Prospective assessment of concomitant lumbar and chronic subdural hematoma: is migration from the intracranial space involved in their manifestation?

Clinical article

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Object. Spinal subdural hematomas (SDHs) are rare and some are concomitant with intracranial SDH. Their pathogenesis and etiology remain to be elucidated although their migration from the intracranial space has been suggested. The authors postulated that if migration plays a major role, patients with intracranial SDH may harbor asymptomatic lumbar SDH. The authors performed a prospective study on the incidence of spinal SDH in patients with intracranial SDH to determine whether migration is a key factor in their concomitance.

Methods. The authors evaluated lumbar MR images obtained in 168 patients (125 males, 43 females, mean age 75.6 years) with intracranial chronic SDH to identify cases of concomitant lumbar SDH. In all cases, the lumbar MRI studies were performed within the 1st week after surgical irrigation of the intracranial SDH.

Results. Of the 168 patients, 2 (1.2%) harbored a concomitant lumbar SDH; both had a history of trauma to both the head and the hip and/or lumbar area. One was an 83-year-old man with prostate cancer and myelodysplastic syndrome who suffered trauma to his head and lumbar area in a fall from his bed. The other was a 70-year-old man who had hit his head and lumbar area in a fall. Neither patient manifested neurological deficits and their hematomas disappeared under observation. None of the patients with concomitant lumbar SDH had sustained head trauma only, indicating that trauma to the hip or lumbar region is significantly related to the concomitance of SDH (p < 0.05).

Conclusions. As the incidence of concomitant lumbar and intracranial chronic SDH is rare and both patients in this study had sustained a direct impact to the head and hips, the authors suggest that the major mechanism underlying their concomitant SDH was double trauma. Another possible explanation is hemorrhagic diathesis and low CSF syndrome.

Key Words • concomitant • lumbar spine • MRI • incidental • subdural hematoma • chronic • trauma

Spinal subdural hematomas (SDHs) are rare. Their pathogenesis remains unclear although coagulopathy, anticoagulant therapy, lumbar puncture, vascular malformations, traumatic injuries, low CSF pressure, and idiopathic causes have been suggested.6,15,19 Magnetic resonance imaging is the most useful tool for the diagnosis of spinal SDH.

In rare instances, spinal SDH is coincidental with intracranial SDH.1,4,11,13–22,24–26,28–31,34–45 Although the mechanism of concomitant SDH in the intracranial and lumbar region remains to be established, migration from the intracranial area to the spinal area has been suggested as a key factor.4,9,14,15,22,24,26,28,29,31,36,38,42–44 We postulated that if SDH can migrate from the intracranial area to the lumbar area, patients with intracranial SDH may harbor asymptomatic lumbar SDH. As this issue has not been addressed in detail, we investigated whether the migration of SDH from the intracranial to the spinal area is a key factor in the concomitance of these conditions in trauma patients.

Methods

Patient Eligibility and Experimental Design

Between August 2007 and September 2011, 221 patients with intracranial chronic SDH underwent surgical...
irrigation procedures at the Department of Neurosurgery, Chiba Hokuso Hospital, Nippon Medical School. To evaluate the incidence of concomitant intracranial and lumbar SDH, lumbar MRI was performed within the 1st week after the surgical procedure. We eliminated 53 patients from our prospective study because it was difficult to obtain MRI scans due to restlessness or claustrophobia or because they had undergone pacemaker implantation. Consequently, MR images obtained in 168 patients were available for study. Prior informed consent for participation in this study was obtained from all participants, and the Ethics Committee of Chiba Hokuso Hospital of Nippon Medical School approved the study protocol. The patient group included 125 men and 43 women; their ages ranged from 29 to 90 years (mean 75.6 years). In 32 cases the chronic intracranial SDH was bilateral and in 136 it was unilateral. All images were evaluated independently by experienced spinal surgeons and radiologists.

The study protocol was approved by the Ethics Committee of Nippon Medical School Chiba Hokuso Hospital.

