, she was discharged after a 12-day hospital stay.

Three months after surgery, the patient remains symptom-free, so performing more radiological tests was considered unnecessary.

#### Discussion

Analgesia epidural proves extremely pleasant as it controls labour pains, but PDPH sometimes occurs, resulting in an unsatisfactory situation. This is the reason for research into PDPH by anaesthetists. PDPH symptoms include a postural frontooccipital headache, accompanied by dizziness, nauseas and vomiting, visual disturbances, interscapular pain, nuchal rigidity, photophobia and auditory symptoms.

The prevention and treatment of PDPH is a therapy ladder of bed rest, fluids, caffeine and, if necessary, an epidural blood patch. The autologous blood patch is considered the most effective treatment for PDPH, but in some patients, headaches may persist despite a blood patch.

The incidence of PDPH after dural puncture is 76–85%,<sup>12</sup> but it is not the only cause of headache in pregnancy.<sup>13</sup> Headache in pregnancy also includes pre-eclampsia, migraine, drug-induced headache and intracranial pathologies. Intracranial pathologies include subdural hygromas and hematomas, intracerebral haemorrhaging and venous sinus thrombosis.<sup>1–5,8,9,13–23</sup> In the event of subdural hygromas or hematomas, which act as mass lesions, presentation may range from mild confusion and drowsiness to unstable gait, severe headache and grand mal convulsion.<sup>1–5,8,9</sup>

Subdural hygromas are collections in the subdural space of clear xanthochromic or bloodtinged fluid. This is caused by the creation of a space between the arachnoid and the dura mater, which results in a virtual space.<sup>24</sup> This virtual space is produced when a caudal displacement of the brain parenchyma occurs, thus separating the dura-arachnoid interface and causing an effusion from injured arachnoids tissues or vessels.<sup>7,10</sup> This mechanism explains why the emergence of hygromas may occur in cases of cortical atrophy, alcoholism, dehydration, ventricular tap or fistula dura mater.<sup>24</sup> These fistulas may be caused by accidental dura mater puncture. It is identified by observing the flow of CSF at the hub of the epidural needle, by aspirating CSF from epidural catheter, or by observing the response to a test dose of local anaesthetic.<sup>25</sup> However, between 16% and 33% of accidental dural punctures may remain unrecognised.<sup>26,27</sup> This results in a CSF leak into epidural and subdural spaces, which may lead to the intracranial hypotension syndrome<sup>28</sup> with the compensatory dilatation of the pachymeningeal vessels and increased brain volume.29 Sinking of the brain, and the resultant traction on painsensitive suspending structures of the brain, is thought to be the main cause of the orthostatic headache in CSF leaks. Dilatation of the cerebral veins and venous sinuses may also be a participatory mechanism.<sup>30</sup>

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The most common head MRI abnormalities of CSF leak are subdural fluid collections (hygroma, subdural hematoma), enhancement of the pachymeninges, engorgement of venous structures, pituitary hyperaemia and sagging of brain.<sup>30–32</sup>

Enhancement of the pachymeninges is diffuse, linear, spares the leptomeninges, and involves both the supratentorial and intratentorial compartments. The pathological substrate for enhancement is small thin-walled dilated blood vessels in the subdural zone. Engorgement of venous structures may be difficult to detect on the initial MRI scan, but if it is detected, it usually affects the dural venous sinuses or large cerebral veins.

The sagging of the brain is associated with decrease in size of the ventricles, effacement of perichiasmatic cisterns with bowing of the optic chiasm over the pituitary fossa, effacement of the prepontine cistern with flattening of pons against the clivus and downward displacement of cerebellar tonsils. Sagging of the brain is occasionally attributed to coexisting subdural fluid collections, and unusual manifestations such as encephalopathy and coma have been identified in these patients.<sup>33–35</sup>

Subdural haematomas may be caused by tearing of bridging veins or rupture of the dilated thinwalled blood vessels in the subdural zones. They may be thin and asymptomatic but can be large with enough mass effect to compress the underlying brain. If symptomatic and growing, surgical intervention will become necessary.<sup>32</sup>

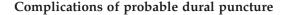
Other rare complications include subarachnoid and intraparenchymal bleeding,<sup>13-17</sup> cortical vein thrombosis<sup>18</sup> or intraspinal puncture combined with cerebral subarachnoid haemorrhaging.<sup>19-23</sup> The emergence of a cranial subarachnoid haemorrhage after a postdural puncture is a rare complication with no clear production mechanisms, which are multifactorial. Its actual incidence is unknown. Some are resolved spontaneously, but others may have a catastrophic result, and may even lead to death. With CCT, it is possible to diagnose 95% of subarachnoid haemorrhages to enable the diagnosis of other underlying problems, such as tumours, intraparenchymal haemorrhaging, arteriovenous malformations and aneurysms.

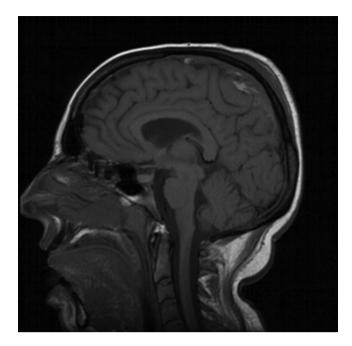
When conservative management fails, the next treatment for PDPH is an epidural blood patch. In our patient, a blood patch was not possible because she was not fasting and she began to become clinically worse within a few hours. The psychiatrist assessed the patient because she confirmed drug use in the anamnesis. The toxic analysis was positive for opioids because she was being treated with codeine, but illicit drugs were ruled out in our patient. Moreover, the patient was evaluated by a psychiatrist with possible post-partum psychosis. She was treated with diazepam and olanzapine, but no clinical changes were observed. We believe the patient did not properly understand due to language problems, which made the diagnosis difficult and delayed doing the imaging tests.

