Bilateral interhemispheric subdural hematoma after inadvertent lumbar puncture in a parturient

Hématome sous-dural inter-hémisphérique bilatéral après ponction lombaire accidentelle chez une parturiente

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Abstract

Purpose Interhemispheric subdural hematomas (ISH) are rare in adults and occur most often after cranial trauma. We describe a parturient who developed bilateral acute ISH after inadvertent dural puncture associated with placement of an epidural catheter for labour analgesia. We discuss the features, pathophysiology, and management of this type of subdural hematoma.

Clinical features A 38-yr-old woman requested epidural analgesia for relief of labour pain. An inadvertent dural puncture occurred during placement of a 17G Tuohy needle. After labour and delivery, the patient developed symptoms of a postdural puncture headache, which responded only partially to an epidural blood patch. The patient's headache subsequently became less position-dependent and was associated with episodes of sharp pain radiating down her legs and paresthesias on the left side of her body. A computed tomography (CT) scan showed right frontal and left parietal acute ISH without an intracranial mass effect. The patient was monitored in the intensive care unit and treated conservatively because of the relatively

small size of the ISH and the absence of progressive neurological deficits on serial examinations. Daily CT scans showed gradual decreases in the size of the ISH concomitant with improvement of the headache.

Conclusions Rupture of bridging veins between the cerebral cortex and the superior sagittal sinus is the usual mechanism by which ISH occur. Nearly one-quarter of patients with ISH do not survive, although those with smaller hematomas have a better outcome. If the hematoma is < 1 cm in thickness, a conservative approach to ISH is recommended in the absence of mental status changes, seizure activity, or focal deficits, but with larger ISH or evidence of progressive neurological deterioration, surgical evacuation is most often required to prevent mortality.

Résumé

Objectif Les hématomes sous-duraux inter-hémisphériques (ISH) sont rares chez l'adulte et surviennent le plus souvent après un traumatisme crânien. Nous décrivons le cas d'une parturiente qui a présenté un hématome sous-dural aigu bilatéral après ponction durale accidentelle en rapport avec la mise en place d'un cathéter péridural pour analgésie au cours du travail. Nous discutons les caractéristiques, la physiopathologie et la prise en charge de ce type d'hématome sous-dural.

Caractéristiques cliniques Une femme âgée de 38 ans a demandé à bénéficier d'une analgésie péridurale pendant le travail. Il y a eu une ponction accidentelle de la dure-mère pendant la mise en place d'une aiguille de Tuohy 17G. Après le travail et l'accouchement, la patiente a présenté des symptômes de céphalée post ponction dure-mérienne qui n'ont répondu que partiellement à un colmatage sanguin (blood patch) péridural. La céphalée de la patiente est devenue ensuite moins liée à la position, mais elle a été associée à des épisodes de douleurs aiguës

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irradiant dans ses membres inférieurs et à des paresthésies du côté gauche du corps. Une tomодensitométrie a mis en évidence un ISH aigu frontal et pariétal gauche sans effet de masse intracrânienne. La patiente a été suivie en unité de soins intensifs et traitée de façon conservatrice en raison de la relative petite taille de l'ISH et de l'absence de progression des déficits neurologiques au cours des examens répétés. Les tomодensitométries quotidiennes ont montré la diminution graduelle de la taille de l'ISH parallèlement à l'amélioration de la céphalée.

Conclusions *La rupture des veines pontantes entre le cortex cérébral et le sinus sagittal supérieur constitue le mécanisme habituel provoquant la survenue d'un ISH. Près d'un quart des patients présentant un ISH ne survit pas, bien que le pronostic des patients ayant de plus petits hématomes soit meilleur. Si l'hématome a une épaisseur < 1 cm, une approche conservatrice de l'ISH est recommandée en l'absence de modification de l'état mental, d'activité convulsive ou de déficits localisés; en revanche, en cas d'ISH plus important ou de signes de détérioration neurologique progressive, une évacuation chirurgicale est le plus souvent nécessaire pour éviter le décès du patient.*

Intracranial subdural hematoma, an unusual¹ but well-known and potentially life-threatening complication of dural puncture, is associated with leak of cerebrospinal fluid (CSF) and reduced CSF pressure.² Leak of CSF contributes to traction and downward displacement of the brain within the cranium, thereby causing bridging cerebral veins to rupture and producing a subdural hematoma.³ A review of 47 cases emphasized that subdural hematoma after dural puncture carries a substantial risk of morbidity and mortality despite neurosurgical intervention.⁴ When postdural puncture headache (PDPH) fails to respond to conventional treatment, anesthesiologists are most often prompted to obtain the neuroradiology studies [e.g., computed tomography (CT), magnetic resonance imaging (MRI), cerebral angiography] needed to establish the diagnosis of subdural hematoma.⁵ In this report, we describe a parturient who developed bilateral acute interhemispheric subdural hematomas (ISH) after inadvertent dural puncture associated with placement of an epidural catheter for labour analgesia. Bilateral ISH are rare in adults,⁶ as less than ten cases have been described in the peer-reviewed literature to date, most often after cranial trauma.⁷ The patient provided written informed consent for the publication of this report.

