A spontaneous CSF leak is a condition in which CSF spontaneous leaks from the subarachnoid space to the extradural space. The major symptoms of a CSF leak are orthostatic headaches with or without neck stiffness, nausea, photophobia, dizziness, tinnitus, and hearing impairment. Spontaneous CSF leaks are diagnosed by detection of extradural CSF collections by use of CT myelography, MRI, and/or radioisotope cisternography.

The treatment of CSF leaks is both conservative and invasive. Epidural blood patch (EBP) is indicated if improvement does not result from bed rest and intravenous infusion. Detecting the leak site and determining the leakage detection on CT myelography for targeted epidural blood patch in spontaneous cerebrospinal fluid leaks: calcified or ossified spinal lesions ventral to the thecal sac

Clinical article

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Object. The purpose of this study was to describe significant CT myelography findings for determination of the leak site and outcome of targeted epidural blood patch (EBP) in patients with spontaneous CSF leaks. The patients received targeted EBP on the basis of CT myelography assessments.

Methods. During 2005–2013, spontaneous CSF leaks were diagnosed for 12 patients with orthostatic headaches. The patients received targeted EBP on the basis of CT myelography assessments.

Results. Computed tomography myelograms revealed ventral extradural collection of contrast medium distributed over multiple spinal levels (average 16 levels). Intraforaminal contrast medium extravasations were observed at multiple spinal levels (average 8.2 levels). For 8 (67%) of 12 patients, spinal lesions were noted around the thecal sac and included calcified discs with osteophytes, an ossified posterior longitudinal ligament, and an ossified yellow ligament; lesions were mostly located ventral to the thecal sac and were in close contact with the dura mater. The levels of these spinal lesions were considered potential leak sites and were targeted for EBP. For the remaining 4 patients who did not have definite spinal lesions around the thecal sac, leak site determination was based primarily on the contrast gradient hypothesis. The authors hypothesized that the concentration of extradural contrast medium would be the greatest and the same as that of intradural contrast medium at the leak site but that it would decrease with increased distance from the leak site according to the contrast gradient. Epidural blood patch was placed at the level of spinal lesions and/or of the greatest and same concentration of contrast medium between the intradural and extradural spaces. For 10 of the 12 patients, the orthostatic headaches decreased significantly within a week of EBP and disappeared within a month. For the remaining 2 patients, headaches persisted and medical treatment was required for several months. For 3 patients, thick chronic subdural hematomas caused severe headaches and/or disturbed consciousness because of the mass effect of the hematomas, which were removed by bur hole drainage surgery. For 1 patient, bur hole drainage before EBP on the day of admission to hospital resulted in subdural tension pneumocephalus. The patient’s headache immediately disappeared after EBP, and the hematoma did not recur. The other 2 patients underwent EBP followed by bur hole drainage, which resulted in improvements and disappearance of the hematomas. Over the follow-up period (mean 39 months), no CSF leaks or chronic subdural hematomas had recurred in any patient after EBP; by the final follow-up visit, all patients had returned to their jobs.

Conclusions. The most significant finding of this study was that spinal ventral calcified or ossified lesions, which may be associated with a dural tear, were present in approximately 70% of patients. Targeted EBP to these lesions resulted in good outcomes.

Abbreviations used in this paper: CSDH = chronic subdural hematoma; EBP = epidural blood patch.
Detecting spontaneous CSF leaks with CT myelography

spinal level for EBP can be difficult because extradural fluid is often extensively distributed at multiple spinal levels and the exact leak site is unclear in many cases.

Previous studies reported that EBP was performed at lumbar levels, which resulted in a cure rate of 30%–40%.1 Lumbar EBP with the patient in the Trendelenburg position or lumbar EBP with a long vascular catheter has been used to spread the injected blood over a large area in the cranial direction along the spinal epidural space.4,11 However, for 50% of patients, nontargeted lumbar EBP requires 2 or more injections with relatively larger amounts of blood (20–75 ml).

