Diffuse Pachymeningeal Enhancement on Brain MRI: Spontaneous Intracranial Hypotension and Head Trauma

Chang Woo Ryu, M.D., Byung Hee Lee, M.D., Seung Ik Lee, M.D., Young A Kim, M.D., Hee Jin Kim, M.D., Young Sik Ko, M.D.

Purpose: We evaluated the MRI finding of pachymeningeal enhancement in patients with intracranial hypotension and head trauma with particular attention to differential findings and change in follow-up study, and in order to support the knowledge about the pathophysiology of dural enhancement.

Materials and Methods: The findings of enhanced brain MRI of fifteen patients who showed diffuse pachymeningeal enhancement were retrospectively examined. Seven of fifteen patients were finally diagnosed as spontaneous intracranial hypotension (SIH). Eight of fifteen patients had a recent history of head trauma. We analyzed the shape, thickness, continuity and extent of dural enhancement, and the others concerned with positive MR findings. We also analyzed findings suggested displacement of brain parenchyma- displacement of the iter and cerebellar tonsil, and flattening of the anterior aspect of the pons. Four of seven patients with SIH and four of eight patients with head trauma, underwent follow-up MRI. In the follow-up study, the presence of resolving pachymeningeal enhancement and symptom improvement was investigated.

Results: In all cases of SIH, the dura showed diffuse, even 3 (1 mm thick, global and contiguous enhancement along both cerebral convexities, both tentoria, and the falx. Displacement of the iter was noted in six cases and flattening of the anterior aspect of the pons in five. Displacement of the cerebellar tonsil was noted in one case. Five of seven cases showed small amount of subdural fluid collection. In all cases of head trauma, the dura was enhanced diffusely and asymmetrically, and showed no contiguity. Its distribution was consistent with the locations of traumatic lesions. Displacement of the iter was noted in one case. In four cases of SIH, clinical symptoms had improved, and three showed complete resolution of dural enhancement, in one patient continuously showed partial dural enhancement. Four cases of head trauma showed complete resolution of dural enhancement.

Conclusion: Reversible diffuse pachymeningeal enhancement and additional findings in brain MRI of SIH and head trauma are valuable for differentiation from other irreversible pathological conditions related to pachymeningeal enhancement and to support the knowledge about pathophysiology of dural enhancement.

Index words: Brain, MR

Meninges, MR

Diffuse pachymeningeal (dural) enhancement may be caused by inflammatory process such as infectious meningitis, neurosarcoidosis, or neoplastic process such as carcinomatous meningitis, and it can also be seen in patients with head trauma and intracranial hypotension and in who went craniotomy and ventricular shunting. Although meningeal enhancement is a sensitive finding, it has been considered nonspecific finding to differentiate various disease processes.
Spontaneous intracranial hypotension (SIH) is a syndrome of postural headache associated with a low CSF pressure. Schaltenbrand (1, 2) assumed three possible causes of this syndrome: CSF leakage, reduced CSF production, and increased CSF absorption. Reported MR findings of SIH were pachymeningeal enhancement, flattening of the pons, displacement of the optic chiasm, and downward displacement of the pons and iter (3, 4, 5).

Meningeal enhancement after head trauma has been regarded as a form of reactive meningitis which can be found postsurgically and in postventricular shunt placement and radiation therapy (6). There have, however, been a few reports of specific MR findings and the pathophysiology of reactive meningeal enhancement.

We evaluated the MRI findings of pachymeningeal enhancement in patients with intracranial hypotension and head trauma with particular attention to differential findings and change in follow-up study, and to support the knowledge about pathophysiology of dural enhancement.

Material and Methods

Fifteen patients who showed diffuse pachymeningeal enhancement on enhanced brain MRI were retrospectively evaluated during recent 26 months.