Case report

A healthy 38-yr-old woman (weight, 83 kg; height, 180 cm; G1P0) in active labour at term requested epidural

analgesia for relief of labour pain. An inadvertent dural puncture occurred during epidural placement of a 17G Tuohy needle at the L3-4 interspace using the loss of resistance to air technique. The needle was removed and repositioned at the L2-3 interspace, and a catheter was successfully threaded 4.5 cm into the epidural space at this level. A test dose of 1.5% lidocaine and 1:200,000 epinephrine 3 mL was administered to confirm correct positioning of the catheter, and excellent analgesia was then obtained using an epidural bolus (12 mL in three divided doses over 15 min) of a solution of 0.25% bupivacaine and fentanyl 100 µg. Analgesia was subsequently maintained throughout labour using a continuous infusion (10 mL·hr⁻¹) of 0.125% bupivacaine with fentanyl 1 µg·mL⁻¹. The catheter was removed shortly after normal spontaneous vaginal delivery of a healthy male infant. The duration of labour was approximately 15 hr; the second stage of labour was not prolonged.

The patient's recovery was uneventful until the second postpartum day when she developed a severe postdural bilateral frontal-occipital headache accompanied by nausea and vomiting. She was treated conservatively with bed rest in a recumbent position, administration of intravenous fluids, and oral analgesics (acetaminophen, ibuprofen, and hydrocodone), but her headache failed to respond to these initial interventions. An epidural blood patch was performed using a 17G Tuohy needle in the L4-5 interspace (one level below the dural puncture). Loss of resistance to saline was used to identify the epidural space, and 16 mL of autologous blood was injected. The patient's symptoms markedly improved but did not completely resolve after the epidural blood patch. The patient was instructed to remain in a recumbent position and caffeinated beverages were encouraged. Her headache gradually improved over the next 24 hr, but she continued to complain of mild to moderate pain which she now described as "pressure in the neck and head". A physical examination showed the presence of modest nuchal rigidity, which was attributed to "muscle spasm", and as a result, she was treated with cyclobenzaprine 5 mg *per os* tid. The patient was released from the hospital with minimal headache and instructed to contact the anesthesiology team if her symptoms persisted.

Two days after discharge, the patient returned to the hospital. She stated that her headache had worsened. The headache was partially improved but not abolished when she assumed a recumbent position. Nausea, vomiting, flushing, and myodesopsia accompanied her headache. She also described several brief episodes of sharp pain radiating down either her right or left leg and reported transient paresthesias on the left side of her body. She continued to complain of neck stiffness and reported "fullness" in her ears, but she denied fever, visual field deficits, vertigo, bulbar symptoms, seizures, focal neurological deficits, and

bowel or bladder incontinence. A neurological examination revealed no sensory and motor deficits; cranial nerves were fully intact. The patient's temperature on admission was 36.9°C and her white blood cell count was 10.2 (10^3 cells·mL⁻¹). A CT scan without angiographic contrast showed right frontal (Fig. 1) and left parietal (Fig. 2) ISH which were 0.33 and 0.49 cm in maximum diameter, respectively. There was no evidence of an intracranial mass

Fig. 1 Axial computed tomography image without angiographic contrast showing right frontal interhemispheric subdural hematoma

Fig. 2 Axial computed tomography image without angiographic contrast showing left parietal interhemispheric subdural hematoma

Fig. 3 Axial computed tomography image without angiographic contrast obtained one month after admission showing complete resolution of the bilateral interhemispheric subdural hematomas

effect. A lumbar MRI was normal. The patient was admitted to the intensive care unit after consultation with a neurosurgeon and maintained in a recumbent position. The patient was treated conservatively because of the relatively small size of the bilateral ISH and the absence of neurological deficits. Her headache pain was treated using acetaminophen and ibuprofen. Complete neurological examinations conducted at regular intervals remained unremarkable. Serial CT scans were performed over the following three days and showed gradual decreases in the size of the ISH, which occurred in conjunction with improvement of the patient's headache symptoms. The remainder of the patient's hospital course was unremarkable, and she was discharged in satisfactory condition with complete resolution of the headache on the ninth post-partum day. A follow-up CT scan performed one month later showed complete resolution of the ISH (Fig. 3).

Discussion

In 1940, Aring and Evans first reported ISH as a distinct type of subdural hematoma.⁸ Interhemispheric subdural hematoma is usually unilateral⁹ and is sometimes described in cases of child abuse ("shaken baby syndrome"),¹⁰ but this form of subdural hematoma rarely occurs in adults.⁶ Cranial trauma is responsible for the majority of adult cases of ISH,¹¹ but cerebral aneurysm rupture may also cause ISH.¹² Intrinsic coagulopathy, anticoagulant therapy, and

