Case Studies 2

Chapter 27

Three Patients, Three Similar Headaches

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Case History I

A 36-year-old Mexican citizen, a migrant farm worker in the United States for 4 years, was admitted previously to the hospital 1 year prior to the current admission for headache and papilledema. Findings at that time were consistent with cysticercosis with hydrocephalus, and the patient was treated with antibiotics and a ventriculoperitoneal (VP) shunt. Lost to follow-up, compliance with outpatient antibiotics was not established, and the patient presented for current admission with complaints of diplopia, dizziness, and phonophobia. Further questioning revealed positional headache not completely relieved by recumbency. Additional complaints included photophobia, nausea, and an episode of near syncope while painting a ceiling. He reported a normal computed tomography (CT) scan and shunt series 3 months ago. Examination showed ophthalmoplegia, lower extremity hyperreflexia, and a subtle sensory level to pinprick below cervical vertebra C7. Shunt series showed good continuity, but the neurosurgery resident was unable to obtain fluid from the shunt reservoir. Spinal tap was attempted, and only a few drops of fluid could be aspirated. No opening pressure was recorded. Cerebral spinal fluid (CSF) contained six mononuclear cells, and a slight increase in protein. The following day, magnetic resonance imaging (MRI) of the brain showed enhancing meninges and "Chiari malformation." MRI of the cervical spine revealed a central syrinx extended from the craniocervical junction to thoracic vertebra T1. Symptoms improved after replacing the pressure valve on the VP shunt with a programmable valve at higher-pressure settings. Diplopia did not improve.

Case History II

A 24-year-old graduate student was seen for acute and severe headache associated with fever. A prior history of occasionally severe menstrual headache was elicited. After a normal CT scan, spinal tap showed an opening pressure of 140 mm H_2O, with 40 white blood cells (WBCs) and 10 red blood cells (RBCs). CSF protein and glucose were both normal. The patient was admitted to the hospital for observation and with improvement over 24 hours was sent home. Less than 24 hours later, she complained of a severe, throbbing occipital headache with nausea and a feeling of being "confused" when standing. She had slight back pain at the lumbar puncture site. Her headache resolved when supine. CT scan was unremarkable and a second spinal tap had an opening pressure of 20 mm H_2O with 32 WBCs and 1 RBC. Protein was slightly elevated. Headache improved after intravenous fluid bolus and caffeine sodium benzoate, but was still present with standing. A blood patch was performed at the site of the previous spinal tap with near instantaneous resolution of the orthostatic headache.

Case History III

A 68-year-old female with a benign past medical history presented with a history of migraine, retinal hemorrhage 1 year ago, and a left hemiplegic transient ischemic attack 3 months ago. Around that time, she developed a moderate bilateral occipital pain that was present throughout the day. Over the course of days, there was a steady worsening of the headache intensity, becoming so severe that she required bedrest. She also complained of true vertigo with head movement and throbbing pain that became excruciating after standing for more than 5 minutes. Photophobia and, to a lesser extent, phonophobia were present. Her last migraine occurred at the age of 54 years, associated with visual and sensory aura. Family history was remarkable for the death of a brother from heart attack at age 34 years. Social history included the fact that the patient was a basketball player in her youth. Her neurologic

examination was normal except for date confusion and difficulty with medium-term memory. There were clinical signs of dehydration. Laboratory tests were remarkable for a slight elevation of creatinine. MRI showed diffuse meningeal enhancement with a left frontal fluid collection. Attempted spinal taps yielded no fluid.

Questions on the Cases

Please read the questions, try to answer them, and reflect on your answers before reading the author's discussion.

- What do these cases all have in common? How are they different?
- What are the causes of postural headache, and how should they be evaluated?
- What are the consequences of low CSF pressure besides headache?
- Why and how often does a blood patch work, and should it be done to prevent low-pressure headache?

Case Discussion

The river running through these three cases is the literal stream of cerebral spinal fluid. The postural/orthostatic, low-pressure headache (LPHA) links their diverse demography and various etiologies.