As an alternative method for nontargeted lumbar EBP, targeted EBP has recently been introduced.2 The cure rate for targeted EBP is 70%–100% (which is higher than that for nontargeted lumbar EBP), and the amount of blood injected is smaller; however, targeted EBP requires identification of the exact leak site. Although the use of targeted EBP is increasingly being reported, previous studies have not described in detail how to determine the leak site and target the spinal level for EBP.8,16–18

A common cause of CSF leaks is considered to be a ventral dural defect or meningeal diverticulum at the dural root sleeves.9 In terms of leakage detection, the diagnostic method with the highest sensitivity is CT myelography.7,17 The purpose of the study reported here was to describe significant CT myelographic findings with regard to leakage detection and outcomes of targeted EBP.

Methods

This study protocol was approved by the Institutional Review Board at Tokyo Metropolitan Neurological Hospital (no. 24–4). Because this was a retrospective and noninvasive study, written patient informed consent was not obtained; instead, a public notice that provided information on this study was placed on the Tokyo Metropolitan Neurological Hospital website.

Patient Selection

Study participants were 12 consecutively selected patients (age range 32–61 years, male/female ratio 6:6) who had orthostatic headaches and for whom spontaneous CSF leaks were diagnosed during 2005–2013 (Table 1). The radiological diagnosis of spinal CSF leaks was based primarily on the findings of CT myelography (CT myelographic presence of extradural contrast medium, which was confirmed to have been injected into the subarachnoid space through lumbar puncture).15

Previously, when we performed CT myelography for patients with spinal stenosis, extradural contrast medium was not observed unless it had been wrongly injected into the extradural space. Therefore, it is reasonable to assume that contrast medium leaks from the subarachnoid space into the extradural space through the leak site if there is no technical failure in the lumbar puncture. We do not consider brain MRI findings of intracranial hypotension to be essential. Over the course of the present study, 8 patients were excluded: 2 patients with a history of traffic accidents, 1 patient with a psychogenic disorder, 2 patients in whom extradural fluid collections were suspected on MR imaging but not verified on CT myelograms, 2 patients for whom headaches improved with bed rest and intravenous infusion, and 1 patient in whom EBP was placed on the basis of MRI findings alone.

CT Myelography Technique

We injected a total volume of 15 ml contrast medium (Omnipaque, Daiichi Sankyo) into the subarachnoid space via lumbar puncture with a 21-gauge spinal needle. After the injection, patients underwent thin-cut CT from the lumbar to midthoracic levels. We confirmed that the contrast was not wrongly injected into the extradural space in the lumbar region. Patients were then required to rest in bed, supine, for 1 hour. Patients again underwent thin-cut CT from the midthoracic to the craniovertebral junctions. CT scans were performed on a 4-channel helical CT scanner (Toshiba). The imaging parameters for CT were as follows: electrical voltage 120 kV, electrical current 200 mA, and slice thickness, 4 mm.

CT Myelography Assessment

We determined potential leak sites and targeted the spinal levels for EBP on the basis of a comprehensive assessment of CT myelography findings as described below and the concentration gradient of contrast medium. Computed tomography myelography findings included the following 3 points: spinal lesions around the thecal sac (Fig. 1), extradural collection of contrast medium, and intraforaminal contrast medium extravasations. Regarding the concentration gradients of extradural and intradural contrast medium, we hypothesized that the concentration of extradural contrast medium would be the greatest and the same as that of intradural contrast medium at the leak site but would decrease with increased distance from the leak site according to the contrast gradient (Fig. 2). Epidural blood patch was placed at the site with the greatest and the same concentration of contrast medium between extradural and intradural spaces.