**MRI Technique**

Axial and sagittal T1-weighted (TR 500 msec, TE 11.5 msec) and T2-weighted (TR 2550 msec, TE 102 msec) MR images were obtained on a 1.5-T scanner (GE Signa Excite, GE Healthcare). Sagittal images were obtained from the lower thoracic to the upper sacral level. Axial images were obtained from the T12–L1 level to the L5–S1 level, parallel to the disc plane. The slice thickness was 4 mm for all images; the interslice gap was 2 mm. The field of view was 28 × 28 cm for sagittal images and 18 × 18 cm for axial images.

The cross-sectional area of the dural sac at the lumbar intervertebral spaces was separately measured with Synapse version 3.2.1 (Fuji Film Co.). On axial images the dural sac in the lumbar spine was considered absolutely narrowed if its cross-sectional area was less than 75 mm².

**Statistical Analysis**

Statistical analysis was performed with the Fisher exact test. Differences with p < 0.05 were considered statistically significant.

**Results**

Of the 168 MRI studies evaluated, only 2 (1.2%) revealed concomitant intracranial and lumbar SDH. One was obtained in an 83-year-old man with prostate cancer and myelodysplastic syndrome who suffered trauma to his head and lumbar area in a fall from his bed (Case 1, Fig. 1). The other was obtained in a 70-year-old man with hypertension and diabetes mellitus who had hit his head and lumbar area in a fall (Case 2, Fig. 2). Neither manifested neurological deficits and their hematomas disappeared under observation.

Although earlier studies reported that lumbar SDH arose after direct trauma to the hip or lumbar region, we compared their rate in patients who had sustained head trauma only with the rate in patients with multiple traumas.

Among the 168 patients with intracranial SDH, 75 had no recorded history of trauma or actual injury mechanisms. In 80 cases, the trauma mechanism was known (multiple traumas in 2 cases; head trauma only in 70 cases; a history of falling or slipping in 8 cases), and 13 presented with recurrent intracranial SDH. Of the 70 SDH patients with head trauma only, none presented with concomitant lumbar SDH. On the other hand, both patients with concomitant intracranial and lumbar SDH had a history of head trauma and lumbar and/or hip injury. In our series, 10 patients presented with multiple traumas (n = 2) or had slipped (n = 4) or fallen (n = 4), raising the possibility of their having sustained trauma not only to the head but also to the hip or lumbar region. Our finding that the rate of concomitant SDH was significantly higher in this group than in patients with head trauma only (p < 0.05) indicates that double trauma is a major risk factor for coincidental lumbar SDH.

Of our 168 patients with intracranial SDH, 29 (17.3%) presented with lumbar spinal stenosis, 9 (5.4%) with lumbar disc herniation, 17 (10%) with old compression fractures involving 21 vertebral bodies, and 4 (2.4%) with Schmorl’s nodule, and 1 patient harbored a perineural cyst. The mean lumbar canal area for the 29 patients with lumbar spinal stenosis was 58.3 mm² (range 29 to 74 mm²); their mean age was 80.7 years (range 71 to 91 years).

**Discussion**

Of the 168 MRI studies reviewed, only 2 (1.2%) revealed lumbar SDH. Both patients had sustained a direct impact to the lumbar spine. None of the remaining 166 patients with intracranial SDH manifested concomitant lumbar SDH.

Spinal SDH is a rare condition. Its pathogenesis has been considered secondary to coagulopathies (including SDH arising after anticoagulant therapy), lumbar puncture, vascular malformations, traumatic injuries, low CSF pressure, and idiopathic causes, factors that may be present singly or in various combinations. No etiologic factor for bleeding was identified in 17.3%–29.7% of these patients, and most spinal SDHs were observed in patients with vascular malformations and in patients treated by anticoagulant therapy, although this therapy alone is not thought to elicit spinal hemorrhage. Diagnostic MRI studies show the size and extent of the hemorrhage and its relationship to the spinal cord.

There are few reports on the concomitance of intracranial and spinal SDH. The etiology remained unclear in the 35 previously reported cases (Table 1). Of these cases, 3 involved patients who had undergone ventriculoperitoneal (VP) shunt placement, 3 were postcraniotomy, and 17 were posttrauma, and in 12 cases the SDH arose spontaneously. Leakage of intracranial SDH fluid into the spinal subdural space may be involved in patients who manifest spinal SDH after VP shunt placement. Low
Concomitant subdural hematomas

CSF pressure is a risk factor for spinal SDH in the absence of intracranial SDH extension, and low CSF pressure syndrome elicited by the overflow of CSF in the VP shunt may result in concomitant hemorrhages.

