Lumbar and Thoracic Epidural Blood Injections to Treat Spontaneous Intracranial Hypotension

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HEADACHE secondary to low cerebrospinal fluid (CSF) pressure occurs after diagnostic lumbar puncture, myelography, cranial or spinal injury, or spinal anesthesia. Rarely, however, is it spontaneous, a condition called “spontaneous intracranial hypotension” (SIH). The clinical characteristics of the syndrome are the same as those occurring after dural puncture, the most important being the presence of postural headache. Treatments of low CSF pressure headache consists of caffeine, corticosteroids, continuous epidural saline infusion, and epidural blood patch. We describe three patients with SIH: epidural blood injections at the lumbar and thoracic levels resulted in complete and permanent resolution of the headache in two patients, and a lumbar epidural blood injection relieved the headache in the third patient. We believe these are the first case reports of the use of epidural blood injections at different vertebral levels to manage low CSF pressure headache.

Case Reports

Case 1

A 27-year-old woman was admitted with a 3-week history of postural headache. She had no history of chronic headache, meningitis, cranial or spinal trauma, or parasanal sinus infection. Her headache started suddenly at the occiput and radiated to involve the entire cranium. She had no fever, nausea, or vomiting but developed diplopia 2 weeks after the onset of the headache. Other than a left cranial nerve VI paresis, results of her neurologic examination were normal. Magnetic resonance imaging (MRI) of her brain and cervical spine showed diffuse thickening and enhancement of the meninges and tentorium. Lumbar puncture, aided by fluoroscopy, showed an opening pressure of 2 mm H2O (normal, 70 to 180 mm). Cerebrospinal fluid analysis showed one leukocyte (normal, 0 to 10 cells), 930 erythrocytes, a glucose level of 60 mg/dl (normal, 45 to 80 mg/dl), and total protein concentration of 462 mg/dl (normal, 15 to 45 mg/dl). Intrathecal radionuclide cisternography showed the inability of the isotope to enter the cranium and the absence of CSF leak at the 1-h, 4-h, 24-h, and 48-h scans. A diagnosis of spontaneous intracranial hypotension was made, and the patient was treated with bed rest and acetaminophen with codeine. Her headache did not resolve after 1 week of bed rest and an epidural injection of blood was done on the eighth hospital day; 20 ml of autologous blood was injected through a 17-gauge Tuohy needle inserted at the L2–L3 interspace. Her headache immediately resolved and she was discharged. Two days after the epidural blood injection, the postural headache recurred. The patient was treated in the ambulatory setting and 11 intravenous fluid was given over 1 hour, and 20 ml of autologous blood was injected at the T9–T10 level. Her headache resolved completely and did not recur in the ensuing 7 months.

Case 2

Postural headache developed suddenly in a 29-year-old woman with no history of chronic headache, head or neck trauma, or sinus infection. Raising her head 30 degrees resulted in severe and diffuse headache. Results of a neurologic examination were normal. Magnetic resonance imaging of her brain showed meningeal enhancement, and MRI of her spine showed a bulging disc at T11–T12. A lumbar puncture revealed an opening pressure of 45 mm H2O. Cerebrospinal fluid analysis showed 17 leukocytes, 8 erythrocytes, a glucose level of 55 mg/dl, and protein concentration of 59 mg/dl. Stains and cultures for bacteria and fungi were negative. Ketonolac (30 mg given intramuscularly every 6 h) did not relieve her headache and the patient was given 50 mg meperidine intramuscularly every 6 h. An isotope study was recommended several times but the patient refused. An epidural injection of 20 ml autologous blood was performed at the L3–L4 level. There was no relief of her headache. Two daily intravenous infusions of 1 g caffeine also failed and a thoracic (T11–T12) epidural blood injection was performed. Only 11 ml autologous blood was injected because the patient experienced severe pressure in her back after 8 ml had been injected. Her headache resolved completely and she remained asymptomatic for 5 months after the procedure.

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Case 3

A 37-year-old woman with no previous history of headache, head or neck trauma, or sinus infection experienced a sudden onset of postural headache. Two weeks later, blurred vision, nausea, vomiting, vertigo, and ataxia developed. She was admitted to another hospital, where MRI of her head and her cervical spine showed meningeal enhancement. Lumbar punctures were difficult to perform and only a few milliliters of blood and CSF were aspirated. Cerebrospinal fluid analysis showed 1,540 erythrocytes, 10 leukocytes, a protein level of 72 mg/dl, a glucose concentration of 43 mg/dl, and negative cultures for bacteria and fungi. She was admitted to our hospital for further work-up and management. Results of her physical examination were significant for an unsustained horizontal nystagmus on lateral gaze and a positive Romberg sign. Radiouclide lumbar cisternography showed an opening subarachnoid pressure of 0 mm H₂O. No leak was demonstrated and the dye could not reach the cranium. Analysis of the CSF obtained during the cisternography showed 11 leukocytes, a protein level of 85 mg/dl, and a glucose concentration of 61 mg/dl. A lumbar (L2-L3) epidural injection of 20 ml autologous blood was performed on the fourth hospital day. Her headache was relieved completely and she remained asymptomatic 5 months later.