EBP Technique

Patients were placed prone, and a 17-gauge spinal needle for extradural anesthesia was inserted at the targeted levels. Using fluoroscopic guidance, we gradually advanced the needle between the spinous processes and checked whether the tip of the needle was at the extradural space. We manually injected a small amount of the mixture of autologous blood and contrast medium at a rate of 4:1. To spread the blood over the extradural space, we injected it in 4 directions (rostral, right, left, and caudal sides). We then confirmed whether the injected blood spread around the thecal sac.

Data Analysis

We analyzed the following radiographic data: the distribution of extradural contrast medium collections and intraforaminal contrast medium extravasation on CT myelograms, spinal lesions around the thecal sac, targeted spinal levels of EBP, and brain MRI findings. We assessed clinical outcomes regarding headaches and other symptoms before EBP, 1 and 6 months after EBP, and at
<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yrs), Sex</th>
<th>Symptoms Associated w/ CSF Leaks</th>
<th>Extent of Extradural Contrast Medium Collections†</th>
<th>Intraforaminal Contrast Medium Extravasations†</th>
<th>Spinal Levels of Calcified or Ossified Lesions</th>
<th>Target of EBP (no. of injections)</th>
<th>Amount of Blood Injected (ml)</th>
<th>Complications Associated w/ EBP</th>
<th>Duration of Complete Symptom Relief</th>
<th>Follow-Up Period (mos)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>32, F</td>
<td>orthostatic HA, neck pain, dizziness</td>
<td>C2–T9 ventral</td>
<td>C4–T4</td>
<td>C7–T1, T1–2</td>
<td>T1–2 (1)</td>
<td>8</td>
<td>none</td>
<td>1 mo</td>
<td>102</td>
</tr>
<tr>
<td>2</td>
<td>38, M</td>
<td>orthostatic HA, vomiting</td>
<td>C5–T12 ventral</td>
<td>C5–T1</td>
<td>T-2</td>
<td>C7–T1 (1)</td>
<td>10</td>
<td>none</td>
<td>6 mos</td>
<td>32</td>
</tr>
<tr>
<td>3</td>
<td>53, F</td>
<td>orthostatic HA</td>
<td>C2–T7 ventral</td>
<td>C4–T1</td>
<td>NA</td>
<td>C6–7 (1)</td>
<td>10</td>
<td>none</td>
<td>1 mo</td>
<td>74</td>
</tr>
<tr>
<td>4</td>
<td>45, M</td>
<td>orthostatic HA, neck pain, dizziness, hearing impairment</td>
<td>C3–T12 ventral</td>
<td>C3–T1</td>
<td>NA</td>
<td>C6–7 (1)</td>
<td>12</td>
<td>none</td>
<td>2 wks</td>
<td>71</td>
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<tr>
<td>5</td>
<td>32, M</td>
<td>orthostatic HA</td>
<td>C1–T12 ventral</td>
<td>C2–T1</td>
<td>NA</td>
<td>C6–7 (1)</td>
<td>12</td>
<td>none</td>
<td>1 wk</td>
<td>1</td>
</tr>
<tr>
<td>6</td>
<td>61, F</td>
<td>orthostatic HA</td>
<td>C3–T12 ventral</td>
<td>C4–T3</td>
<td>T-2</td>
<td>T1–2 (1)</td>
<td>12</td>
<td>none</td>
<td>1 mo</td>
<td>50</td>
</tr>
<tr>
<td>7</td>
<td>46, M</td>
<td>orthostatic HA, neck pain, ear fullness</td>
<td>C3–T10 ventral</td>
<td>C4–T1</td>
<td>NA</td>
<td>T1–2 (1)</td>
<td>10</td>
<td>none</td>
<td>2 wks</td>
<td>46</td>
</tr>
<tr>
<td>8</td>
<td>49, F</td>
<td>orthostatic HA, neck pain, hearing impairment, tinnitus</td>
<td>T1–5 dorsal, T-6 ventral, T7–12 dorsal</td>
<td>T5–12</td>
<td>T-7</td>
<td>T6–7 (2)</td>
<td>5</td>
<td>technical failure of the 1st EBP</td>
<td>7 mos, except for tinnitus</td>
<td>38</td>
</tr>
<tr>
<td>9</td>
<td>40, M</td>
<td>orthostatic HA, neck pain, nausea, dizziness</td>
<td>C3–7 dorsal, T1–2 ventral, T3–L1 dorsal</td>
<td>C7–T3</td>
<td>T1–2</td>
<td>T2–3 (1)</td>
<td>8</td>
<td>none</td>
<td>1 mo</td>
<td>16</td>
</tr>
<tr>
<td>10</td>
<td>43, F</td>
<td>orthostatic HA, vomiting, ear fullness</td>
<td>C6–T12 ventral</td>
<td>C7–T7</td>
<td>T10–11</td>
<td>C6–7, T10–11 (2)</td>
<td>5, 8</td>
<td>none</td>
<td>1 wk</td>
<td>18</td>
</tr>
<tr>
<td>11</td>
<td>41, F</td>
<td>orthostatic HA, neck pain, dizziness</td>
<td>C3–T12 ventral</td>
<td>C5–T6</td>
<td>T3–4</td>
<td>T1–2 (1)</td>
<td>8</td>
<td>none</td>
<td>1 mo</td>
<td>8</td>
</tr>
<tr>
<td>12</td>
<td>39, M</td>
<td>orthostatic HA, neck pain, vomiting, tinnitus</td>
<td>C2–T12 ventral</td>
<td>C3–T1</td>
<td>T2–3</td>
<td>T1–2 (1)</td>
<td>12</td>
<td>none</td>
<td>2 wks</td>
<td>6</td>
</tr>
<tr>
<td>average</td>
<td>43</td>
<td>NA</td>
<td>16 spinal levels, ventral dominant</td>
<td>8.2 spinal levels</td>
<td>NA</td>
<td>NA</td>
<td>9.1</td>
<td>NA</td>
<td>NA</td>
<td>39</td>
</tr>
</tbody>
</table>

