Two young women with chronic daily headache and cognitive impairment: why we need to ask about headache in the postpartum period

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Summary: Headache, which has a variety of causes, is a common and disabling complaint following childbirth. An important aetiology not to be missed is headache from epidural spinal anaesthesia, known as postdural puncture headache (PDPH), which has been reported in upwards of 85% of pregnant women and is a manifestation of intracranial hypotension from leakage of cerebrospinal (CSF) fluid through a dural tear. The common presenting symptom of PDPH is head pain occurring when a patient is in an upright position that resolves with recumbency. Other neurological issues associated with intracranial hypotension can include cranial nerve palsies, encephalopathy and Parkinsonism. We present two cases of persistent PDPH after pregnancy with secondary cognitive impairment. A review of the clinical manifestations, neuroimaging findings and treatment for PDPH will be presented. Better recognition of this disorder by obstetricians, physicians and anaesthetists will help to reduce the considerable morbidity this syndrome can produce in young mothers.

Keywords: intracranial hypotension, chronic daily headache, headache, CSF leak, epidural

INTRODUCTION

Headache can be a common postpartum complaint which has a broad differential diagnosis and requires careful investigation especially in individuals with minimal or no prior headache history before pregnancy. Potential aetiologies of new onset daily headache in the postpartum period include postdural puncture headache (PDPH) after spinal epidural anaesthesia, cerebral venous sinus thrombosis, reversible cerebral vasospasm syndrome and pre-eclampsia.¹ Migraine can also start or exacerbate during this time period and go on for days at a time because of the presumed shifts in oestrogen levels after childbirth. Because of the multiple potential aetiologies and complications that may evolve, it is important to ask each woman following delivery and at their first postpartum visit if headache is present, and its manner of presentation. We will focus on PDPH which is a common iatrogenic and sometimes incapacitating complication following spinal anaesthesia for childbirth.

The most common clinical manifestation of PDPH is headache in an upright position that is relieved by recumbency (positional headache).² The brain is kept buoyant by spinal fluid volume, but in a state of persistent leakage of CSF from a dural tear, this results in intracranial hypovolaemia or hypotension. Thus when a patient is upright the brain will sag into the posterior fossa and cause headache and other neurological

symptoms. In the supine position and without gravity, head pain and associated symptoms are significantly lessened. In most instances, even without intervention, this form of secondary headache will alleviate on its own but sometimes if unrecognized and untreated this form of 'treatable' chronic daily headache can progress unmitigated for years with a worsening neurological status in addition to chronic head pain. We present two cases of chronic PDPH that presented not only with daily head pain for over three years duration but also progressive memory loss. Both patients were severely disabled by their condition and in both cases early recognition of a postepidural induced state of intracranial hypotension would have dramatically altered their experience as a mother of a young baby.

CASE 1

A 25-year-old woman presented to a headache specialty clinic with a complaint of a daily headache that began out of the blue three years prior following childbirth. She had a history of episodic migraine without aura since her teens occurring approximately once per month. This new headache seemed to start immediately following epidural anaesthesia for her delivery and in addition to head pain she noticed an acute decrease in hearing. She was not able to maintain an upright position without a worsening of her headache. The pain was primarily located in her right temple but she also complained of bifrontal and occipital-nuchal based pain which she described as pressure and throbbing in quality. Her headache was not acknowledged as an inpatient; however following hospital discharge the

