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Two Cases of Spontaneous Intracranial Hypotension

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= Abstract =

Spontaneous intracranial hypotension(SIH) is a rare syndrome which causes postural headaches associated with low CSF pressure. Headaches are usually accompanied by on rare occasions diplopia, and transient visual obscuration as well as nausea, vomiting, tinnitus, neck stiffness, vertigo, photophobia, and phonophobia. CSF leakage from a spinal meningeal defect may be the most common cause of this syndrome. Downward sagging of the brain due to low CSF pressure may produce headaches by traction of intracranial and meningeal pain-sensitive structures. Lumbar puncture reveals low opening pressure. CSF protein and cell count may be slightly elevated. Brain and cervical MRI may show diffuse and continuous pachymeningeal enhancement with gadolinium, subdural effusion, or downward displacement of the brain. We present two patients with SIH, in whom epidural blood patch produced an immediate and complete resolution of the symptoms. We couldn' t identify CSF leakage site in either of them.

Key Words:Spontaneous intracranial hypotension, Epidural blood patch, Dural enhancement

Introduction

The International Headache Society (IHS) defines postural headache as one that "occurs or worsens less than 15 minutes after assuming the upright position, and disappears or improves less than 30 minutes after resuming the recumbent position" (Schievink *et al*, 1994). It may be classified according to etiology: 1) post-lumbar puncture (diagnostic, with myelography or spinal anesthesia), 2) spontaneous, 3) traumatic (with or without clinically obvious CSF leakage), 4) postoperative, 5) that associated with other medical conditions (severe dehydration, diabetic coma, uremia, hyperpnea, meningoen-cephalitis, severe systemic infection) (Marcelis & Silberstein, 1990; Schievink *et al*, 1994; Rendowden *et*

al, 1995). Postural headache secondary to low intracranial pressure is commonly enco-untered following lumbar puncture for cerebrospinal fluid (CSF) examination or myelography, spontaneous, and CSF shunt overdrainage sydrome (Good & Ghobrial, 1993; Chung *et al*, 1996).

Spontaneous intracranial hypotension syndrome was first proposed in 1938 by a german neurosurgeon Schaltenbrand who termed it "aliquorrhea" and described it as a headache syndrome virtually identical to that following lumbar punct-ure (Labadie *et al*, 1976; Gaukroger & Brown-ridge, 1987). Spontaneous intracranial hypotension(SIH), by definition, is not associated with any significant trauma but is frequently associated with innocuous precipitating factors, such as sneezing, coitus, or a minor fall (Wilton *et al*, 1986; Fernandez, 1990; Rando & Fishman, 1992; Chung *et al*, 1996).

The lumbar puncture characteristically reveals a low opening pressure of less than 60 mmH_2O (Bang *et al*, 1997). The CSF examination is usually normal but may show a moderate pleocytosis, presence of red blood cells, and elevated protein (Bang *et al*, 1997). A brain CT may show slit-shaped ventricles with tight basal cistern and scant CSF over the cortex (Murros & Fogelholm, 1983). A brain and cervical MRI may show diffuse and continuous pachymeningeal enhancement with gadolinium, brain shifting, and subdural effusion (Fishman & Dillon, 1993; Pannullo *et al*, 1993; Honma *et al*, 1996; Kinoshita *et al*, 1997). Using CSF analysis, CT or MRI abnorm-alities are important in diagnosing of the SIH, thus avoiding the use of unwarranted invasive investigations and treating this syndrome effectively.

Only nine cases of SIH have been repo-rted in South Korea until now, moreover it has been rarely reported that epidural blood patch improves the postural heada-che in SIH successfully without identifi-cation of the CSF leakage (Han *et al*, 1995; Lee *et al*, 1995; Kim *et al*, 1995; Chung *et al*, 1996; Bang *et al*, 1997). We present two patients with SIH in whom the epidural blood patch produced immediate and complete relief from incapacitating postural headache, and review those previously described in the literature.