alcohol abuse have been identified as predisposing factors for ISH.¹³ Traumatic rupture of bridging veins between the cerebral cortex and the superior sagittal sinus is the usual mechanism by which ISH occur.^{6,14} Tearing of cerebral bridging veins has also been implicated as the putative mechanism of injury in the development of other forms of subdural hematoma after dural puncture and reduced CSF pressure^{2,15}; it is highly likely that the ISH observed in our patient may have occurred through such a mechanism involving the superior sagittal sinus. It is plausible that the epidural blood patch performed for the initial presumptive diagnosis of PDPH may have limited the development and facilitated partial resolution of the ISH by inhibiting further CSF leak from the dural rent. Interhemispheric subdural hematomas are confined to the falx cerebri and the adjoining cerebellar tentorium because the arachnoid trabeculae hold the brain and parasagittal dura in close proximity. This anatomic confinement prevents the ISH from spreading to the ipsilateral subdural space along the cranial convexity.¹⁶ Nearly one-quarter of patients with ISH do not survive,^{6,14} although those with smaller hematomas, like our patient, generally have a better outcome (e.g., mortality rate of 10.5% for conservative management vs 31.7% with neurosurgical intervention).⁶ Glasgow coma scale score, hypovolemic shock, and the presence of a skull fracture or concomitant subarachnoid hemorrhage are correlated with poor outcome in patients with traumatic ISH,¹⁷ but our patient denied a history of head injury and clearly did not have any of these risk factors for mortality. Despite previous data to the contrary, the mortality rates of patients with ISH may be similar to those of patients with other forms of acute subdural hematoma when adjusted for neurological status.¹⁸ Nevertheless, Bartels *et al.* reported that a large majority (69%) of cases of ISH included in their review required operative intervention (e.g., craniotomy, burr holes),⁶ contrary to other forms of subdural hematoma.¹⁹

The clinical manifestations of ISH depend on the specific location and size of the hematoma and the progression of the neurological symptoms that it produces.¹³ In our patient, the clinical picture was assumed to be PDPH because of the temporal relationship between the appearance of the patient's symptoms and the onset of a position-dependent headache after accidental dural puncture. However, our patient's initial symptoms did not respond completely to conservative modalities and an epidural blood patch, and subsequently, the character of her headache changed. These findings suggested that PDPH was not the only cause of her headache, and this prompted us to investigate other potential causes. Migraine, cluster, tension, and caffeine withdrawal headaches and new subarachnoid hemorrhage were excluded on the basis of the patient's history and physical examination. The patient

did not have preeclampsia. Meningitis was considered, at least in part, because of the development of nuchal rigidity, but this diagnosis was excluded because the patient was afebrile and her white blood cell count was normal. Superior sagittal sinus thrombophlebitis was also part of the differential diagnosis, but its CT appearance ("cord sign") and associated findings (e.g., venous ischemia, cerebral edema)²⁰ were not observed in our patient. Indeed, the segmental lenticular appearance of hematomas along the falx cerebri was entirely consistent with the diagnosis of ISH.⁹ Before the advent of cranial neuroimaging, ISH were suspected when a patient presented with acute mental status changes (e.g., lethargy, confusion) and "falx syndrome" (contralateral leg weakness or crural hemiparesis) occurring hours to days after head trauma.^{21,22} Altered level of consciousness, hemiparesis, contralateral leg monoparesis, and seizures were the most frequent presenting signs and symptoms of ISH.⁶ Ataxia, cognitive dysfunction, language abnormalities, and oculomotor nerve dysfunction were less frequently described.⁶ Our patient reported episodes of transient brief leg pain, paresthesias, neck stiffness, and persistent headache, but she denied other symptoms of ISH. This presentation of ISH is somewhat atypical, but it may have occurred because the patient's subdural hematomas were small. Isolated headache was previously reported as the only complaint in an elderly woman with a small ISH,²³ but a dural puncture had not been performed in this patient before she developed symptoms. We cannot completely exclude the possibility that our patient had intracranial pathology before she was admitted to our hospital for labour and delivery, but this possibility appears highly unlikely considering she was fully functional and did not have headaches or neurological complaints at any time before or during her pregnancy.

Computed tomography imaging is considered the gold standard for the diagnosis of ISH and, as observed in our patient, usually demonstrates ISH as a hyperdense fluid collection along the interhemispheric fissure.⁹ A cranial CT scan without angiographic contrast may be unreliable seven to 21 days after all forms of subdural hematoma (including ISH) because the hematoma and surrounding brain tissue have similar radiographic density ("isodense" phase),^{3,5} but we did not encounter this potential cause of a false negative result with our patient. Under these circumstances, MRI or cerebral angiography are useful neuroimaging techniques to establish the diagnosis.⁹ A conservative approach to ISH has been recommended in a patient without mental status changes, seizure activity, or focal neurologic deficits when the hematoma is < 1 cm in maximum thickness and a significant intracranial mass effect is absent.^{6,7} The larger of the two ISH in our patient had a maximum thickness of < 0.5 cm, and she did not have evidence of a midline shift on her initial CT scan.

As a result, she was carefully monitored for the development of new neurological symptoms in the intensive care unit, but evacuation of the ISH²⁴ proved to be unnecessary because her headache improved spontaneously concomitant with gradual resolution of the hematomas.

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