Seven of 15 patients were finally diagnosed as SIH by medical history, clinical presentation, CSF analysis, and radiologic findings. There was no clinical evidence of head trauma, infection or malignancy, but specific neurologic symptoms consistent with SIH such as postural headache, nausea and vomiting were present. They all showed low CSF pressure or dry tapping in lumbar puncture. Age distribution of seven patients were between 30 to 50 years (mean age; 39) and sexual distribution were five males and two females.

Eight of 15 patients with a history of head trauma had undergone enhanced brain MRI before two days to two weeks. Age distribution of eight patient were between 18 to 49 years (mean age; 29) and sexual distribution were six males and two females.

MRI was performed using a 1.5T (Magnetom Vision, Siemens, Germany). Axial, sagittal and coronal T1- and axial and coronal T2 weighted spin-echo sequences with TR/TE intervals of 500 – 600 ms/20 – 30 ms and 2500 ms/80 ms were obtained. Slices thickness was 5 mm with a 2 mm gap. Contrast-enhanced images in the axial and coronal plane were obtained using intravenously injected gadolinium-DPTA of a concentration of 0.1 mmol/kg.

We analyzed the shape, thickness, continuity and extent of dural enhancement, and the relationship with other positive MR findings. When enhanced dura show regular thickness (difference between maximal and minimal thickness is less than 3 mm), it is defined as 'even', if not, as 'uneven'. We analyzed the MRI findings suggesting displacement of brain parenchyma. MRI findings used in this study to identify displacement of brain parenchyma were following: the displacement of the iter and the cerebellar tonsil, and the flattening of anterior aspect of the pons (5, 7). The position of the iter was estimated using a straight line (incisural line) from the anterior tuberulum sellae posteriorly to the point marking the confluence of the straight sinus, great cerebral vein, and inferior sagittal sinus. When inferior deviation of the iter more than 1.8 mm is noted, it is interpreted as positive. The inferior deviation of the cerebellar tonsil was estimated using the foramen magnum and was interpreted as positive when the distance is more than 4 mm. In the eight patients with head trauma, we ascertained the presence of traumatic lesions such as subdural hematoma, cerebral contusion and hemorrhage, and scalp contusion or swelling on the cerebral convexity with the dural enhancement.

In four of seven patients with SIH and four of eight with head trauma, the follow-up study of axial, coronal and enhanced T1-weighted images was obtained. On follow-up MRI, the presence of resolving of pachymeningeal enhancement and symptom improvement was ascertained.

Results

In all seven cases of SIH, the enhanced dura showed even thickening of about 3 ± 1 mm. It showed global and contiguous with both cerebral convexities, both tentoria and the falx. Displacement of the iter was noted in six cases, flattening of the anterior aspect of the pons in five cases and displacement of the cerebellar tonsil in one case. All seven cases showed at least one evidence of cerebral displacement and five cases showed more than two evidences of brain displacement (Fig. 1A, B). four of seven cases showed small amount of subdural fluid collection and one case showed chronic subdural hematoma (Table 1).

In all cases of head trauma, the enhanced dura showed even thickening of about 3 ± 1 mm. It was distributed asymmetrically and was not contiguous with either cerebral convexities or the tentoria, or falx. Its distribution corresponded with the location of subdural hematoma, brain parenchymal contusion,
Table 1. Summary of Spontaneous Intracranial Hypotension

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age/sex</th>
<th>Thickness</th>
<th>Shape</th>
<th>Extent</th>
<th>Tonsil</th>
<th>Iter</th>
<th>Pontine flattening</th>
<th>SDF</th>
<th>Duration</th>
<th>Symptom</th>
<th>Resolution</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>30 / F</td>
<td>3</td>
<td>Even</td>
<td>Global</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Complete</td>
</tr>
<tr>
<td>2.</td>
<td>33 / M</td>
<td>3</td>
<td>Even</td>
<td>Global</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Improved</td>
</tr>
<tr>
<td>3.</td>
<td>50 / M</td>
<td>3</td>
<td>Even</td>
<td>Global</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Improvem.</td>
</tr>
<tr>
<td>4.</td>
<td>35 / M</td>
<td>3</td>
<td>Even</td>
<td>Global</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>41 / M</td>
<td>3</td>
<td>Even</td>
<td>Global</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>45 / M</td>
<td>2</td>
<td>Even</td>
<td>Global</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Complete</td>
</tr>
<tr>
<td>7.</td>
<td>41 / F</td>
<td>3</td>
<td>Even</td>
<td>Global</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Improved</td>
</tr>
</tbody>
</table>