CASE 1

A 38-year-old previously healthy woman presented with severe occipital headache for 2 days, which was aggra-vated when she erected and relieved when she lay down. The headache was accompanied with nausea, vomiting, and chills. She spent most of a day in the supine position to relieve the headache. On examination, she was slender, pale, and appeared acutely ill. She was afebrile with no meningeal signs. A lumbar puncture in the lateral decubitus positon revealed opening pressure of 70 mmH_2O. There was mild pleocytosis, 23 lympho-cytes per mm^3, and protein level of 77 mg/dl. The findings in cytology and multiple cultures were negative. The following laboratory studies were normal: complete blood count, erythrocyte sedim-

entation rate, blood chemistry, and blood serology. She was managed with a conservative therapy as having viral meningitis. Her headache improved gradually. She was discharged a week later with slight occipitalgia. One week after discharge, she was readm-itted, complaining of postural headache, nausea, vomiting, tinnitus, neck stiffness, and transient visual obscuration. Her vital signs were normal. Physical and neurologic examination remained normal except for very mild nuchal rigidity. Repeated CSF examination revealed an opening pressure of 40 mmH_2O, 12 lymphocyes per mm^3, and protein level of 55 mg/dl. Repeated smear, culture, and cytology remained negative. Brain MRI showed diffuse and continuous pachymeningeal enhancement with gadolinium (Figure 1). Conservative therapy and complete bed rest were begun on admission and produced slight improv-ement. Two days after readmission, an epidural blood patch with 12 ml autologous blood was performed at the L_{3-4} interspace level. Thereafter, she was kept supine for 6 hours. Four days after this procedure, she obtained complete relief from her postural headache. Follow-up study in several months later showed that she remained completely free of postural headache.

CASE 2

A 57-year-old woman with a history of rheumatoid arthritis and osteoarthritis developed severe occipital headache, worsening over a week period, which was precipitated by sitting or standing for more than 10 minutes. The headache was relieved by lying flat for 10 minutes. The headache was usually accompanied by

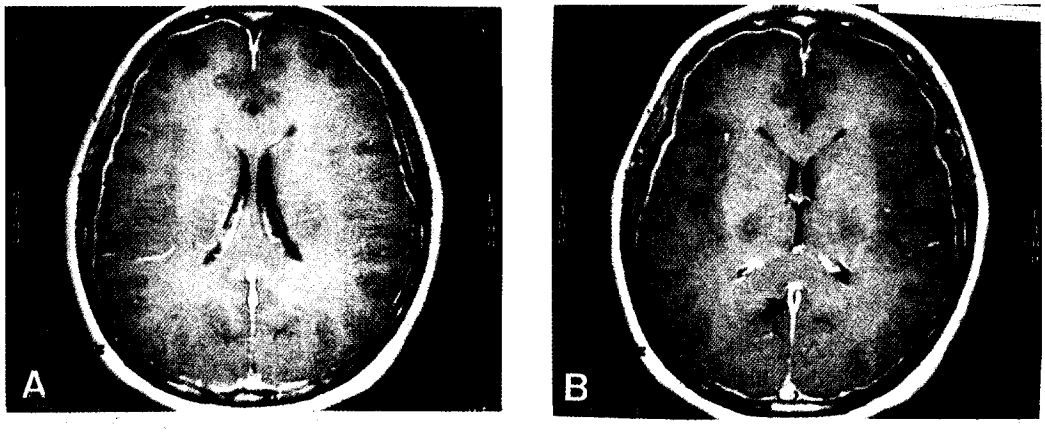

Figure 1. Axial view of gadolinium-enhanced T1-weighted MRI on admission. (A) The scan shows diffuse thickening and enhancement of the pachymeninges along both cerebral hemisphere and falx cerebri (arrows). (B) The scan shows thickening and enhancement of tentorium cerebelli (arrows)

nausea, vomiting, and tinnitus. General physical and neurologic examination were normal except for deformities of several fingers. Lumbar puncture in the lateral decubitus position revealed opening pressure of 60 mmH_2O. There was a mildly elevated protein level of 51 mg/dl, but the other findings were normal. The findings in cytology and multiple cultures were negative. Routine blood test and chest radiography were normal. Brain MRI without contrast enhancement was normal. Postgadolinium MRI revealed diffuse thickening and enhancement in the pachymeninges (Figure 2). There were neither mass effect nor tonsillar herniation. Radioisotope cisternography was normal.