Our first patient, a male, non–English-speaking worker, suffers from an overshunted obstructive hydrocephalus, leading to chronically low CSF pressure and a symptom complex referable to traction on oculomotor, trigeminocervical, and vestibulocochlear cranial nerves. The descent of brain causes complications of the craniocervical junction, including a syringomyelia and syncope with neck extension. The main concerns in this patient, with whom communication may be less than optimal, is to rule out continued infection, infarction, or diffuse disease of the central nervous system (CNS). In the emergency room, where an MRI might not be immediately available, the first goal is to guarantee the safety of the patient by checking the continuity of the shunt, rule out shunt infection, and then think through the other possible causes of headache, cranial nerve dysfunction, and midcervical spine disease with long tract signs below the lesion. A Chiari malformation with syrinx of the cervical spinal cord, combined with traction on cranial nerves, ties most of these together.

Our second case is a young woman with a suggestive history of migraine presenting for her "worst" headache associated with systemic signs of infection. The astute clinician immediately attempts to rule out meningitis, and upon finding normal pressure and suggestive spinal fluid, the patient is admitted until fluid analysis confirms no sepsis. The onset of LPHA within 24 to 48 hours after spinal tap is typical, and her accompanying signs of feeling dizzy and confused are nonspecific. The repeated spinal tap reveals improvement in WBCs, although her pressure is low.

Dural puncture either for testing CSF or as a complication of obstetric epidural anesthesia is common in modern medical practice. Operative procedures, including cranial and spinal surgery, constitute larger and more directed breaches of dura mater. Failure to reestablish the integrity of these vital coverings in the aftermath of such procedures is the assumed etiology of LPHA and the attendant nonspecific symptoms of dizziness and nausea. Specific changes, including cranial nerve dysfunction, may be a result of traction on vascular and neural structures that occurs as the brain "sags" under the force of gravity while standing. CSF leaks should always be suspected if recent surgery has been performed, although the time to onset and duration of LPHA may be misleading, and the headache character may be nondiagnostic.

Although the most frequently published cause of LPHA is obstetric epidural anesthesia, it can occur after spinal tap 32 to 36% of the time, most often in thin females. This frequency can be reduced using blunt-tip needles. Eighteen percent of obstetric and 13% of nonobstetric epidural procedures result in symptomatic low CSF pressure. The headache is typically orthostatic, improved with lying down, and diffuse or occipital. Duration can vary from days to weeks, and chronic LPHA does occur.

Since the reasons for which lumbar punctures are performed are various, it is not clear whether normal CSF, or a sloppy, bloody tap predicts a lower incidence of LPHA. Classical teaching states that lying flat for a determined period of time afterward may prevent the onset of this headache. No preference for position, such as supine vs prone vs lateral decubitus, is clear from the literature. Accepted symptomatic treatments include post-onset blood patch, intravenous caffeine sodium benzoate (500 mg in 1 liter of 5% dextrose in water (D5W) infused over 1 hour or more), or other forms of caffeine and large fluid volumes, and nonspecific analgesics including opioids. Antiemetics should be made available to the patient with significant nausea and vomiting. Further evaluation is not necessary unless the headache and other symptoms persist beyond 7 to 14 days, at which time an MRI may be diagnostic. The minimal CSF pleocytosis in LPHA should not confuse a more important diagnosis, although caution in interpretation should always be exercised. Rarely, LPHA may be accompanied by encephalopathy, obtundation, or even coma. This should be kept in mind when seeing a progressively ill patient with encephalopathy, since some inflammatory and infectious conditions including herpes simplex virus encephalitis may present with an initially normal or traumatic CSF.

