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Subarachnoideal blood spread following epidural blood patch given to treat spontaneous intracranial hypotension: Can it cause neurological complications?

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ABSTRACT

Objective: (1) To determine the frequency of subarachnoid blood spread following epidural blood patch (EBP) in a cohort of subjects with spontaneous intracranial hypotension (SIH). (2) To describe the outcome of these patients.
Patients and methods: In a cohort of 106 patients exhibiting SIH, spiral spinal CT scans were obtained post-lumbar EBP and neuroradiological data was reviewed for evidence of subarachnoideal bleeding.
Results: Subarachnoideal blood spread was detected on spinal CT scans following EBP in 9 of 106 patients with SIH. All patients exhibited a complete recovery and no neurological complications were observed.
Conclusions: A low incidence of subarachnoideal blood spread was observed following EBP given to treat SIH. Instances of subarachnoideal blood spread were not associated with neurological complications or altered efficacy of the EBP procedure.

1. Introduction

Spontaneous intracranial hypotension (SIH) is a syndrome resultant from cerebrospinal fluid (CSF) leakage and a subsequent loss of CSF pressure. SIH produces a range of symptoms, but most notably manifests with orthostatic headache (other symptoms listed in Table 1) [1]. Lumbar autologous epidural blood patch (EBP) is a procedure that has been primarily used for the treatment of post-lumbar puncture headache [6], but in recent years EBP has become a reliable treatment (and often the first line treatment) for orthostatic headache due to SIH [3].

Complications from EBP are quite rare but can include pneumocephalus [7], spinal subdural haematoma or iatrogenic subarachnoid haemorrhage [8,9], with secondary communicating hydrocephalus and arachnoiditis [10]. To date, it is not known whether neurological complications can arise from subrarachnoidal blood spread following EBP. Here we report the frequency of subarachnoideal blood spread in a cohort of SIH patients and describe the outcome of 9 patients with intrathecal blood spread following lumbar autologous EBP.

2. Patients and methods

We evaluated a population of 106 patients over a 20-year period between April 1992 and May 2012. Patients were referred to us for orthostatic headache from SIH (according to the International Classification of Headache Disorder 2nd ed, 2004 criteria for headache attributed to spontaneous or idiopathic low CSF pressure [11]) and were treated with lumbar EBP. For 23 cases observed between the years of 1992–2004 (prior to the development of the 2004 diagnostic criteria), lumbar puncture was performed to measure the CSF opening pressure, which was low in 18 of those cases (78%) and normal in 5 cases (22%). The later also demonstrated orthostatic headache and diffuse pachymeningeal enhancement on brain MRI typical of SIH.

Prior to EBP, all patients had failed to recover from SIH during a conservative non-invasive treatment period that ranged from 9 days to 13 months (median 62 days), where treatment consisted of one or more of the following: bed rest, over-hydration, caffeine, non-steroidal anti-inflammatory drugs, steroids, antidepressants, and analgesic administration. All patients gave full informed consent before treatment, and patient data (case series, clinical,

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Table 1
Symptoms associated to SIH other than headache [1].

- Spinal pain
- Nausea with or without emesis
- Diplopia, horizontal and due to unilateral or bilateral 6th cranial nerve palsy
- Diplopia due to 3rd or 4th cranial nerve palsy or both(much less common) [2]
- Cochleovestibular manifestations
- Photophobia, visual blurring
- Upper limb numbness, paresthesias
- Gait unsteadiness
- Facial numbness, vague paresthesias, or weakness
- Change in level of consciousness (from lethargy to coma) [3,4]
- Personality change, memory decline, apathy, frontotemporal dementia-like presentation
- Movement disorders: choreiform, parkinsonism, torticollis, tremor
- Bibrachialamyotrophy mimicking motor neuron disease [5]
- Galactorrhea
- Meniere-like syndrome
- Upper limb radiculopathy
- Trouble with bowel or bladder control

diagnostic, therapeutic and follow-up information) was catalogued in an Excel database.