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patient reported her new symptoms to the obstetrician's office at which time she was informed 'headache is a common complication after an epidural and to manage it with bed rest'. Eventually her headache became present even when supine but the pain level would always intensify as soon as she moved from a lying to a sitting or standing position. The pain intensity level would always worsen as the day progressed. She noted that if she was able to lie down, her level of pain would decrease and the best time of day for her head pain was first thing in the morning before she would arise. In addition to the headache, she developed nausea, intermittent blurred vision, photophobia and phonophobia when erect which would resolve with recumbency. Her decreased hearing post-procedure was not altered by position. Apart from the head pain and maybe even more disturbing to the patient was worsening cognition with short-term memory loss. The patient described the inability to comprehend and recall important information she had been told only moments or days prior. She also admitted she could not remember how to complete tasks she had long known how to accomplish, such as computer work at her job. She had tried multiple over-the-counter analgesics and vitamin therapies without any improvement, otherwise her headaches had not been medically managed and the patient did not pursue further treatment. Her general and neurological exam was essentially non-focal. A routine mini mental status exam was normal however neuropsychometric testing revealed abnormalities in functions associated with the dominant frontal lobe. In the office she was placed in the Trendelenberg position (a test developed by author TDR to help determine if a patient with chronic headache may indeed have intracranial hypotension³) at approximately 15° head down tilt and noted resolution of her headache. Once she was returned to the supine position, the headache quickly returned followed by worsening severity with upright position. A presumed diagnosis of PDPH was made.

Magnetic resonance imaging (MRI) with and without gadolinium was reported as normal by neuroradiology but appeared to show slight pachymeningeal enhancement (PME). The patient's symptomatology was consistent with intracranial hypotension and her imaging was possibly suggestive of the diagnosis so the decision was made to treat the patient with an epidural blood patch with a presumed persistent dural puncture tear at the site of her lumbar epidural. After the blood patch the patient initially noted marked improvement in her headache. Her baseline pain resolved and she had minimal headache when upright. However over several days she developed a worsening headache while supine from re-bound intracranial hypertension requiring a short treatment course with a diuretic. This headache resolved and now she is basically headache-free with intermittent migraines. Her memory issues have improved but she is still not yet back to baseline.

CASE 2

A 31-year-old woman with no prior headache history presented to a headache specialty clinic for evaluation of a new headache that began daily 1.5 years prior to consultation. The headache started abruptly at the time of childbirth. Of note multiple attempts at an epidural for anaesthesia for childbirth occurred before success. Following the procedure and childbirth she was unable to maintain an erect position due to headache

and subsequently a lumbar epidural blood patch was performed which provided some relief although she left the hospital with a low grade daily headache which had persisted 1.5 years later. Approximately 14 months after her initial headache onset she delivered a second child with an epidural but reported no change in her headache at that time. At our meeting she described her headache as throbbing, located in the occipital-nuchal region and in the temples, as well as a pulling sensation in her neck. The pain was worse when she became upright. The headache was present with initial awakening but its severity would increase as the day progressed. The pain was associated with photophobia and phonophobia, as well as poor concentration and poor short-term memory. She expressed difficulty recalling conversations or completing familiar tasks, and was unable to remember new information. She did not complain of any change in her hearing, and she did not medicate for her headaches. A second blood patch had never been discussed between the patient and her treating physicians.

Her general and neurological exam was non-focal, and mini-mental status exam was normal. A brain MRI with gadolinium was ordered and there was questionable PME on conventional post-gadolinium images. It was felt she had a PDPH secondary to a single or multiple dural tears with persistent CSF leakage. A high volume lumbar epidural blood patch (using 37cm³ of blood) was performed. She immediately had nearly 100% resolution of her headache and also had significant improvement in her cognition. However one week following the procedure she regressed to her baseline headache. At follow-up she reported she had been attempting to resolve her baseline headache with over-the-counter analgesics but this was unsuccessful. A repeat blood patch was suggested however the patient was lost to follow up for 15 months. When she returned, now more than three years from initial onset of headache, she reported continuation of her daily positional headache. Her history of rapid improvement and then quick recurrence after epidural blood patch suggests she has persistent single or even multiple dural tears which likely will require surgical intervention for complete resolution of PDPH.