*: mean thickness of enhanced dura (mm). SDF: subdural fluid collection.

Fig. 1. MR imaging of spontaneous intracranial hypotension.
Gadolinium enhanced coronal image (A) show diffuse enhancement of dura (open arrows) on both convexities, both tentoriums and falx. Sagittal T1 weighted image (B) show inferior displacement of the iter (arrow) from the incisural line (Line A), and flattening of anterior contour of pons (double arrows). After 3months later, Gadolinium enhanced coronal image (C) show resolution of dural enhancement.

Fig. 2. MR imaging of head trauma.
Gadolinium enhanced coronal image (A) show enhancement of dura (open arrows) on left cerebral convexity and tentorium, and coronal T2 weighted image (B) show cerebral contusion (arrow) on left temporal lobe. In follow-up MR image (C) show resolution of dural enhancement.
Table 2. Summary of Posttrauma

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age/sex</th>
<th>Thickness</th>
<th>Shape</th>
<th>Convexity</th>
<th>Tentorium</th>
<th>Falx</th>
<th>Iter</th>
<th>Displacement</th>
<th>Resolution</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>46/M</td>
<td>2</td>
<td>Even</td>
<td>Right</td>
<td>Right</td>
<td></td>
<td></td>
<td>Both</td>
<td>Complete</td>
<td>2months</td>
</tr>
<tr>
<td>2.</td>
<td>39/M</td>
<td>2</td>
<td>Even</td>
<td>Both</td>
<td>Left</td>
<td>+</td>
<td>Both</td>
<td>Both</td>
<td>Complete</td>
<td>5months</td>
</tr>
<tr>
<td>3.</td>
<td>18/M</td>
<td>3</td>
<td>Even</td>
<td>Right</td>
<td>—</td>
<td>—</td>
<td>+</td>
<td>Right</td>
<td>Complete</td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>25/M</td>
<td>2</td>
<td>Even</td>
<td>Both</td>
<td>Right</td>
<td>—</td>
<td>—</td>
<td>Both</td>
<td>Complete</td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>18/F</td>
<td>2</td>
<td>Even</td>
<td>Both</td>
<td>Left</td>
<td>—</td>
<td>—</td>
<td>Both</td>
<td>Complete</td>
<td>7months</td>
</tr>
<tr>
<td>6.</td>
<td>22/M</td>
<td>3</td>
<td>Even</td>
<td>Both</td>
<td>Right</td>
<td>—</td>
<td>—</td>
<td>Right</td>
<td>Complete</td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>34/M</td>
<td>2</td>
<td>Even</td>
<td>Left</td>
<td>Left</td>
<td>—</td>
<td>—</td>
<td>Left</td>
<td>Complete</td>
<td>7months</td>
</tr>
<tr>
<td>8.</td>
<td>49/F</td>
<td>2</td>
<td>Even</td>
<td>Both</td>
<td>Right</td>
<td>Left</td>
<td>—</td>
<td>Left</td>
<td>Complete</td>
<td></td>
</tr>
</tbody>
</table>

*: mean thickness of enhanced dura (mm), Cb: cerebral

and scalp contusion or swelling (Fig. 2A, B). Focal enhancement of the falx was seen in two cases in which there was traumatic lesion adjacent to the vertex. Evidence of brain displacement was seen in only one case involving displacement of the iter (Table 2).