As all spinal SDH in patients who had undergone craniotomy were identified within 1 week after the operation, these hematomas may be attributable to the gravity-induced downward movement of blood from the cranial subdural space. However, no earlier reports identified concomitant residual hematomas in the subdural space and the cervical or thoracic spine. Therefore, low CSF pressure due to excessive surgical CSF drainage may have played an important role in the development of spinal SDH. Spinal SDH after craniotomy without intracranial SDH has also been reported. Rader suggested that a sudden increase in abdominal

Fig. 1. Case 1. Images obtained in an 83-year-old man with a history of prostate carcinoma and myelodysplastic syndrome. a: Brain CT image showing bilateral intracranial chronic SDH. b–e: Lumbar MR images revealing spinal SDH, which manifests as signal hyperintensity on the T1-weighted axial image acquired at the L5–S1 level (b) and T1-weighted sagittal image (d) and as an area of slight hypointensity to isointensity on the T2-weighted axial (c) and sagittal (e) images.

Fig. 2. Case 2. Images obtained in a 70-year-old man treated with drug therapy for hypertension and diabetes mellitus. a: Brain CT image revealing bilateral chronic SDH. b–e: Lumbar MR images revealing spinal SDH, which manifests as signal hyperintensity on the T1-weighted axial image acquired at the S-1 level (b) and the T1-weighted sagittal image (d) and as an area of slight hypointensity to isointensity on the T2-weighted axial (c) and sagittal (e) images.
and thoracic pressure may rapidly elevate the pressure in the spinal vessels crossing the subdural and subarachnoid spaces and if CSF pressure fails to immediately buffer this force, vessel rupture may ensue. Under that condition, spinal SDH may be provoked without migration of intracranial SDH, and low CSF pressure due to its excessive drainage may give rise to simultaneous intracranial and spinal SDH.