The patient was managed with bed rest, hydration and analgesics but continued to complain of the headache. Three days later, first epidural blood patch with 12 ml autologous blood was performed at the L_{1-2} interspace level, then she was kept supine for about 10 minutes. Five days after this procedure, she felt mild improvement in her headache and was able to sit upright for 30 minutes until an occipital headache developed. Several days later, her headache was exacerbated by upright position to the point that she could stand or sit upright for more than 5 minutes. A second autologous epidural blood patch was done at the same level, with the same amount. Within two days, there was a dramatic improvement in her headache with the persistent and posturally related headache resolving after three days.

Discussion

Symptoms of intracranial hypotension present themselves when CSF pressure fall below 90 mmH_2O with the headache

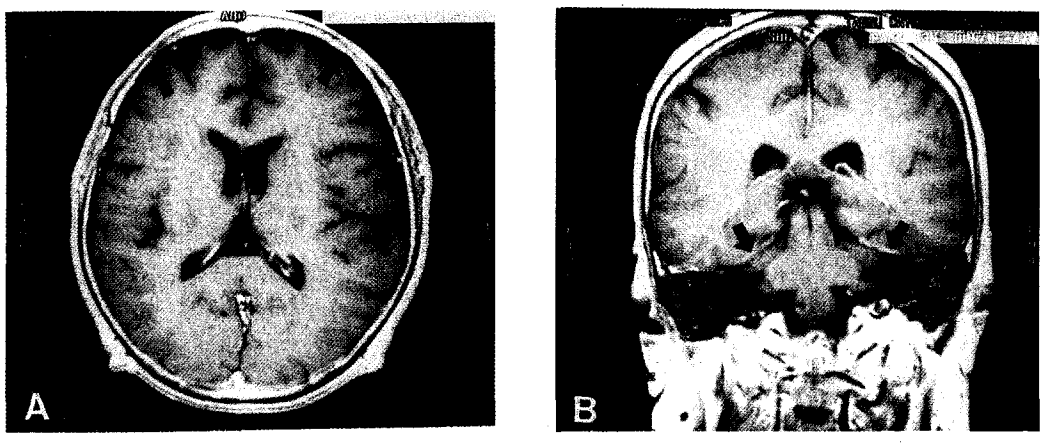

Figure 2. (A) Axial gadolinium-enhanced T1-weighted MRI on admission shows diffuse pachymeningeal enhancement along both cerebral hemisphere (arrows). (B) coronal plane through the posterior fossa also shows abnormal meningeal enhancement (arrows).

being the most common clinical symptom (Silberstein *et al*, 1995). SIH headache may be sudden or gradual in onset and is usually accompanied by intense, severe, throbbing, or dull pain, which may be generalized or localized to the frontal or occipital region (Christine *et al*, 1997). The headache is aggravated by the upright position, head shaking, and jugular venous compression (Silberstein *et al*, 1995). The most charateristic feature (Silberstein *et al*, 1995) of SIH is that the pain can be relieved by lying flat, usually within minutes. The headache is probably the result of increasing brain volume, secondary to the dilatation of the cerebral vein and meningeal vasculature to compensate for the low CSF pressure. We consider the changes to be a consequence of the Monro-Kellie rule that says that the CSF volume fluctuates reciprocally with these changes in intracranial blood volume when the skull is intact (Fishman & Dillon, 1993). Alternatively, the headache may be secondary to traction on pain sensitive structures, in particular, cranial nerve Ⅴ, Ⅸ, Ⅹ and the upper three cervical nerve following downward "sagging" of the brain due to low CSF pressure in the upright posture (Pannullo *et al*, 1993).