* HA = headache; NA = not applicable.
† Determined by CT myelography.
Detecting spontaneous CSF leaks with CT myelography

Fig. 1. Axial CT myelograms showing spinal lesions around the thecal sac, including calcified discs with osteophytes (arrows in panels A, B, and E–H), an ossified posterior longitudinal ligament (arrow in panel D), or an ossified yellow ligament (arrow in panel C). These spinal lesions were in close contact with the dura mater and were determined as potential leak sites and targeted for EBP. A: Patient 1. B: Patient 2. C: Patient 6. D: Patient 8. E: Patient 9. F: Patient 10. G: Patient 11. H: Patient 12.

Fig. 2. A and B: Illustrations showing the contrast gradient hypothesis. C–F: Patient 4. Axial CT myelograms showing ventral extradural contrast collections and intraforaminal contrast extravasations at C6–T1. The concentration of extradural contrast medium would be the same as that of intradural contrast medium at the leak site (A, B, and D, arrows) but would decrease with increased distance from the leak site according to the contrast gradient (C and F). Arrowheads indicate the dura mater of the thecal sac, and asterisks indicate the spinal cord.
the final follow-up visit on the basis of medical records and telephone interviews. We also evaluated the work status (full-time work, part-time work, or unemployed) of patients before the onset of headaches, before EBP, 1 month after EBP, and at the final follow-up visit. More than 6 months of follow-up data to determine the efficacy of the treatments (mean follow-up 39 months, range 1–102 months) were obtained for 11 (92%) of 12 patients.

Results

The radiological findings and clinical outcomes of targeted EBP and chronic subdural hematomas (CSDHs) in patients with spontaneous CSF leaks are summarized in Tables 1 and 2.