DISCUSSION

PDPH is a potential complication of spinal anaesthesia in obstetrics, and the incidence is affected by a multitude of factors including gender, age, operator technique, needle selection and gauge size.⁴ Additionally, it has been suggested that the incidence of accidental dural puncture during administration of spinal anaesthesia is inversely related to operator experience.⁵ It can also be hypothesized that obstetric patients may actually be at heightened risk for the development of dural tears after epidural anaesthesia because of an inherent weakness in the dura at the time of childbirth as a result of connective tissue changes that occur in the obstetric patient near the time of delivery. These changes include degradation of collagen, an important fibrous protein essential to supporting and providing 'strength' for many bodily tissues, and also alteration in the amount of elastin which works together with collagen.⁶ Accidental dural puncture complicating epidural anaesthesia varies in incidence from 0.19% to 4.4%, while the incidence of epidural-induced PDPH in pregnant women has been reported to range from 76% to 85%.⁷ A weakened dura could also predispose the obstetric patient to persistent CSF

Table 1 Headache presentation in intracranial hypotension²

- (1) Orthostatic headache which is present when upright, relieved by laying supine
- (2) Orthostatic headaches evolving into chronic lingering daily headache
- (3) Non-orthostatic chronic daily headache
- (4) Exertional headache without any positional features
- (5) Thunderclap headache (headache of maximum intensity without latency)
- (6) Second half or later in the day headache
- (7) Paradoxical orthostatic headache so present when supine and relieved when sitting or standing
- (8) Intermittent headaches from intermittent spinal fluid leaks

Figure 1 Brain magnetic resonance imaging postgadolinium axial-based image showing bilateral diffuse pachymeningeal enhancement (arrows)

leaks that would not resolve spontaneously as would typically occur in non-connective tissue altered individuals; thus helping to explain how our two patients had ongoing CSF leaks for three years plus.

The diagnosis of intracranial hypotension is made based on clinical history and neuroimaging. The typical headache of intracranial hypotension is of a positional or orthostatic nature (Table 1). When lying supine the patient has minimal or no pain but when moving out of a supine position the headache becomes either acutely worse (normally early on in the course) or has a gradual worsening recognized as a 'later in the day headache'. The orthostatic or positional headache of intracranial hypotension is felt to be caused by descent of the brain when the patient is upright, which leads to traction on or distortion of the pain-sensitive structures of the posterior

Figure 2 Brain magnetic resonance imaging sagittal image in a patient with prolonged intracranial hypotension. Main imaging findings include crowding of the posterior fossa with cerebellar tonsillar herniation-false Chiari malformation (blocked arrow), flattening of the basis pontis (thin arrow) and enlargement of the pituitary gland (curved arrow)

Table 2 Brain neuroimaging findings in intracranial hypotension²

- (1) Diffuse pachymeningeal enhancement
- (2) Subdural fluid collections
- (3) Brain sag
 - (a) Descent of cerebellar tonsils which can mimic type 1 Chiari malformation
 - (b) Decreased size of prepontine and perichiasmatic cisterns
 - (c) Flattening of the optic chiasm
 - (d) Flattening of the basis pontis
 - (e) Crowding of the posterior fossa
- (4) Enlargement of pituitary gland (engorged pituitary vessels) known as false pituitary tumour sign
- (5) Engorgement of cerebral venous sinuses
- (6) Decrease in size of the ventricles

portion of the brain.¹ The cerebral vein congestion that results as a compensation for CSF volume depletion (Monro-Kellie Doctrine) may also be a potential source of head pain. Importantly in chronic cases the positional component becomes much less distinct or lost all together making the diagnosis even more difficult especially in those with spontaneous CSF leaks, thus no recognized dural trauma. For the obstetric patient most often they have had an epidural at the time of delivery so cause and effect should be easier to establish even without an orthostatic headache but with a history of a new daily headache beginning at or near time of delivery. One bedside test is putting the patient in the Trendelenburg position which rapidly increases intracranial pressure. Thus if a patient has an improvement of headache within several minutes of being in a 10–15° head down tilt position a diagnosis of intracranial hypotension should be suspected even in patients without a positional component to their pain. In many instances outside of some neck stiffness and occipito-nuchal trigger

points, patients with intracranial hypotension may have no abnormal findings on physical and neurological examinations. Possible neurological findings however include unilateral or bilateral sixth cranial nerve palsies, visual field deficit, upper extremity radicular symptoms, Parkinsonism and decreased cognition.¹