Nausea, vomiting, dizziness, photophobia, anorexia and general malaise are commmonly associated (Marcelis & Silberstein, 1990; Pannullo *et al*, 1993; Silberstein *et al*, 1995; Rendowden *et al*, 1995; Ramadan, 1996; Christine *et al*, 1997). In addition, tinnitus, bilateral hyperacusis, unsteadiness or staggering gait, diplopia, transient visual obscuration, hiccups, dysgeusia, and mental change may be accompanied with SIH (Christine *et al*, 1997; Pleasure *et al*, 1998). Our two patients complained of tinnitus in both ears and one of them complained of transient visual obscuration in both eyes. Auditory symptoms of our patients are thought to occur due to lowering of CSF pressure which reduces intralabyrinthine pressure and fails to transmit high frequency sound (Rando & Fishman, 1992). Additionally, there may be an alteration in the pressure across the vestibular and cochlea aqueducts and it may cause vestibular symtoms. Visual problems include diplopia, transient visual obscu-ration, blurred vision and peripheral visual field depression, which are probably secondary to distortion or traction of the optic nerve and chiasm or abducens nerve due to downward displacement of the brain, following low intracranial pressure (Berlit *et al*, 1994; Horton & Fishman, 1994). Physical examination may show mild neck stiffness and slow pulse rate ("vagus pulse") (Marcelis & Silberstein, 1990; Silberstein *et al*, 1995).

Clinical history is sufficient to diagnose post-dural puncture headache, but SIH needs to low CSF pressure to be identified (Han *et al*, 1995). The diagnosis of low-pressure headache is confirmed by lumbar puncture in the lateral decubitus position demonstrating CSF pressure of less than 70 mmH_2O (Marcelis & Silberstein, 1990; Silberstein *et al*, 1995; Bang *et al*, 1997; Silberstein *et al*, 1995; Christine

et al, 1997). However, the opening pressure may be in the normal range, especially if the measurement was made after a period of recumbency. In our cases, the lumbar puncture showed low CSF pressure of less than 70 mmH_2O, mild lymphocytic pleocytosis, mild elevation of protein while severe headache persisted. On examination, CSF is often normal but may show mild elevation of the total protein content or an increased cell count. These CSF abnormalities may be due to hyperemia of the brain and meninges, resulting from low CSF pressure (Rando & Fishman, 1992). Subsequently, this results in diapedesis of red cells into subarachnoid space. Low CSF pressure leads to change in hydrostatic and oncotic pressure across the venous sinus and arachnoid villi, especially if secondary to leak from disruption of the meninges, may also facilitate the passage of serum protein into the CSF (Marcelis & Silverstein, 1990). Pleocytosis also represents local inflammatory reaction to the CSF leakage site or, in some cases, a response to the presence of red blood cells (Rando & Fishman, 1992).

Schaltenbrand proposed three mechanisms to explain the pathophy-siology of SIH in 1938 : 1) diminished CSF production, 2) increased CSF absorption, and 3) CSF leakage (Lavadie *et al*, 1976; Gaukroger & Brownridge, 1987; Fernandez, 1990; Marcelis & Silberstein, 1990; Rando & Fishman, 1992; Pannullo *et al*, 1993; Schievink *et al*, 1994; Rendowden *et al*, 1995). Radionuclide cisternography may reveal abnormal findings of early uptake in the bladder and kidneys as well as spontaneous CSF leakage from subarachnoid space and subsequently may help in choosing a more effective treatment (Lavadie *et al*, 1976; Chung *et al*, 1996). It can also help us explain the pathophysiology of SIH. If SIH is caused by decreased production of cerebrospinal fluid, then cisternography would likely show slow progression of the isotope and high concentration which remained for a prolonged period of time at the injection site (Lavadie *et al*, 1976; Marcelis & Silberstein, 1990; Chung *et al*, 1996). Radionuclide cisternography usually shows a combination of normal flow with rapid loss of radioactivity without evidence of CSF leakage due to isotope leakage which is so small as to be under the scanning resolution power (Lavadie *et al*, 1976; Schievink *et al*, 1994). Alternatively, the radioisotope would have to be escape directly into the epidural paravertebral venous plexus, which would rapidly carry them away, and it would remain undetectable until reconcentrated in the urinary bladder (Schievink *et al*, 1994). The combination of normal isotope flow with systemic activity and no evidence of leak is most consistent with CSF hyperabsorption (Marcelis & Silberstein, 1990; Chung *et al*, 1996). This abnormal pattern, however, does not always differentiate between a CSF leak and CSF hyperabsorption. Reabsorption of CSF does not occur below the gradient pressure of 60 mmH_2O between the subarachnoid space and drainage venous pressure. Thus, we consider CSF hypera-