Symptoms Associated With CSF Leaks and CSDHs

At the time of hospital admission, patients reported the following signs and symptoms: orthostatic headaches (100% of patients); neck pain (58%); nausea and vomiting (33%); dizziness (33%); and auditory symptoms (42%), including hearing impairment, tinnitus, and ear fullness (Table 1). Of 8 patients with thick CSDHs, 3 had severe persistent headaches and/or disturbed consciousness resulting from the mass effect of the hematomas (Table 2). No patients had hemiparesis.

Radiological Findings

According to CT myelograms, ventral extradural collection of contrast medium was distributed over multiple spinal levels (average 16), from the upper cervical to lower thoracic regions. Intraforaminal contrast extravasations were observed at multiple spinal levels (average 8.2), from the midcervical to upper thoracic regions (Fig. 2). Spinal lesions were observed around the thecal sac for 8 of 12 patients (Patients 1, 2, 6, and 8–12) and included calcified discs with osteophytes, an ossified posterior longitudinal ligament, and an ossified yellow ligament. These spinal lesions were in close contact with the dura mater (Fig. 1). Brain MR images of 12 patients indicated dural enhancements for 9 patients and CSDHs for 8 patients; both were associated with intracranial hypotension. Of the 8 patients with CSDHs, hematomas were bilateral for 5 patients.

Clinical Outcomes

For 7 of the 8 patients with spinal lesions (Patients 1, 2, 6, 8, 9, 11, and 12), the levels of spinal lesions were determined as potential leak sites and were targeted for EBP (Fig. 1). Because most spinal lesions were in the upper thoracic region and some were in the lower thoracic region, the leakage sites were determined and targeted for EBP accordingly (Fig. 3, left). The spinal levels of these lesions were the same as one of the levels of greatest extradural collection of contrast medium. For 1 of these patients (Patient 10), a discrepancy was observed between the level of the ventral calcified disc with osteophytes and the levels of the greatest ventral extradural collection of contrast medium (Fig. 4). This patient underwent targeted EBP at the level of C6–7, the location of the greatest ventral extradural collection of contrast medium and intense intraforaminal contrast extravasation; this procedure failed. The patient then underwent targeted EBP at the level of T10–11, the location of the calcified disc with osteophytes; this procedure was successful.

Determination of the leak site in patients without spinal lesions (Patients 3–5 and 7) was based primarily on the contrast gradient hypothesis: the comparison of concentrations of contrast medium between the intradural and extradural spaces on CT myelograms. Epidural blood patch was placed at the site with the greatest and the same concentration of contrast medium between the intradural and extradural spaces (Fig. 2D). The average amount of injected blood was 9.1 ml.

We hypothesized that the concentration of extradural contrast medium would be the greatest and the same as that of intradural contrast medium at the leak site but would decrease with increased distance from the leak site according to the contrast gradient (Fig. 2). The leak sites were commonly determined in the cervico–thoracic junction (right).
TABLE 2: Summary of the radiological findings and clinical outcomes for patients with CSDH and spontaneous CSF leaks