Discussion

Low CSF pressure headache, also called Schaltenbrand syndrome, spontaneous sialorrhea, or spontaneous intracranial hypotension, is a rare syndrome. Its principal feature is the presence of postural headache with no preceding trauma or lumbar puncture. The headache is intense and usually involves the entire cranium, although it may be localized to the occipital or frontal areas. Other symptoms include neck stiffness, nausea and vomiting, photophobia, anorexia, general malaise, vertigo, tinnitus, and diplopia. The vestibular and auditory symptoms have been attributed to changes in the intralabyrinthine pressure secondary to changes in the pressure gradient across the cochlear aqueduct. The diplopia is often due to lateral rectus palsy caused by downward displacement of the sixth cranial nerve.

Lumbar puncture is necessary to document low CSF pressure. Cerebrospinal fluid pressure less than 70 mm H₂O with the patient in the lateral decubitus position is considered sufficient for diagnosis, although the usual finding is approximately 50 mm H₂O. In some patients, low CSF pressure precludes spontaneous drainage of CSF. Analysis of the CSF usually reveals increased protein content and increased red and white cell counts. The increased protein may be due to alteration of the normal hydrostatic and oncotic pressures across the venous sinus and arachnoid villi or due to the passage of proteins into the CSF if there is a microscopic leak or a disruption of the meninges. The increased erythrocyte count may be due to meningeal hyperemia and subsequent diapedesis of erythrocytes into the subarachnoid space.

Normal meninges, with the possible exception of the falx cerebri, are difficult to visualize on noncontrast MRI studies because of the high signal from the CSF. Meningeal enhancement on the MRI is a recently noted abnormality in patients with SIH in which the meningeal enhancement is usually thick, diffuse, and continuous. Its pathogenetic mechanism is unknown, although it may be related to dural venous dilatation that accompanies reduced CSF volume. Follow-up MRI of patients whose headaches were relieved showed resolution of the meningeal enhancement.

The proposed mechanisms of headache in SIH include CSF leakage through a small dural tear, reduced production of CSF, or hyperabsorption of CSF. However, no scientific evidence exists to support the latter two theories. Furthermore, the probability of CSF hyperabsorption as a cause of low CSF pressure has been questioned because bulk absorption of CSF does not occur when the subarachnoid pressure decreases to less than 60 mm H₂O because CSF absorption is related to the pressure difference between the subarachnoid space and draining venous channels. A more plausible explanation is the presence of a leak through the dura or through a nerve root sleeve. There may be a variation of the normal root sleeve anatomy that predisposes to small tears or dehiscence of the dural sheath after a minor fall or exercise. Alternatively, there may be small defects in the meninges, such as epidural or perineural cysts that are susceptible to rupture after minor trauma.

Radionuclide cisternography has been recommended as a diagnostic test, but its utility has been limited in persons with SIH. In patients in whom no leak is demonstrated, it is presumed that the CSF escapes through a microscopic dural tear. A common finding in patients with SIH is the rapid appearance of radioisotope tracer in the bladder and kidneys, probably due to leakage of CSF through the dura and uptake into the circulation through the epidural venous plexus. The inability of the tracer to ascend to the foramen magnum and basal cisterns is consistent with a low CSF pressure state.

The headache of SIH takes several weeks or months to resolve spontaneously. Treatments for SIH include bed rest, caffeine, abdominal binder, steroids, continuous epidural saline infusion, and epidural...
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blood "patch."\(^1\)\(^4\)\(^5\)\(^8\)\(^9\) With regard to epidural blood injection, case reports have documented the effectiveness of a single blood injection,\(^3\)\(^8\)\(^9\) whereas in another report, the blood injection had to be repeated after 2 months because of recurrent headache.\(^4\) In that case report,\(^4\) neither the volume injected nor the vertebral level of the blood injection was mentioned. We suspect both blood injections were done at the lumbar level because all the references in the article involved lumbar placement of a blood "patch." Our study differs from this report\(^1\) in that the initial lumbar epidural blood injections in two of our patients were either transiently effective or ineffective and the subsequent thoracic blood injections were performed after a 2- to 3-day interval. It is possible that two lumbar epidural blood injections would have been effective in our patients. We decided on a thoracic approach because we suspected a CSF leak in the thoracic region after our first patient had a transient response to the lumbar epidural blood injection and after our second patient had no response to the lumbar epidural blood injection.

We do not know why the lumbar epidural blood injections were not completely effective. The increased subarachnoid pressure after the first blood injection\(^1\)\(^3\) may have been inadequate in our first patient or the blood may not have reached the level of the CSF leak. Our second patient may have had a CSF leak at a higher vertebral level. We injected only 11 ml autologous blood in our second patient because she experienced increasing back pressure. We did not want to precipitate nerve root compression with additional volume. This risk of epidural blood injection has been noted with a cervical level of injection or where there is little space between the dura and the ligamentum flavum.\(^1\)

We described three patients who exhibited the signs and symptoms of low cerebrospinal fluid pressure headache. Two of them responded to successive lumbar and thoracic epidural blood injections, and the third patient improved after a lumbar epidural blood injection. Anesthesiologists and pain management specialists should consider these treatments in patients with low CSF pressure headache.

References