### TABLE 1: Summary of reported cases of concomitant intracranial and spinal subdural hematomas*

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Age (yrs), Sex</th>
<th>Factors in Hemorrhage</th>
<th>Cause of Injury</th>
<th>Lesion</th>
<th>Tx</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lee et al., 1996</td>
<td>15, M</td>
<td>—</td>
<td>fell &amp; hit H &amp; back</td>
<td>L1–5</td>
<td>punc</td>
<td>good</td>
</tr>
<tr>
<td>Shimada et al., 1996</td>
<td>68, NR</td>
<td>—</td>
<td>fell</td>
<td>T5–12</td>
<td>op</td>
<td>good</td>
</tr>
<tr>
<td>Leber et al., 1997</td>
<td>54, M</td>
<td>—</td>
<td>slipped on ice (H &amp; L)</td>
<td>L1–S2</td>
<td>op</td>
<td>good</td>
</tr>
<tr>
<td>Tiliich et al., 1999</td>
<td>54, M</td>
<td>—</td>
<td>fell on ice (H &amp; L)</td>
<td>L1–S2</td>
<td>op</td>
<td>good</td>
</tr>
<tr>
<td>Chen et al., 2001</td>
<td>31, M</td>
<td>—</td>
<td>fell</td>
<td>L3–5</td>
<td>op</td>
<td>good</td>
</tr>
<tr>
<td>Hung et al., 2002</td>
<td>12, M</td>
<td>—</td>
<td>fell down (H &amp; L)</td>
<td>L1–5</td>
<td>obs</td>
<td>good</td>
</tr>
<tr>
<td>Bortolotti et al., 2004</td>
<td>23, F</td>
<td>—</td>
<td>fell on snow</td>
<td>L4–S2</td>
<td>op</td>
<td>good</td>
</tr>
<tr>
<td>Ahn &amp; Smith, 2005</td>
<td>4, F</td>
<td>—</td>
<td>fell</td>
<td>C1–T4</td>
<td>obs</td>
<td>good</td>
</tr>
<tr>
<td>Sari et al., 2006</td>
<td>56, M</td>
<td>—</td>
<td>H trauma</td>
<td>L1–S2</td>
<td>op</td>
<td>good</td>
</tr>
<tr>
<td>Kim et al., 2010</td>
<td>24, F</td>
<td>—</td>
<td>slip on snow</td>
<td>L4–S2</td>
<td>obs</td>
<td>good</td>
</tr>
<tr>
<td>Kishen et al., 2009</td>
<td>76, M</td>
<td>APD</td>
<td>fell</td>
<td>L4–5</td>
<td>obs</td>
<td>good</td>
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<tr>
<td>Wong et al., 2009</td>
<td>73, F</td>
<td>APD</td>
<td>fell &amp; slipped on ground</td>
<td>T4–10</td>
<td>obs</td>
<td>good</td>
</tr>
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<td>Hagihara et al., 2010</td>
<td>47, M</td>
<td>APD</td>
<td>multiple trauma</td>
<td>L3–S1</td>
<td>obs</td>
<td>good</td>
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<tr>
<td>Moscovici et al., 2011</td>
<td>88, M</td>
<td>APD</td>
<td>fell</td>
<td>L5–S1</td>
<td>op</td>
<td>good</td>
</tr>
<tr>
<td>Nagashima et al., 2010</td>
<td>60, M</td>
<td>low platelets</td>
<td>fell down</td>
<td>L5–S2</td>
<td>obs</td>
<td>good</td>
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<tr>
<td>LaMela et al., 2012</td>
<td>58, M</td>
<td>ACD</td>
<td>hit H</td>
<td>T5–12</td>
<td>obs</td>
<td>good</td>
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<tr>
<td>Wajima et al., 2012</td>
<td>78, F</td>
<td>APD</td>
<td>fell</td>
<td>S1–2</td>
<td>obs</td>
<td>good</td>
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<td>present report</td>
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<tr>
<td>Case 1</td>
<td>83, M</td>
<td>MDS</td>
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<td>obs</td>
<td>good</td>
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<td>Case 2</td>
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<td>—</td>
<td>fell &amp; hit H &amp; L</td>
<td>L4–S2</td>
<td>obs</td>
<td>good</td>
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<tr>
<td>spontaneous hematoma (12 cases)</td>
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<tr>
<td>Kirsh et al., 2000</td>
<td>42, M</td>
<td>—</td>
<td>C1–L3</td>
<td>op</td>
<td></td>
<td>poor</td>
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<tr>
<td>Konistiotis et al., 2003</td>
<td>60, F</td>
<td>low platelets</td>
<td>T3–L5</td>
<td>obs</td>
<td></td>
<td>good</td>
</tr>
<tr>
<td>Lecouvet et al., 2003</td>
<td>31, M</td>
<td>—</td>
<td>L1–S2</td>
<td>obs</td>
<td></td>
<td>good</td>
</tr>
<tr>
<td>Yamaguchi et al., 2005</td>
<td>59, M</td>
<td>APS</td>
<td>T11–S1</td>
<td>obs</td>
<td></td>
<td>good</td>
</tr>
<tr>
<td>Jimbo et al., 2006</td>
<td>72, M</td>
<td>ACT</td>
<td>L4–S2</td>
<td>op</td>
<td></td>
<td>good</td>
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<tr>
<td>Lee et al., 2007</td>
<td>68, F</td>
<td>—</td>
<td>L5–S1</td>
<td>op</td>
<td></td>
<td>good</td>
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<tr>
<td>Morishige et al., 2007</td>
<td>54, F</td>
<td>—</td>
<td>C1–S2</td>
<td>punc</td>
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<td>good</td>
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<tr>
<td>Jain et al., 2008</td>
<td>12, M</td>
<td>AA</td>
<td>C1–S3</td>
<td>obs</td>
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<td>good</td>
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<td>Yang et al., 2009</td>
<td>35, F</td>
<td>—</td>
<td>L3–S1</td>
<td>op</td>
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<td>good</td>
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<td>Nagashima et al., 2010</td>
<td>66, M</td>
<td>—</td>
<td>L2–S1</td>
<td>obs</td>
<td></td>
<td>good</td>
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<tr>
<td>Sirin et al., 2010</td>
<td>77, M</td>
<td>MDS</td>
<td>L1–5</td>
<td>obs</td>
<td></td>
<td>good</td>
</tr>
<tr>
<td>Jibu et al., 2012</td>
<td>73, F</td>
<td>—</td>
<td>L3–S2</td>
<td>obs</td>
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<td>good</td>
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<tr>
<td>hematoma after craniotomy (3 cases)</td>
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<td></td>
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<tr>
<td>Shimizu et al., 1999</td>
<td>52, F</td>
<td>—</td>
<td>clipping of aneurysm</td>
<td>L4–S2</td>
<td>obs</td>
<td>good</td>
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<tr>
<td>Yamaguchi et al., 2003</td>
<td>52, F</td>
<td>—</td>
<td>clipping of aneurysm</td>
<td>L1–S2</td>
<td>obs</td>
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<tr>
<td>Liu et al., 2011</td>
<td>26, F</td>
<td>—</td>
<td>tumor resection</td>
<td>L3–S1</td>
<td>op</td>
<td>good</td>
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<tr>
<td>hematoma after VP shunt placement (3 cases)</td>
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<td>Silver &amp; Wilkins, 1991</td>
<td>14, F</td>
<td>—</td>
<td>VP shunt</td>
<td>T12–L2</td>
<td>obs</td>
<td>good</td>
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<tr>
<td>Wurm et al., 1996</td>
<td>16, M</td>
<td>—</td>
<td>VP shunt</td>
<td>L3–S2</td>
<td>obs</td>
<td>good</td>
</tr>
<tr>
<td>Ohta et al., 2001</td>
<td>10, F</td>
<td>—</td>
<td>VP shunt</td>
<td>L3–S2</td>
<td>op</td>
<td>good</td>
</tr>
</tbody>
</table>