If our first two cases are the headwaters and upper stream of intracranial hypotension, then the last is the rapids. A confusing history of earlier life migraine, vascular events from both large and small vessels, and slowly progressive mild dementia would not usually be what we would associate with low CSF pressure. Where the other two patients have clear precipitants for low pressure, this third patient does not. Recent reports of spontaneous intracranial hypotension (SIH) have revealed comorbidities with connective tissue disorders and diverse presentations including reversible frontal lobe dementias. Case reports abound with the common themes of distinctive headache, cranial nerve complaints or findings, changes in mental status, variable duration, and partial or complete response to blood patch, even late in the course. Plausible etiologies for SIH have included trauma to the head or further caudad along the neuraxis, violent coughing, exercise, and falls. Often, this history is not recalled.

Dural tears, dehiscence of dural spinal nerve sheaths especially at the root exits, and dural diverticula are presumed to be the cause of SIH. Mokri recently reported examination findings of hyperextensible skin and joints, marfanoid features, arachnodactyly, and tall slender build in SIH. He believes that connective tissue abnormalities predispose to weakness in the meninges with subsequent tear. When seeing a patient with SIH, therefore, a detailed history and examination should also include looking at the presence of hyperextensible joints; Marfan's syndrome or features; personal or family history of aortic or other large vessel aneurysms; and retinal detachment, especially at a young age. In our patient, the family history of young cardiac complications, and her tall athletic build in her youth, bolster our intuitive sense that she has SIH. Ultimately, a spinal MRI was suggestive of an enlarged nerve root at T6, and contrast myelogram confirmed a fluid leak at that location. A neurosurgical consult recommended against an open spinal procedure, and a blood patch placed at lumbar vertebrae L2–3 resulted in moderate improvement of the clinical symptoms. However, 1 month later, as her "dementia" worsened, a laminectomy was performed revealing a dural outpouching, which was oversewn with complete resolution of the headache and photophobia. Interestingly, her memory did improve, although date confusion still remained. A repeat CT scan done 3 months later showed no change in the frontal fluid collection.

Investigation

The 1988 International Headache Society (IHS) criteria for headache associated with low CSF pressure (IHS code 7.2) includes the following:

A. Bilateral headache

B. Headache that occurs or worsens less than 15 minutes after assuming the upright position and disappears or improves less than 30 minutes after assuming the recumbent position

The IHS also included 7.2.1 postlumbar puncture headache, and 7.2.2 posttraumatic, postoperative, or idiopathic cerebrospinal fluid leak.

The newer (2004) IHS classification for headache attributed to low pressure is outlined in Appendix 27-1; however, the principles of diagnosis are the same, with now a greater need to separate out the subtypes.

In order to clarify the possible etiologies of LPHA, a simple division into "provoked" and "spontaneous" types could be made, with extensive overlap between these arbitrary groupings. Of course, those occurring after spinal tap are the most familiar, and those after epidural anesthesia for childbirth are the most commonly studied. Additional clinical symptoms besides headache appear to be localizable to stretching of penetrating veins, cranial nerves, and separation of meningeal membranes including hygromas (Table 27-1).

The more uncommon symptoms listed in the table appear to be the result of mechanical forces, not from neuronal injury. Resolution of LPHA and neurologic symptoms is nearly universal. The presence of cranial nerve or spinal symptoms should be reevaluated regularly for improvement. Repeated evaluations may be necessary to confirm resolution of CSF leak.

CSF is usually normal, although a slight pleocytosis and increase in protein may occur. Biopsies of dura mater resemble an organized hygroma with a variable thin zone of fibroblasts and small thin-walled vessels. These nonspecific changes are felt to result from CSF volume changes, not infiltration or inflammatory.

It is the distinctive findings on MRI with gadolinium that define the fit between the clinical presentation and structural changes attendant upon LPHA. Initial hypo-

Table 27-1. Clinical Symptoms Associated with Low-pressure Headaches and Possible Etiologies

Symptom	Localization
Postural headache	Traction on dural nerves
Nausea, emesis	Unknown
Neck tightness and interscapular pain	Root traction
Dizziness	CN VIII
Horizontal diplopia	CN III or VI
Photophobia	Unknown
Distortions of hearing	CN VIII
Blurring of vision	Unknown
Superior binasal visual field deficits	Traction on optic chiasm
Radicular upper limb symptoms	Syrinx or traction on nerve root
Rare facial dysesthesia or weakness	CN VII

CN = cranial nerve.

volemia in CSF spaces results in compensation by venous structures that become engorged, taking up gadolinium into the pachymeninges. The resultant enhancement and displacement of hindbrain structures creates a pathognomonic picture on MRI that includes diffuse pachymeningeal enhancement, sagging brain including Chiari malformation, and subdural fluid collections.