bsorption to be an untenable hypothesis in SIH where the CSF pressure is, by definition, less than 60 mmH_2O and is often unmeasurable (Lavadie *et al*, 1976; Rando & Fishman, 1992).

The underlying defect, causing CSF leak, such as nerve root sleeve, spinal epidural cyst, Tarlov's cyst, or arachnoid diverticulum, is seldom identified and may be more susceptible to small tears, resulting from a trivial fall, a sneeze, or strain (Murros & Fogelholm, 1983; Rando & Fishman, 1992; Schievink *et al*, 1994; Rendowden *et al*, 1995). Myelography or spinal MRI helps diagnose these disorders (Rando & Fishman, 1992; Bang *et al*, 1997).

Biopsy revealed extensive fibrocollagenous proliferation in the leptomeninges without showing evidence of inflammation. Dural border cell layers of the dura mater may demonstrate nonspecific granulation tissue with mild inflammatory change which corresponds to the pachymeningeal gadolinium enhancement zone of the MRI (Good & Ghobrial, 1993; Bang *et al*, 1997; Niwa *et al*, 1997). This nonspecific pathologic change may be caused by low intracranial pressure, altered flow dynamics of CSF in the subarachnoid space, subdural fluid collections that occur in many (but not all) cases, or venous dilatation, resulting from reduced CSF volume and the cause of pathologic change is unknown at present (Good & Ghobrial, 1993).

Brain CT may reveal slit-shaped ventricle, tight basal cistern and scant CSF over the cortex, filling fit well with diffuse brain edema. With resolution of the headache, abnormal CT scan findings were normalized (Murros & Fogelholm, 1983).

Gadolinium-enhanced MRI revealed an extraordinary degree of dural enhancement, striking displacement of the optic chiasm, flattening of the pons along the clivus, and downward displacement of the iter and the cerebellar tonsil (Hochman *et al*, 1992; Rando & Fishman, 1992; Horton & Fishman, 1994; Reich *et al*, 1993; Rendowden *et al*, 1995; Kinoshita *et al*, 1997). Subdural fluid effusion may have occurred as a secondary phenomenon in SIH and may arise presumably by the rupture of bridging veins when the brain pulls away from the dura as CSF volume decreases (Pavlin *et al*, 1979; Sipe *et al*, 1981). In our patients, brain MRI showed typically diffuse, continuous dural enhancement with slight downward displacement of the brain and cerebellar tonsils. Diffuse meningeal enhancement was secondary to 1) compensatory meningeal hyperemia due to low CSF pressure which Schaltenbrand had suggested, 2) underlying inflammation of the pachymeninges, 3) a nonspecific meningeal reaction to low CSF pressure (Hochman *et al*, 1992; Chung *et al*, 1996). Meningeal enhancement has been described as the involvement of peripheral meninges, including membranes along cerebral and cerebellar convexities, interhemispheric fissure, and tentorium but not involving the brainstem or the depths of the cortical sulci (Pannullo *et al*, 1993). Meningeal enhance-ment and brain shifting may either resolve completely or improve