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>CSDH</th>
<th>Other Brain MRI Findings</th>
<th>Neurological Deficit Associated w/ CSDH*</th>
<th>Treatment of CSDH</th>
<th>Time of CSDH Disappearance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>bilat thin CSDH</td>
<td>dural enhancement</td>
<td>none</td>
<td>none</td>
<td>1 mo after EBP</td>
</tr>
<tr>
<td>2</td>
<td>none</td>
<td>fluid collections, dural enhancement</td>
<td>none</td>
<td>bur hole drainage 1 mo after EBP because of development of hematoma</td>
<td>1 mo after drainage</td>
</tr>
<tr>
<td>3</td>
<td>bilat thin CSDH</td>
<td>none</td>
<td>none</td>
<td>1 mo after EBP</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>lt thick CSDH</td>
<td>severe persistent HA</td>
<td>1) bur hole drainage 1 mo before EBP resulted in recurrence; 2) bur hole drainage just before EBP resulted in tension pneumocephalus</td>
<td>4 mos after EBP &amp; 2nd drainage</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>lt thin CSDH</td>
<td>dural enhancement</td>
<td>none</td>
<td>1 mo after EBP</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>bilat thick CSDH</td>
<td>dural enhancement</td>
<td>severe persistent HA, disturbed consciousness</td>
<td>bur hole drainage just after EBP</td>
<td>3 mos after EBP &amp; drainage</td>
</tr>
<tr>
<td>7</td>
<td>bilat thick CSDH</td>
<td>dural enhancement</td>
<td>severe persistent HA</td>
<td>1) bur hole drainage 2 wks before EBP resulted in recurrence; 2) bur hole drainage just after EBP</td>
<td>1 mo after EBP &amp; 2nd drainage</td>
</tr>
<tr>
<td>8</td>
<td>none</td>
<td>dural enhancement, asymptomatic pituitary adenoma</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>9</td>
<td>none</td>
<td>dural enhancement</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>10</td>
<td>lt thin CSDH</td>
<td>none</td>
<td>none</td>
<td>1 mo after EBP</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>bilat thin CSDH</td>
<td>dural enhancement</td>
<td>none</td>
<td>none</td>
<td>1 mo after EBP</td>
</tr>
<tr>
<td>12</td>
<td>none</td>
<td>fluid collections, dural enhancement</td>
<td>none</td>
<td>none</td>
<td>1 mo after EBP</td>
</tr>
</tbody>
</table>

* Except for orthostatic HA.
For 10 of 12 patients, signs and symptoms of orthostatic headaches, neck pain, vomiting, dizziness, and auditory symptoms decreased significantly within a week and were completely resolved within a month (Fig. 5, left). For the other 2 patients, intensity of the headaches decreased 50% within a week; however, medical treatment was required for several months. Although 1 patient continued to experience tinnitus at the final follow-up visit, she could engage in full-time work. All patients had returned to full-time or part-time work by the final follow-up visit (Fig. 5, right). No recurrence of CSF leaks or CSDHs after EBP has been reported for any patient during the follow-up period (mean 39 months).

Of 8 patients with thick CSDHs, 3 (Patients 4, 6, and
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7) had severe headaches and/or disturbed consciousness resulting from the mass effect of the hematomas. Of these 3 patients, 2 (Patients 4 and 7) had undergone bur hole drainage at another institution before referral to our hospital; however, both patients had a recurrent CSDH at the time of admission to our hospital. Patient 4 underwent bur hole drainage before EBP on the day of admission to our hospital, which resulted in subdural tension pneumocephalus (Fig. 6). The patient’s headache immediately disappeared after EBP, and the hematoma did not recur thereafter. Patients 6 and 7 underwent EBP followed by bur hole drainage, which resulted in improved condition of these patients and disappearance of the hematomas.

Of 8 patients with thin CSDHs without the mass effect of the hematomas, 5 (Patients 1, 3, 5, 10, and 11) were managed conservatively, and the hematomas spontaneously disappeared within a month after EBP. Subdural fluid collection in Patient 2 required drainage surgery 1 month after EBP because of an enlargement in the hematoma. No CSDH recurrence after EBP was reported for any patient during the follow-up period (mean 39 months).

Discussion

We describe the significant findings of CT myelography used to determine the potential sites of spontaneous CSF leaks and outcomes of targeted EBP. Our main finding was that approximately 70% of patients with spontaneous CSF leaks had spinal calcified or ossified lesions that were located mostly ventral to the thecal sac and in close contact with the dura mater (Fig. 1). In most cases, the spinal level of calcified or ossified lesions was the same as one of the levels of the greatest extradural collection of contrast medium; therefore, the levels of these lesions were determined as potential leak sites and were targeted for EBP. For other patients without definite spinal lesions around the thecal sac, determination of the leak site was based primarily on the contrast gradient hypothesis (Fig. 2). Headaches disappeared within a month after targeted EBP, in which a small amount of blood was injected epidurally (Figs. 3 and 5). To the best of our knowledge, the detailed analysis of CT myelography, which is less invasive and can be performed easily on a routine basis, has yet to be described in the literature. The study reported here raises the following 4 points with regard to the diagnosis and treatment of spontaneous CSF leaks.