In an extensive review of his own cases and the literature, Mokri has described four clinical-imaging syndromes, including one without headache and one with normal imaging. For those interested in more detail, further reading is recommended (see Selected Readings).

Management Strategies

It goes without saying that identifying persistent CSF leaks should be the first goal of the treatment of LPHA. This is especially true in suspected postoperative or spontaneous LPHA. In the more common cases of postdural puncture headache, a high index of suspicion is the next goal. Orthostatic headache in the wake of a procedure should be assumed, even if later in onset, longer in duration, or less severe than the clinician might usually expect. In most of these atypical cases, an MRI will confirm your suspicion.

Treatments that have been suggested in the past have included bedrest, caffeine, abdominal binders, and continuous epidural infusion of saline. Outpatient treatment should include a consideration for intravenous fluid and caffeine sodium benzoate 500 mg in 1 liter of D5W, although severe cases should be triaged immediately to blood patch.

The standard treatment for symptomatic LPHA remains the epidural blood patch (EBP), although a recent meta-analysis by Sudlow and Warlow has questioned its efficacy and utility. In contrast to the results of this meta-analysis, clinical practice routinely encounters good results. In their excellent review of the literature, Duffy and Crosby recommended that EBP be performed by a skilled practitioner using blood volume of 10 to 20 mL. Persistent relief and resolution occurs in 60 to 75% after first EBP. The use of non-blood solutions, such as saline, appears to result in a higher incidence of headache recurrence. They do recommend prophylactic blood patch or saline infusion if there is an obvious breach of the dura during a procedure, although Sudlow and Warlow do not concur. The report by Sudlow and Warlow found that time alone (LPHA > 7 days) was an indication for treatment with epidural blood patch, finding no distinctive advantage to prophylactic EBP or non-blood infusion. Opposing this, the American Academy of Neurology recommends the continued use of EBP for symptomatic LPHA.

To this practitioner, the most fascinating occurrence amongst all of these controversies is the frequent and nearly immediate relief of postural headache after blood patch. Originally felt to be a consequence of "plugging" the hole, this dramatic event probably results from a direct effect of the "pressure patch," which the mass of fluid places on the CSF compartment. Transmitted via the dura, this rapid increase in pressure restores intracranial CSF volume, relieving the brain sag and thus the headache. The "plug" created by the blood then begins to form, allowing the continuously created CSF to replenish the system quickly without concurrent loss via leakage. Surgical measures, including closure of meningeal diverticula, when identified, or direct packing of a defect, require additional evaluation and surgical expertise.

Case Summary

These three very different cases of LPHA point out the importance of history and the need to be suspicious.

- Whereas an overfunctioning shunt may be obvious, so too is the need to rule out other causes of progressive neurologic symptoms in a young worker.
- Although we feel overly confident when recognizing LPHA in an otherwise healthy young woman after lumbar puncture or childbirth, persistent symptoms require ongoing care and understanding, since not all patients immediately improve.
- The complex older patient with multiple possible diagnoses may elude our clinical acumen, only to have the obvious changes on MRI unraveling a fascinating new area of clinical associations and understanding of the make-up of the coverings of the CNS and the vital fluid contained within its banks.

Overview of Low-pressure Headache: A Pathobiologic Correlation

Surrounding the precious structures contained within the CNS, the meninges and bony structures protect the brain and spine by providing a barrier to trauma and unwanted invasion from without. The CSF bathes and buoys. Nearly simultaneous with the recognition of the existence and importance of CSF, attempts to evaluate this vital fluid were made. Our understanding of CSF production and dynamics has been growing since the original publications of Schaltenbrand and others in the late 1930s. He coined the term aliquorrhea, where low, unmeasurable, or negative pressures were recorded on human spinal taps. Originally, pathophysiologic ideas included underproduction of CSF, although no example of this has yet been clearly demonstrated. Instead, loss of CSF has been established as the consistent cause of LPHA.