A standardized protocol for lumbar autologous EBP was observed as follows: patients maintained a 30° Trendelenburg position from one hour prior to the EBP procedure through the duration of the procedure and for 24 h after the procedure. The EBP was administered under aseptic operatory conditions by two experienced anaesthesiologists. With the patient in a prone position, local aesthetic was administered followed by placement of an 18G Tuohy needle at the L2–L3, L3–L4, or L4–L5 level. Loss of resistance to air injection technique was used to localize the epidural space and appropriate needle placement was confirmed using fluoroscopic guidance: injection of 3 mL of contrast medium (Iopamidol) into epidural space (Fig. 1). Autologous blood taken from a cannulated big calibres vein mixed with 5 mL contrast medium was injected slowly into the L2–L3, L3–L4, or L4–L5 epidural space until the development of patient discomfort (low back pain, headache, or nausea). Injection volumes ranged from 26 mL to 40 mL (high volume EBP). At 30 min post-procedure, a spiral spinal CT scan was performed to observe the spread of the blood patch into the epidural space. Spiral spinal CT confirmed correct execution of the procedure. Patients were asked to refrain from strenuous exercise for 2 weeks post-procedure. Follow-up observations were conducted through clinical evaluation at 1 and 3 months post-EBP and via telephone evaluation after 3 months. Mean follow-up time was 8 months (range 6–24 months).

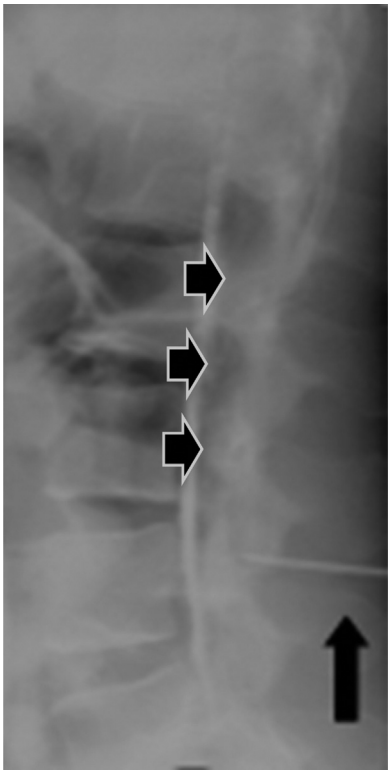

Fig. 1. Epidurography showing right needle placament (black arrow) and spread of contrast medium in the epidural space (arrow heads).

3. Results

In total, 100% (106/106) patients indicated relief of orthostatic headache at 24 h post-EBP. Complete recovery was obtained in all patients following 1 (90%), 2 (4%) or 3 (6%) EBP procedures. The actual dural leak was localized in 55% of patients using spinal MRI (37 patients), MRI myelography (16 patients), CT myelography (3 patients) or radioisotope cisternography (2 patients). Spinal MRI and MRI myelography was unsuccessful in localizing the spinal leak in 48 patients (45%).

Neuroradiological evidence of subarachnoideal blood spread following EBP was observed in 9 of 106 patients (8%), including 5 women and 4 men (mean age 44 years, range 35-58 years). For 1 of 9 patients with subarachnoideal blood spread, accidental dural puncture was reported during the EBP procedure and confirmed by CSF leak from the Tuohy needle; the other 8 EBP procedures reported no dural puncture and correct needle placement as confirmed by epidurography (Fig. 1).

Of 9 patients with subarachnoideal blood spread, 3 patients reported headache triggers from mild trauma ($n = 1$), sneezing ($n = 1$), and cervical manipulation ($n = 1$). Other manifestations included changes in hearing and tinnitus ($n = 6$) and diplopia ($n = 3$). All patients appeared normal on brain CT scan and showed diffuse pachymeningeal enhancement on brain MRI. Spinal MRI (6 patients) showed CSF collection at the cervical level ($n = 1$) and at the cervicothoracic junction ($n = 2$). Spinal taps performed in 4 patients demonstrated a low CSF opening pressure. The level of the CSF leak was determined in 3 patients however the actual site of the leak was undetermined.

None of the 9 patients with subarachnoideal bleeding (indicated by the presence of blood mixed with Iopamidol in subarachnoideal space, Fig. 2) developed neurological complications and orthostatic headache was completely resolved following 24 h of the Trendelemburg position post-EBP.

4. Discussion

In recent years, spontaneous intracranial hypotension (SIH) has become recognized as a substantial contributor to headache in patients. Intracranial hypotension typically results from a CSF leak-that, in turn, leads to a decrease in CSF volume. Older theories of CSF over-absorption or under-production have not been substantiated. A decrease in CSF volume rather than decrease in CSF pressure is the core pathogenetic factor (independent variable), while CSF pressures and clinical or imaging changes are variables dependent on CSF volume. CSF opening pressure is normal in a substantial minority of patients. Essentially, all cases of spontaneous intracranial hypotension result from spontaneous CSF leaks, typically at the spinal level, particularly the thoracic spine or cervicothoracic junction. Preexisting dural weakness (meningeal diverticula, dilated nerve root sleeves, ectasia of dural sac), likely related to a heritable disorder of the connective tissue matrix (Marfan syndrome or marfanoid features, joint hypermobility, retinal detachment at young age, personal or family history of arterial dissections,