

Bilateral Subdural Effusions and Brainstem Sagging Following Labor Epidural—Case Report and Literature Review

Abstract

Epidural analgesia is a technique commonly used in labor. Intracranial subdural hygromas are a rare complication of neuraxial techniques and have only been reported in a handful of patients. We report a case of a female with postpartum headache, presenting 5 days after delivery where epidural analgesia was used, who was found to have bilateral subdural effusions and brainstem sagging. Our literature review summarizes pathophysiology, imaging, and approach to treatment, as well as guidance on long-term outcomes and follow-up needed in this patient population.

Keywords: *Dural puncture, epidural analgesia, post-dural puncture headache, subdural effusion, subdural hygroma*

Introduction

Intracranial subdural hygromas are a rare complication of dural breach with six obstetric cases documented to date. The suspected pathophysiology suggests that in the context of a cerebrospinal fluid (CSF) leak, the brain loses buoyancy and sags toward foramen magnum. As a compensatory mechanism, bridging veins dilate with formation of subdural collections, mainly hygromas.^[1]

We present a case where unrecognized dural puncture resulted in bilateral subdural effusions and sagging of brainstem, with spontaneous resolution of symptoms. Literature review of reported cases of subdural hygromas following labor epidural is summarized. Pathophysiology, imaging, and approach to treatment of this complication are discussed.

Case History

A 25-year-old nulliparous female at 36 + 6 weeks estimated gestational age presented with spontaneous rupture of membranes. Her past medical history was notable for anxiety with tendency for panic attacks, small ventricular septal defect (VSD), with otherwise normal ECHO. She had an early labor epidural catheter placed using 16G Touhy needle

after two attempts, both in the same interspace. No CSF was observed during the procedure. Her labor was augmented with oxytocin, and after 12 h Neville Barnes forceps delivery was performed, to shorten the second stage. This was complicated by postpartum hemorrhage. Blood loss was calculated by weighing of all swabs, as per OBS Cymru guidelines.^[2] Measured loss totalled at 1,655 ml and required a blood transfusion. The patient was discharged the following day, but returned 5 days later, with 24 h history of positional headache described as pressure and pulsation, neck stiffness, and one episode of vomiting. Neurological examination was normal. She was found to be newly hypertensive 158/105 with no previous history of raised blood pressure or pre-eclampsia. She was started on labetalol 200 mg three times daily. Computed tomography (CT) scan of her head was performed to rule out sinus vein thrombosis. Figures 1 and 2 demonstrate bilateral thin supra and infratentorial subdural effusions with features suspicious for sagging of brainstem. CT venogram showed no thrombosis. The case was discussed with regional neurosurgical centre and routine post dural puncture headache (PDPH) management with inpatient observation was advised. The patient received oral rehydration, paracetamol, codeine, and caffeine. Her symptoms resolved spontaneously

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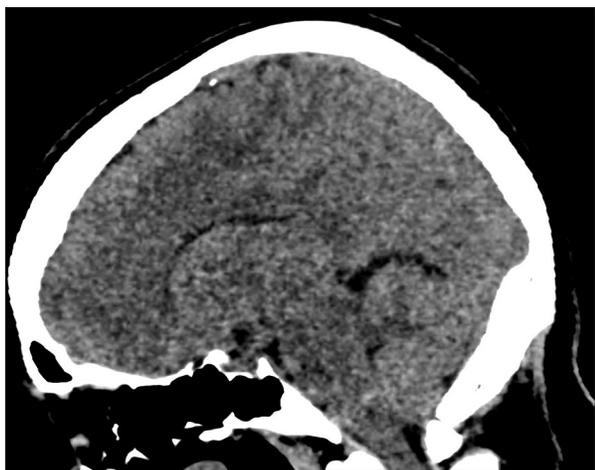


Figure 1: CT head (sagittal plane) showing sagging of brain

overnight, and she was discharged from the hospital on day eight postpartum with no further readmissions. Follow-up magnetic resonance imaging (MRI) brain a month later showed complete resolution of intracranial changes.

Discussion

Unintentional dural puncture in obstetrics has an incidence of around 1.5% labor epidurals. PDPH ensues in approximately half of the cases. Symptoms can occur between 1 to 7 days, most commonly within 72 h.^[3] In our case, the dural breach was not recognized at the time of the procedure, which is a common occurrence in clinical practice, as 38% of dural tears are not apparent at the time of the procedure.^[4]

Dural puncture allows CSF to leak from the subarachnoid space and consequently CSF pressure falls. Two pathophysiological mechanisms are disputed for the origin of the headache - adenosine receptor activation in a low CSF volume state and traction on pain sensitive structures such as cranial nerves, sinuses, and meninges.^[5] According to the Monroe-Kellie hypothesis, the total volume of cerebral tissue, intracranial blood, and CSF remains constant. In support of this theory, in intracranial hypotension, compensatory dilatation of the venous structures and increase in brain volume is observed.^[2] Subdural fluid collections are observed in 50% of patients with spontaneous intracranial hypotension.^[6] Gravitational pull of caudally displaced brain can result in tearing of bridging veins in the subdural space resulting in subdural haematomas. This is likely a less common complication of dural puncture, however because of its potential severity is more widely reported.^[7] It can be argued that subdural hygromas and hematomas in this context represent a spectrum of symptoms exhibited depending on severity of the leak, and the former can progress into the latter^[8] if CSF loss exceeds daily production and remains untreated.