Four of seven patients diagnosed as SIH underwent the follow-up MRI of intervals of 1 to 15 months. In all four, clinical symptoms improved and three showed complete resolution of dural enhancement (Fig. 1C). In the patient who has the shortest follow-up period (1 month), incomplete resolution of dural enhancement was noted. Four of eight patients with a history of head trauma underwent follow-up MRI of intervals of 2 to 17 months. All four showed complete resolution of dural enhancement and traumatic lesion (Fig. 2C).

Discussion

On high-field-strength MR images, normal meninges may be seen slightly enhanced after the administration of contrast material (8). In general, normal meningeal enhancement is visualized as a thin, markedly discontinuous rim covering the surface of the brain and is seen primarily in the dura and venous structure. Abnormal enhancement of the meninges is thicker and more extensive, and may be focal or diffuse, sheet-like, or nodular (6, 8).

Patterns of enhancement of the different layers of the meninges are divided into two types: leptomeningeal (pia and arachnoids), when enhancement of meninges follows the convolutions of the gyri and/or involves the meninges around the basal cisterns; and pachymeningeal (dura), when enhancement is along the inner surface of the calvarium, falx, or tentorium, without extension into the cortical gyri or basal cistern involvement (6). In our study, meningeal enhancement seen in intracranial hypotension and head trauma was pachymeningeal.

One of the causes of SIH diminished CSF production due to reversible disturbance of the choroid plexus. Such cessation of CSF secretion would lead to hyperemia of the brain and meninges (9). Before the reports of meningeal biopsy of SIH, some presumed that diffuse meningeal enhancement of SIH has been attributed to inflammation of the pachymeninges (10). Hochman et al (3) presented the followings as the cause of diffuse pachymeningeal enhancement in SIH 1) meningeal hyperemia secondary to reversible disturbance of choroidal plexus, which Schaltenbrand had suggested as a mechanism of SIH; 2) underlying inflammation of pachymeninges; 3) a nonspecific meningeal reaction to low CSF pressure. Fishman et al (11) suggested that dural enhancement was secondary to vascular dilatation, chiefly venous in nature, as a consequence of reduced CSF volume, and such venous engorgement resulted in a greater concentration of gadolinium-DTPA in dural vasculature and its accumulation in interstitial fluid of the dura. In our study, the fact that evidence of cerebral displacement on MRI and complete (n = 3) or incomplete (n = 1) resolution of dural enhancement in follow-up MRI was shown in all cases would be helpful to explain the possibility that the pachymeningeal enhancement in SIH results from the secondary change by low CSF pressure rather than the inflammatory change of dura.

Pannullo et al (7) reported seven cases of MRI findings of the intracranial hypotension (four cases: spontaneous, three cases: after lumbar puncture) in which the dural enhancement was global including along cerebral convexities and falx and tentorium without skip areas and the configuration of enhancement was linear. Although similar findings with previous studies were also noted, the membrane thickness was about 3 mm in all cases in our study. In that, our
study is totally different from other existing studies that showed different membrane thickness, ranged from 2 to 8 mm.

Subdural effusions are probably secondary to rupture of bridging veins due to decreased CSF volume and downward displacement of brain (12). In our study, subdural effusion was noted in six of eight cases; when it is associated with intracranial hypotension, subdural effusion requires further study; various reports have noted different incidences of subdural fluid collection in intracranial hypotension (5, 12, 13).

Dural enhancement in head trauma was different from dural enhancement in SIH since the former accompanied traumatic lesion and showed discontinuous asymmetric distribution. As the mechanism of posttraumatic dural enhancement we considered meningeal enhancement representing meningeal inflammation and subsequent fibrosis in ventricular shunt placement and postcraniotomy, but we concluded that it would be different mechanism since it was different from our study that did not show leptomeningeal enhancement but noted complete resolution in follow-up. We finally decided that the cause of posttraumatic dural enhancement would be reversible chemical inflammation due to blood or focal contusion of dura. But we could not find the report describing incidence and pathology of dural enhancement of post-trauma and in light of this, believe that it is necessary to research about this subject using an adequate number of cases with statistical validity as base data.