* AA = anaplastic anemia; ACD = anticoagulant therapy; APD = antplatelet drug; H = head; L = lumbar; MDS = myelodysplastic syndrome; NR = not reported; obs = observation; punc = puncture and aspiration of hematoma; Tx = treatment.
Concomitant subdural hematomas

Others\(^1\,4\,5\,7\,8\,13\,17\,20\,21\,24\,29\,30\,34\,35\,39\,-41\) have speculated that in some of their trauma patients the spinal SDH migrated from the intracranial lesion because the intensity on MR images was the same for both types of hematoma. However, the mechanism of migration of the intracranial SDH through the membrane remained unclear.\(^4\,24\,29\) While it is difficult to confirm migration of the convex SDH from the intracranial to the lumbar region, the migration hypothesis is strengthened by the observation that the intensity of the concomitant lesions was the same. Also, if their concomitance is indeed due to migration, we can expect that many patients with intracranial SDH harbor concomitant asymptomatic spinal hematomas. As SDHs tend to coagulate quickly or become encapsulated within months, their gravity-induced migration to the lumbar region appears unlikely. In fact, in our prospective study of 168 patients with intracranial SDH, only 2 presented with concomitant lumbar SDH. This weakens the case for migration from the intracranial to the lumbar space. Others\(^8\,19\) have suggested that lumbar SDH is the result of direct trauma to the hip or lumbar area. To identify the incidence of concomitant intracranial and lumbar SDH, we separately evaluated patients with head trauma only and those with multiple traumas. We found that no patients with only head trauma manifested concomitant lumbar SDH, suggesting that in this group there was no SDH migration.

In the 17 previously reported trauma cases,\(^4\,5\,7\,8\,13\,17\,20\,21\,24\,29\,30\,34\,35\,39\,-41\) 11 patients had slipped or fallen, 3 had fallen from some height, and 1 patient was involved in a traffic accident. In those cases both the hips and head may have suffered trauma. Of our patients, only 2 presented with concomitant intracranial and lumbar SDH and both had a history of direct trauma to the head and the hips and/or lumbar area. As none of our patients with only head trauma presented with concomitant lumbar SDH, concomitant trauma to these areas was significantly related to the concomitance of cranial and spinal SDH. We posit that double trauma due to slipping or falling was a major mechanism underlying their concomitant SDH.