Initially, the headache was typical of a PDPH, but it became a severe constant headache with time, including blunting of consciousness, psychiatric symptoms, focal deficit, slurred speech and drowsiness. The patient underwent surgery for neurologic deterioration with a suspected subdural hematoma diagnosis by the CCT, which proved to be subdural hygromas. After the burr-hole drainage of hygromas, the mass effect on the brain improved in the cranial MRI. The symptoms disappeared completely, with the exception of persistent fronto-occipital headache, so a blood patch was performed to seal a possible CSF leak. Finally, the patient remained symptom-free. The epidural blood patch results in a pressure effect with the cranial displacement of the CSF and a plugging effect, with a success rate of over 75% for post-spinal headache.<sup>36</sup> Upon her first admission, the patient complained of a typical PDPH, but she improved after 48 h. Upon the second admission, the patient developed an atypical headache with abnormal neurology, so a differential diagnosis and early neurological imaging were necessary to establish an early diagnosis.

Treatment of subdural fluid collections can include a conservative treatment consisting of analgesics, corticoids and a blood patch, but sometimes the mass effect with neurological impairment requires a rapid diagnosis with CCT or cranial MRI and surgical evacuation. Initially, we suspected our patient suffered a pontine compression due to the brain sagging towards the foramen magnum with compression of the pons. This fact could explain the rapid deterioration of consciousness. However, it was not identified in midline sagittal image of the MRI scan performed 48 h after surgery (Fig. 3). Neither CCT performed before surgery could demonstrate cerebellar tonsillar herniation or ventricular collapse. The patient clearly improved after surgical evacuation of hygromas. The symptoms disappeared completely, with the exception of persistent fronto-occipital headache.

Furthermore, given done that the patient presented seizures, an alternative diagnosis might be a non-convulsive status epilepticus, but a electroen-





*Fig. 3. Midline sagittal image of the magnetic resonance imaging (MRI) scan performed 48 h after surgery. Note the enhancement of the pachymeninges.* 

cephalogram was not performed so there was no indication for anticonvulsant therapy.

After considering all possible causes of poor neurological progress made by our patient, we think it was due to the mass effect caused subdural hygromas, despite that they were not large enough to produce the deterioration presented.

Throughout her pregnancy, normal arterial pressure, and neither oedema nor proteinuria was detected. An infusion of oxytocin of 10 milliunit/ min was required to augment labour. During labour, she was monitored and no high arterial pressure was recorded. Infrequent intracranial subarachnoid haemorrhages after lumbar puncture are often associated with the breaking of intracranial aneurysms, arteriovenous malformations or vessel damage as a result of low intracranial pressure and traction of the brain in relation to arachnoid or perianaesthesia stress, whose pathogenesis is unclear.<sup>13–23</sup> Although no arteriovenous malformations or aneurysms were found in the CCT or cranial MRI, the presence or absence of any vascular abnormalities cannot be confirmed because cerebral arteriography was not performed. A thrombosis of the venous sinuses can be secondary to vascular injury by the pressure changes in the CSF due to stretching vessels and stasis blood flow in a vasodilated cerebral vasculature. After thrombosis, venous return is stopped and may produce cerebral venous haemorrhaging. However, thrombosis in the cerebral venous sinuses was ruled out in our patient. Intraparenchymal haemorrhages after postdural puncture are very rare. They are usually spontaneous or are due to the rupture of microaneurysms caused by high blood pressure. The Valsalva manoeuvre performed during vaginal delivery and uterine contractions cause a sharp transient increase in blood pressure and in CSF pressure. The cause is unknown in 40–50% of cases, suggesting abrupt or transient episodes of high blood pressure.

Very few reports describe intracranial haemorrhages after dural puncture and most are subdural hematomas. It was not possible to confirm if the subarachnoid and intraparenchymal haemorrhages were complications of postdural puncture by CSF hypotension, isolated hypertension, perianaesthesia stress, or if they were already existed.<sup>13–23</sup>

Fortunately, the subarachnoid and intraparenchymal haemorrhaging in our patient was mild and did not require surgical decompression. Intracranial haemorrhages after a spinal puncture are rare complications, but may complicate patient's evolution. As to the frequency of these complications, subdural hematoma is seen to be more prevalent, followed by intraparenchyimal haemorrhage and finally by subarachnoid haemorrhage.<sup>22</sup>

Between 16% and 33% of accidental dural punctures can go unrecognised.<sup>26,27</sup> Some theories defend that the needle damages the dura superficially, so it may tear later,<sup>26</sup> while others propose the presence of a tissue plug in the needle that hinders a CSF leak.<sup>37</sup> In any case, it is essential to detect them as soon as possible. Failure to recognise an accidental dural puncture at the time of epidural insertion can lead to a delayed diagnosis with the subsequent serious consequences, such as pituitary damage, hematoma within the tentorium, traction on cranial nerves, subdural hygroma, subdural hematoma and rare events such as intraparenchymal and subarachnoid haemorrhaging. Therefore, anaesthetists should include them in the differential diagnosis of unrecognised PDPH in the puerperium.

Furthermore, we believe that a complete understanding of the language is essential when dealing with women who need epidural analgesia because it is necessary to communicate with them while placing the epidural to detect adverse events during epidural analgesia and to not delay the diagnosis of possible neurological complications. We attempted to contact a translator, but it was not possible.

In conclusion, this case emphasises that clinicians should be aware of these neurological complications after persistent and severe headaches with unusual symptoms following epidural analgesia. Neurological signs such a persistent headache or a change in level of consciousness should alert the clinician to the possibility of subdural collection and other possible complications such as sinking of the brain in order not to delay the request for imaging tests for diagnoses and effective treatments.

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