CSF is created in the choroid plexus and flows through the ventricular system, ultimately forming a stable, con-

stantly replenished pool that bathes and cushions the brain, spine, and nerve roots as they exit the CNS headed to their target destinations. The fluid space contains approximately 150 mL of CSF, although variability is present on the basis of gender and body size. With age and resultant atrophy, decreases in brain mass result in ventricular enlargement, and thus more CSF. Most normal individuals produce 0.35 mL of CSF per minute, replacing the entire fluid compartment at least once or twice a day. The vast majority of CSF is removed via bulk flow through the arachnoid villi of the venous sinuses, with a constant balance being maintained via noncomplex fluid dynamics. CSF pressure at the lumbar sac varies between 80 and 200 mm H_2O, and is equal to intracranial pressure when the patient is recumbent. Large increases in lumbar pressure occur with standing.

In the absence of CSF, the adult brain can weigh between 1,300 to 1,400 g. Buoyed by this aqueous solution, however, the brain in situ "weighs" approximately 50 g. This "apparent weight" is also maintained by suspension from above by cerebral veins originating from the sagittal sinus and from the cerebellar veins that connect to the transverse and straight sinuses. From below, the veins originate in the tentorium, and the skull base ultimately buoys the brain. As CSF is lost, nociceptors attached to the veins are stretched and activated. The brain gets "heavier" and ultimately "sags" into the craniocervical junction, elongating cranial nerves and brainstem structures.

The Monroe-Kellie doctrine states that the sum of the intracranial contents (eg, the brain, blood, and CSF) of the cranium remains constant such that changes in one mandate elicit changes in the others. It is this simple, intuitive equation that determines the changes that occur when an individual suffers the consequences of low CSF pressure, including orthostatic headache. Intracranial pressure falls as a result of a decrease in total contents, and since the brain tends not to swell without definitive cause, the only spaces that can expand are the veins of the sinuses and meninges. In other words, venous engorgement and thickening of the meninges must occur in order to "fill the space." Continued CSF leak compels further expansion of the venous structures, and gravity causes a downward displacement of the infratentorial brain structures, including the cerebellar tonsils. Ultimately, extracerebral hygromas can result, and some have noted increased size of the pituitary, felt to be a result of pituitary venous hypervolemia. The consequences of these structural and functional changes are the easily r ecognizable changes on MRI scans.

Conclusions

LPHA is a symptomatic, secondary headache that can present hours and days after a breach of the CSF-containing spaces, or in rarer cases, can occur spontaneously with a long clinical course. Clinical findings can include changes in mental status, cranial nerve dysfunction, and systemic signs including those complaints typically associated with migraine. Diagnostic findings on MRI consist of diffuse meningeal enhancement with or without Chiari malformation. Spinal tap may result in low, absent, or negative pressure, with normal fluid more the rule than not. Improvement with epidural blood patch should be expected even if the cause is unknown, although repeated treatments may be necessary and incomplete recovery does occur. Spontaneous LPHA may occur in connective tissue disorders, and further evaluation should be considered in those with historic or clinical signs of hyperextensible joints, Marfan's syndrome, or disorders of the large vessels including carotid or abdominal dissection. Symptomatic treatment with nonspecific analgesics is also indicated.

LPHA represents the outcome of hydrodynamic changes and mechanical forces influencing craniocervical nociception, and thus, it is a true secondary headache no matter what its features. Headache that worsens with standing and improves with recumbency in a patient with recent onset, or in rarer cases, chronically, should always lead to an evaluation for this disorder.