considerably when the headache resolves (Pannullo *et al*, 1993). Awareness of this condition, its MRI appearance, and pattern of meningeal enhancement is important to avoid unnecessary investigation. Diffuse meningeal enhan-cement may be seen with malignant meningitis and infectious or granulo-matous meningitis, and may also be seen after craniotomy after which it may persist for years and ventricular shunting (Hochman *et al*, 1992; Good & Ghobrial, 1993; Christine *et al*, 1997). It has also been reported after subarachnoid haemorrhage and venous sinus thrombosis (Hochman *et al*, 1992; Christine *et al*, 1997). It rarely occurs secondary to lumbar puncture. Additi-onally, dilatation of epidural veins was reported which was secondary to low spinal CSF pressure by a similar mecha-nism which occurs intracranially (Chung *et al*, 1996).

Postural headaches usually resolve spontaneously or with strict bed rest, oral or parenteral fluid and salt, and use of abdominal binder (Spielman, 1982). The headache may, however, take up to several months to resolve and analgesics are seldom useful. In the refractory case, an epidural blood patch may be of bene-ficial, even if a CSF leak has not been demonstrated as in our two patients, and may be required (Reno-wden *et al*, 1995). We treated our patients with one or two trials of epidural blood patch. Autologous blood injected into the epidural space produces an organized clot that could work effectively as a gelatineous tampo-nade, preventing further CSF leakage, thus favoring sealing of the dural rent (Wilton *et al*, 1986). The organ-ization of this clot occurs over several days and therefore may accout for the prolonged pain relief (Rando & Fishman, 1992; Christine *et al*, 1997). However, this mechanism has been questioned on the basis of recurrence of orthostatic heada-che four to six months after a successful blood patch (Marcelis & Silverstein, 1990). The rapid recovery from headache seen immediately after the infusion of the blood or a bolus of saline must occur by some other mecha-nism, such as compre-ssion of dural sac, with an increment in CSF pressure, which may serve as a signal that deactiv-ates low CSF pressure headache, possibly by antagonizing adenosine receptors. Szeinfeld *et al* (1986) reported that 12 to 15 ml of blood was sufficient to increase the pressure in the suba-rachnoid space and seal the dural rent and spread cephalad and caudal to involve 8 to 10 spinal segments (Szeinfeld *et al*, 1986). An epidural blood patch has proved successful without identifying the epidural spinal leak site, reflecting the ability of epidural blood to move freely from the lumbar to the cervical region (Szeinfeld *et al*, 1986; Rando & Fishman, 1992; Fishman & Dillon, 1993; Christine *et al*, 1997).

If the effectiveness of epidural blood patch is due to an increase in subarac-hnoid pressure then the precise location of the patch may be unimportant (Rando & Fishman, 1992). One interspace below seems more appropriate because of the greater cephalad spread of blood in the

epidural space (Szeinfeld *et al*, 1986). Even if such stringent recommendations cannot be followed, localization of a CSF leak along the neuraxis could suggest where to place the epidural blood patch in a patient with SIH (Rando & Fishman, 1992). Success of the epidural blood patch in post-dural puncture headache depends on close approximation of the injection of blood to puncture site (Szeinfeld *et al*, 1986). Our experience has demonstrated that lumbar placement of the epidural blood patch can be effective even when the site of leakage is unknown. Side effect includes transient back stiffness and occasional temporary symptoms of nerve root irritation (Fernandez, 1990; Wilton *et al*, 1990).

Other treatment modalities that may prove beneficial include a short course of corticosteroids or epidural or intrathecal saline infusion (Murros & Fogelholm, 1983; Fernandez, 1990; Pleasure *et al*, 1998). In addition, it has been reported that theophilline and caffeine-producing vasoconstriction are effective in SIH (Fernandez, 1990). We suggest that this procedure should be considered as a treatment of choice in SIH without identification of CSF leakage site or in other circumstances where a lumbar puncture leads to persistent postural headache(eg. myelography). If these conservative treatments fail, and there are underlying defects such as a dural tear or complication of subdural hematoma, surgical repairment is recommended (Schievink *et al*, 1994; Renowden *et al*, 1995).

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