First, EBP at the level near the spinal lesions resulted in good outcomes. To our knowledge, our case series represents the first such attempt reported in the literature; our reason for reporting this case series is to draw more attention to spinal calcified or ossified lesions in patients with spontaneous CSF leaks (Fig. 1). A common cause of CSF leaks is considered to be a ventral dural defect.
Although we could not show the exact site of dural defects on CT images, we believe that dural tears may be associated with these spinal lesions. For detecting these lesions, CT myelography has an advantage over MRI because MRI is less sensitive for detecting calcified or ossified lesions.

Second, in the study reported here, the leak sites were commonly detected in the lower cervical and upper thoracic regions. The reason for lower cervical and upper thoracic distribution of potential leak sites may be associated with not only spinal lesions but also dynamic structural changes in the thecal sac in flexion and extension of the cervical spine. Dural tears may be associated with spinal movement and/or extradural lesions in close contact with the dura mater.

Third, intraforaminal extravasation and ventral collection of contrast medium was commonly observed at a number of spinal levels, the incidence of which was markedly higher than that previously reported. Collection of contrast medium around dural root sleeves indicates the presence of a leak point such as a small dural tear or meningeal diverticulum. However, the number of levels of intraforaminal extravasation of contrast medium results in lower specificity for the detection of leaks. For patients without definite spinal lesions, the potential leak site was determined on the basis of the contrast gradient hypothesis: the comparison of concentrations of contrast medium between the intradural and extradural spaces on CT myelography (Fig. 2); the extradural spaces were also targeted for EBP and resulted in good outcomes. Although the spatial and temporal resolution of dynamic CT myelography is higher than that of conventional CT myelography, it requires a multichannel CT scanner with higher radiation exposure and is more invasive, which includes the risk for iatrogenic seizure resulting from rapid ascent of contrast material into the cranial vault. Conventional CT myelography is less invasive and can easily be used in daily practice; therefore, we consider our findings to be beneficial for determining possible leaks and promoting the use of targeted EBP in a large number of institutions.

Fourth, CSDHs associated with intracranial hypotension resulting from CSF leaks can have serious consequences. In this study, for all patients with CSDHs who underwent bur hole drainage surgery before EBP, hematomas recurred within a month. On the other hand, for all patients with CSDHs who underwent bur hole drainage surgery just after EBP, hematomas did not recur and disappeared spontaneously. To the best of our knowledge, tension pneumocephalus as a complication of bur hole drainage surgery in patients with CSDHs resulting from CSF leaks has not been described in the literature. Cerebrospinal fluid leaks should be considered as one of the causes of hematomas in younger patients with CSDHs and no history of trauma.

Patients with CSDHs resulting from spinal CSF leaks should be treated with a combination of EBP and bur hole drainage surgery. If the hematomas are managed with an appropriate approach, they should not recur for a long time thereafter.

The main limitations of this study are the small number of patients examined and the fact that the outcome of the targeted EBP-treated group was not compared with a control nontargeted EBP group. Therefore, other well-designed studies with a larger number of patients are needed.

Conclusions

The most significant finding of this study was that spinal ventral calcified or ossified lesions, which may be associated with a dural tear, were present in approximately 70% of patients. Targeted EBP to these lesions resulted in good outcomes.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Takai. Acquisition of data: Takai, Yoshida. Analysis and interpretation of data: Takai, Yoshida. Drafting the article: Takai, Yoshida. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Takai. Study supervision: Takai, Taniguchi.

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