In conclusion, the diffuse and contiguous pattern of pachymeningeal enhancement in SIH and displacement of brain parenchyma [such as downward displacement of the iter or cerebellar tonsil or flattening of the anterior aspect of pons] seen on MRI are useful additional findings to differentiate these from other dural pathologies. Pachymeningeal enhancement in head trauma show diffuse and contiguous pattern, and its distribution is consistent with adjacent traumatic lesions. Although follow-up MRI was performed in a small number of cases, dural enhancement was resolved in all eight patients who underwent follow-up study. In the situations described above, unnecessary pathologic confirmation and aggressive management of dural change must be avoided because pachymeningeal enhancement is a reversible change. In SIH and post-trauma, acknowledgement of reversible pachymeningeal enhancement with additional MRI finding may be useful to help determine a theory the pathophysiology of the dura.

References

목적: 자발성 두개내 저혈압과 급성 두부 외상에 관찰되는 경막의 조영증강을 중심으로 자기공명영상의 소견을 비교 분석하고 추적 검사에서의 변화를 관찰하여 각 질환의 병리생리학을 이해하는데 도움이 되고자 한다.

대상 및 방법: 뇌 자기공명영상에서 경막의 미만성 조영증강을 보인 15명의 환자 중 두개내 저혈압으로 진단된 7명과 두부외상의 과거력이 있는 8명을 대상으로 하였다. 뇌 자기공명영상에서 조영증강된 경막의 형태, 두께, 연속성 및 범위, 뇌실질의 전위유무 - 실비우스통로(iter), 소뇌편도(cerebellar tonsil), 연수의 형태 -, 다른 병변과의 상관관계를 후향적으로 분석하였다. 추적이 가능한 8명의 환자(두개내 저혈압: 4명, 두부외상: 4명)에서는 추적 뇌 자기공명영상 검사를 시행하였고 임상 증상의 변화를 함께 조사하였다.

결과: 자발성 두개내 저혈압의 환자에서 경막은 전 예에서 미만성의, 두께가 3±1 mm의 비교적 고른 양상을 보였고 대뇌경(cerebral falx), 좌우 대뇌절면(cerebral convexity), 좌우 천막(tentorium)이 모두 연속적으로 조영 증강되었다. 7예 중 6예에서 실비우스통로(iter)의 하방전위, 5예에서 연수 전면부의 편평해짐, 1예에서 소뇌편도의 하방전위가 있었다. 5예에서 경막하 수종이 관찰되었다. 두부 외상의 환자에서 경막은 전 예에서 미만성의 약 3±1 mm의 고른 두께를 보이고 불연속, 비대칭으로 조영 증강되었다. 분포는 경막하 출혈, 뇌실질 좌상, 두피의 좌상 또는 팽윤등의 외상 소견의 위치와 일치하였다. 1예에서 실비우스통로의 하방전위가 관찰되었다. 추적 검사에서 자발성 두개내 저혈압은 4예가 모두 임상 증상의 호전이 있었고 3예에서는 경막조영의 완전한 소실이 있었으나 1예에서는 부분적인 경막조영이 계속 관찰되었다. 두부 외상의 4예에서는 추적검사상 모두 경막조영의 완전한 소실이 있었다.

결론: 뇌 자기공명영상에서 자발성 두개내 저혈압과 두부 외상의 환자에서 관찰되는 특정적인 가역적 미만성 경막조영 소견은 미만성 경막조영증강을 보이는 다른 비가역적 병리와 감별이 될 것으로 사료되며 상기 질환에서의 병리생리학을 이해하는데 도움이 될 것으로 생각한다.