Selected Readings

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Editorial Comments

Headaches that change with posture are well known to physicians and their patients, and of course, even primary headaches can worsen with movement or a change in head position. However, when headaches or associated neurologic findings clearly improve in the recumbent position, or occur in clinical scenarios in which LPHAs are known to arise, such as postlumbar puncture, then it is wise to look harder for a correctable etiology. The spectrum of LPHA disorders continues to expand and contains some of the most interesting of the headache disorders (see the new IHS classification in Appendix 27-1). It is interesting that the evidence-based medicine approach is a wait-and-see approach, in contradistinction to the usual interventions utilized by clinicians in practice. A balanced approach is probably the best, as all good clinicians know from experience.

Final diagnosis:

Low cerebrospinal fluid pressure headaches

Appendix 27-1

7.2 Headache Attributed to Low Cerebrospinal Fluid (CSF) Pressure

7.2.1 Postdural (postlumbar) puncture headache

Diagnostic criteria:

A. Headache that worsens within 15 minutes after sitting or standing and improves within 15 minutes after lying down, with at least one of the following and fulfilling criteria C and D:
   1. Neck stiffness
   2. Tinnitus
   3. Hypacusia
   4. Photophobia
   5. Nausea
B. Dural puncture has been performed
C. Headache develops within 5 days after dural puncture
D. Headache resolves due to either of the following*:
   1. Spontaneously within 1 week
   2. Within 48 hours after effective treatment of the spinal fluid leak (usually by epidural blood patch)

*In 95% of cases, this is so. When headache persists, causation is in doubt.

7.2.2 CSF fistula headache

Diagnostic criteria:

A. Headache that worsens within 15 minutes after sitting or standing, with at least one of the following and fulfilling criteria C and D:
   1. Neck stiffness
   2. Tinnitus
   3. Hypacusia
   4. Photophobia
   5. Nausea
B. A known procedure or trauma has caused persistent CSF leakage with at least one of the following:
   1. Evidence of low CSF pressure on magnetic resonance imaging (MRI) (eg, pachymeningeal enhancement)
   2. Evidence of CSF leakage on conventional myelography, computed tomography (CT) myelography, or cisternography
   3. CSF opening pressure < 60 mm H_2O in sitting position
C. Headache develops in close temporal relation to CSF leakage
D. Headache resolves within 7 days of sealing the CSF leak

7.2.3 Headache attributed to spontaneous (or idiopathic) low CSF pressure

Previously used terms:
Spontaneous intracranial hypotension, primary intracranial hypotension, low CSF volume headache, hypoliquorrheic headache

Diagnostic criteria:

A. Diffuse and/or dull headache that worsens within 15 minutes after sitting or standing, with at least one of the following and fulfilling criterion D:
   1. Neck stiffness
   2. Tinnitus
   3. Hypacusia
   4. Photophobia
   5. Nausea
B. At least one of the following:
   1. Evidence of low CSF pressure on MRI (eg, pachymeningeal enhancement)
   2. Evidence of CSF leakage on conventional myelography, CT myelography, or cisternography
   3. CSF opening pressure < 60 mm H_2O in sitting position
C. No history of dural puncture or other cause of CSF fistula
D. Headache resolves within 72 hours after epidural blood patching

Comments:
The underlying disorder may be low CSF volume. A history of trivial increase in intracranial pressure (eg, on vigorous coughing) is often elicited. In other cases, a sudden drop in atmospheric pressure has occurred.
Postural headache resembling that of low CSF pressure has been reported after coitus. Such headache should be coded here because it is due to CSF leakage.
Many patients with spontaneous low CSF pressure headache respond to epidural blood patching, epidural saline infusion, or pharmacologic therapies such as intravenous caffeine or conventional analgesics. Some have spontaneous resolution of their headache, while others relapse after initial successful treatment. Cases of dural sleeve herniation, particularly in the thoracic area, have been reported and have been successfully treated surgically.
Dural puncture should be avoided in patients with positive MRI signs such as meningeal enhancement with contrast.

Adapted from Headache Classification Subcommittee of the International Headache Society, 2004.