Of 12 previously reported cases of spontaneous SDH, 5 (42%) involved patients with a bleeding tendency; 3 of these patients had blood dyscrasia, and 2 had been treated with drugs that can affect bleeding.\(^9\,11\,18\,38\,41\) Interestingly, among the 17 earlier trauma patients, 7 (41%) also had a bleeding tendency, 1 presented with blood dyscrasia, and 6 were on drug therapy that can affect bleeding.\(^7\,17\,20\,29\,30\,40\,41\)

The patient in our Case 1 also had a history of myelodysplastic syndrome and hemorrhagic diathesis. Russell and Benoit\(^35\) reviewed 58 cases involving patients with spinal SDH and found that in 16%, SDH occurred after anticoagulation therapy. According to Khosla et al.,\(^13\) hematological disorders and trauma account for about 84% of spinal SDH, and spontaneous spinal SDH are often associated with the administration of anticoagulant therapy. These observations suggest that in some patients hemorrhagic diathesis may play a role in the concomitant development of spinal and lumbar SDH.

Spinal SDH can be managed with direct surgical removal, aspiration by percutaneous puncture, or observation. Of 29 earlier patients with posttraumatic or spontaneous hematomas,\(^1\,3\,5\,7\,-11\,13\,16\,-18\,20\,-22\,24\,25\,28\,-31\,34\,-45\) 11 had undergone surgery, 2 aspiration by lumbar puncture, and 16 had been placed under observation. In both of our patients with concomitant asymptomatic spinal SDH the lesions resolved spontaneously with conservative management. Lee et al.\(^24\) reported that their 4 patients with mild symptoms of spinal SDH who were placed under observation or underwent lumbar spinal puncture and/or drainage of the hematoma recovered completely within 3 weeks without permanent neurological deficits. Domenicucci et al.\(^5\) documented a good outcome in 29 (94%) of 31 patients with spinal SDH whose neurological status at the time of admission was satisfactory and in 3 (16%) of 19 patients who had severe neurological deficits. In the presence of acute deterioration and severe neurological deficits, emergency surgical decompression was recommended as the best treatment option.\(^5\,19\) Conservative management is appropriate in patients with mild neurological deficits and progressive improvement in the early period. Lastly, in some patients with stable deficits whose hematomas are located dorsally with moderate extension into the lumbosacral area and no bleeding diathesis, percutaneous drainage may be the best treatment choice.

We found a coincidence of several lumbar diseases in patients with chronic intracranial SDH. In earlier reports on the general population, lumbar spinal stenosis was identified in 17.3% of those examined; 21% of asymptomatic individuals between 60 and 80 years of age manifested lumbar spinal canal stenosis.\(^3\) These findings suggest that asymptomatic and coincidental lumbar diseases have no effect on the incidence of chronic SDH in the lumbar spine. According to others,\(^7\,27\) there is no definite correlation between the degree of narrowing of the lumbar spinal canal and the severity of clinical symptoms.

Our study has some limitations. In some cases, the patients’ memory or clinical status precluded the acquisition of reliable information on their trauma experience. Also, we included only patients with chronic SDH. Although none of them presented with acute SDH, other authors\(^1\,3\,9\,16\,18\,24\,29\,38\,40\,41\) have encountered patients with concomitant SDH who did. Because we did not acquire brain MRI scans, we could not compare the intensity of the intracranial and lumbar SDH in our 2 patients who had concomitant lesions. Furthermore, the findings with respect to the postoperative lumbar MR images may have reflected surgical effects. The issue of a possible relationship between the clinical symptoms of lumbar disease and radiological findings is currently under investigation. Moreover, in this study, cervical and thoracic spine studies are also evaluated.

Conclusions

The concomitance of lumbar and intracranial chronic SDH was rare in our series (1.2%) and was observed only in patients with a history of direct concomitant trauma to the head and the hip and/or lumbar area. None of our patients with concomitant intracranial and spinal SDH had sustained head trauma only. This suggests double trauma as a major mechanism underlying the development of...
concomitant intracranial and lumbar spine SDH. Other risk factors may be hemorrhagic diathesis and low-CSF syndrome.

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: Kim, Kokubo. Acquisition of data: Kim, Kokubo. Analysis and interpretation of data: Kim, Kokubo. Drafting the article: Kim, Kokubo. 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: Morita. Statistical analysis: Kim, Yoshida. Administrative/technical/material support: Kim. Study supervision: Kim, Kokubo. Reviewed submitted version of manuscript: Morita. Statistical analysis: Kim, Yoshida. Administrative/technical/material support: Kim. Study supervision: Kim, Mishina.

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