Intracranial hypotension can have very specific findings on brain neuroimaging (Figure 1). Common brain MRI findings include diffuse PME, descent of the cerebellar tonsils (which can mimic a Type I Chiari malformation), crowding of the posterior fossa and decrease in size of the prepontine and perichiasmatic cisterns (Figure 2). In addition, subdural fluid collections which are typically hygromas, unilateral or bilateral, may be present and are most often noted over the cerebral convexities. Decreased ventricular size and pituitary enlargement (known as false pituitary tumour sign) may also be present² (Table 2). The issue is that these imaging findings are not always present thus making diagnosis more difficult in cases without a typical orthostatic headache pattern. For example, PME can be absent in upwards of 30% of cases and its presence is dependent on latency between MRI study and headache onset as well as the presence or absence of subdural fluid collections.⁸

PDPH is often benign and self-limited but if left untreated it can lead to more serious complications such as Parkinsonism, cranial nerve palsies, persistent back pain, encephalopathy and even coma.² Both of our patients presented with a dementia type picture that improved after treatment of their intracranial hypotension. There are several other prior case series in the literature demonstrating a frontotemporal pattern of dementia in patients with intracranial hypotension.⁹ Recently functional neuroimaging has even demonstrated hypometabolism of the frontotemporal regions in these patients.¹⁰ A 'false or pseudodementia' needs to be recognized as a possible presenting symptom of intracranial hypotension especially in young women with new daily headaches. Depletion of CSF volume can lead to compression, distortion or traction of the brain, brainstem and cranial nerves as a result of brain descent and may account for the various cranial nerve palsies and CNS manifestations that can be seen in patients with PDPH.² In these two presented cases, it is presumed that their memory loss was secondary to persistent sagging of the frontotemporal lobes.

Treatment for PDPH is generally conservative for the first 24–72 hours after symptom onset and includes bed rest, re-hydration, non-steroid anti-inflammatory medications and antiemetics.¹¹ Caffeine, a central nervous system stimulant which produces cerebral and possibly spinal arterial vasoconstriction, has also been recommended orally or intravenously once to twice daily for several days.⁵ If the conservative approach fails, the most effective intervention is an epidural blood patch (EBP) done at the site of the probable dural tear. The optimum volume of blood used in an EBP has yet to be determined, however a volume of 20 mL or higher has been suggested.¹² In cases where patients have failed conservative measures and epidural blood patches, there are other more invasive procedures which can be considered for treatment of PDPH including epidural injection of fibrin glue (although the exact site of the spinal fluid leak needs to be known for this procedure) and open surgical procedures which are done to close more complicated spinal fluid leaks that are not amenable to closure via a blood patch or fibrin glue.²

RECOMMENDATIONS

In order to reduce the disability and potential consequences of untreated PDPH, it is important to recognize headache as a possible issue at the time of delivery, and it is therefore suggested that obstetricians ask their patients about headache at the time of discharge and at their first postpartum follow-up visit. As the postpartum era is a very hectic time for a new mother and they often put their own problems aside to care for their newborn it would also be of benefit to introduce the concept of PDPH from spinal anaesthesia during prenatal visits. This will allow patients to recognize the importance of new headache following childbirth, with the aim that they will alert their physician should headache occur. If patients are educated about the importance of headache following childbirth then it will be possible to prevent suffering from PDPH and its associated symptoms as well as ruling out other more serious headache aetiologies such as cerebral vein thrombosis.

If a new headache is present at childbirth, then the provider should review the history to assess if the headache characteristics are suggestive of a CSF leak and complete the proper neuroimaging studies (brain MRI with and without gadolinium). If daily headache is present but has no orthostatic features placing the patient in the Trendelenburg position can also define the presence or absence of intracranial hypotension, thus a positive improvement in head pain in a head down tilt position suggests the presence of intracranial hypotension and an underlying CSF leak. If, in fact history, exam and/or imaging are suggestive of CSF hypovolaemia then a lumbar epidural blood patch should be performed at the site of the prior spinal anaesthesia and/or the patient should at least be referred to Neurology for further evaluation. For both our case patients significant quality time with their newborn was lost because of an unrecognized spinal fluid leak caused by spinal epidural anaesthesia.

DECLARATION

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