The incidence of intracranial changes following dural puncture cannot be confirmed, as most patients are managed



Figure 2: CT head (coronal plane) showing bilateral subdural effusions

without imaging. Summary of the the six obstetric cases identified through literature review is included in Table 1. CT images obtained at this stage can show a subdural collection, either hygromas or haematomas. As CT alone cannot reliably differentiate between the two, brain MRI can be of benefit to provide more accurate diagnosis.^[8] Classic images of intracranial hypotension on MRI include pachymeningeal enhancement, descent of the brain (sagging or sinking), subdural fluid collections (hygromas, rarely hematomas), enlargement of the pituitary gland, enlarged cerebral venous sinuses and ventricular collapse.^[2]

Majority of subdural hygromas are asymptomatic and resolve without treatment.^[8] Initial treatment follows the same principles as that of PDPH management and includes rehydration, analgesia, and careful caffeine administration. Novel treatments such as triptans, gabapentinoids, ACTH and analogues or steroids do not currently have sufficient evidence to be supported.^[9] When conservative therapy fails, epidural blood patch can be offered as the next step. Injection of autologous blood in the epidural space is believed to relieve intracranial hypotension symptoms through cranial displacement of the CSF and promoting sealing of the leak. This approach is successful in relieving symptoms in between 50 and 95% of cases of PDPH. Epidural fluid administration was performed in all cases in our literature review, with majority being blood patches. One case reports successful use dextran 40 as an alternative in a patient with concurrent infection.^[10] Surgical treatment is rarely necessary with simple PDPH. However, one patient in this review underwent surgical evacuation because of rapidly deteriorating consciousness.^[11]

As hygromas are likely spectrum of symptoms exhibited in intracranial hypotension, progression to subdural hematoma, and serious morbidity accompanied with it, is possible.^[7] To date however, there are no reports of serious long-term morbidity or mortality following an intracranial hygroma associated with labor epidural. All reviewed cases showed complete resolution of intracranial changes.

Table 1: Literature review

Author	Recognised ADP	Time to symptoms *	Symptoms	Brain imaging	Treatment	Long term outcome	Follow-up imaging
Thompson (2018)	No	d2	headache (d2), nuchal rigidity (d3) photophobia (d3)	MRI – bilateral cerebral hemispheric shallow hygromas, aqueduct descent, also extra-axial hygroma collections from foramen magnum to C6, possibly extending to lumbar region	EBP (d4)	Complete resolution	Yes, MRI normal
Vien (2016)	Yes	d1	headache (d1) vomiting (d2) bradycardia (d3)	CT - bilateral cerebral convexity subdural hygromas, small right frontal subdural hematoma MRI - slit-like lateral ventricles, an enlarged pituitary gland, and aseptic pachymeningitis	EBP (d4),	Complete resolution	Yes, MRI normal
Del-Rio-Velossilo (2014)	No	d2	headache (d2) nuchal rigidity (d8) disorientation (d8) nausea (d9) paraesthesia (d9) seizure and GCS 7 (d9)	CT - frontoparietal subdural collections, mild intraparenchymal left parietal haemorrhaging with minimal subarachnoid left haemorrhages MRI (after surgical treatment) - small bilateral subdural hygromas	hydrocortisone, caffeine (d2) surgical evacuation (d9) EBP (d13)	Complete resolution	No
Sinha (2010)	No	d2	headache (d2) nausea (d9) neck rigidity (d9)	CT - left subdural collection, initially reported as a chronic subdural hematoma MRI - large subdural hygroma, marked meningeal enhancement, small cortical vein thrombosis	caffeine (d9) EBP (d9)	Complete resolution	No
Aragones (2004)	Yes? subarachnoid catheter	d2	Headache (d2), tinnitus (d10), unstable gait (d10)	CT - bifrontal subdural hygromas MRI (after blood patch) - bifrontal hygromas, diffuse pachymeningeal enhancement	dexamethasone and caffeine (d2) Epidural patch with dextran 40 (d10)	Complete resolution	Yes, MRI reduced hygromas
Verdu (1999)	Yes	d0	headache (d0) nuchal rigidity (d15)	CT - bilateral frontoparietal subdural hygromas, with gyral effacement and small ventricles	EBP (d15 and d27)	Complete resolution	Yes, CT normal

*d0 is the day of the procedure, d1 is day 1 after epidural ADP – accidental dural puncture EBP – epidural blood patch CT – computed tomography MRI – magnetic resonance imaging

Conclusions

Subdural hygromas represent a collection of fluid located under the dural membrane of the brain. They are likely a common consequence of unintentional dural puncture and symptoms in majority do not differ from those of PDPH. Hygromas can progress into subdural haematomas, but majority resolve after treatment with a blood patch. Follow-up images show improvement or complete resolution of changes in all cases reviewed. This suggests that routine follow-up imaging might not be necessary in these patients.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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