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Asymptomatic spinal subdural epiarachnoid hygroma after lumbar laminectomy for lumbar spinal canal stenosis: illustrative case

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BACKGROUND Spinal subdural epiarachnoid hygroma (SSEH) after lumbar laminectomy is an extremely rare complication.

OBSERVATIONS An 84-year-old man presented to the hospital with lower back pain, radicular pain, and numbness in the lateral aspect of the left leg. Magnetic resonance imaging (MRI) revealed anterior lumbar spondylolisthesis at L3, severe disc herniation at L3–4, and severe lumbar spinal canal stenosis at L3–4 and L4–5. Lumbar laminectomy at L3–4 and L4–5 and discectomy at L3–4 were performed without complications such as cerebrospinal fluid (CSF) leakage and durotomy intraoperatively. Although lower back pain and numbness at the lateral aspect of the left leg were resolved postoperatively, postoperative MRI showed spinal nerve deviation to the ventral side due to SSEH from T12 to S1. Conservative therapy was performed for asymptomatic SSEH, and MRI 1 week postoperatively indicated improved ventral spinal nerve deviation and reduced SSEH.

LESSONS SSEHs after posterior decompression without durotomy are extremely rare. Asymptomatic SSEHs may resolve with conservative treatment. However, surgery should be performed to decompress hygroma in patients with symptomatic SSEH.

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KEYWORDS subdural hygroma; spine; lumbar laminectomy; lumbar canal stenosis

Lumbar posterior decompression (LPD) is an effective surgical method for treating severe lumbar stenosis. Various complications following LPD have been reported; however, spinal subdural epiarachnoid hygroma (SSEH) after lumbar laminectomy is an extremely rare complication. Some cases of SSEHs associated with intraoperative durotomy in cranial and spinal surgery have been reported in the literature.[1–3] Although there have been few reports of symptomatic SSEH after spinal fusion without intraoperative durotomy,[4–6] to the best of our knowledge, a case of asymptomatic SSEH with spontaneous resolution captured through imaging has not been recognized. We herein report a case of postoperative asymptomatic SSEH caused by an uneventful LPD without intraoperative durotomy that spontaneously resolved as observed on magnetic resonance imaging (MRI).

Illustrative Case

An 84-year-old man presented to our hospital with lower back pain, radicular pain, and numbness in the lateral aspect of his left leg. His medical history included percutaneous coronary intervention for angina and hormonal therapy for prostate cancer. Muscle strength was mildly decreased in the quadriceps, hip adductor, and tibialis anterior. Tendon reflex findings were unremarkable. MRI revealed anterior lumbar spondylolisthesis at L3, severe disc herniation at L3–4, and severe lumbar spinal canal stenosis at L3–4 and L4–5 (Fig. 1). Radiography showed no instability in the anterior lumbar spondylolisthesis. The patient was diagnosed with severe disc herniation at L3–4 and severe lumbar spinal canal stenosis at L3–4 and L4–5, for which lumbar laminectomy at L3–4 and L4–5 and discectomy at L3–4 were performed. No obvious durotomy or cerebrospinal fluid (CSF) leakage was observed intraoperatively.

Lower back pain, radicular pain, and numbness at the lateral aspect of the left leg, which presented preoperatively, were resolved postoperatively. A drain was removed on the second postoperative day. One week postoperatively, a routine MRI performed for postoperative assessment showed spinal nerve deviation to the ventral side due to SSEH from T12 to S1 (Fig. 2). Because the patient presented without lower back pain, radicular pain, numbness in both

ABBREVIATIONS CSF = cerebrospinal fluid; LPD = lumbar posterior decompression; MRI = magnetic resonance imaging; SSEH = subdural epiarachnoid hygroma.
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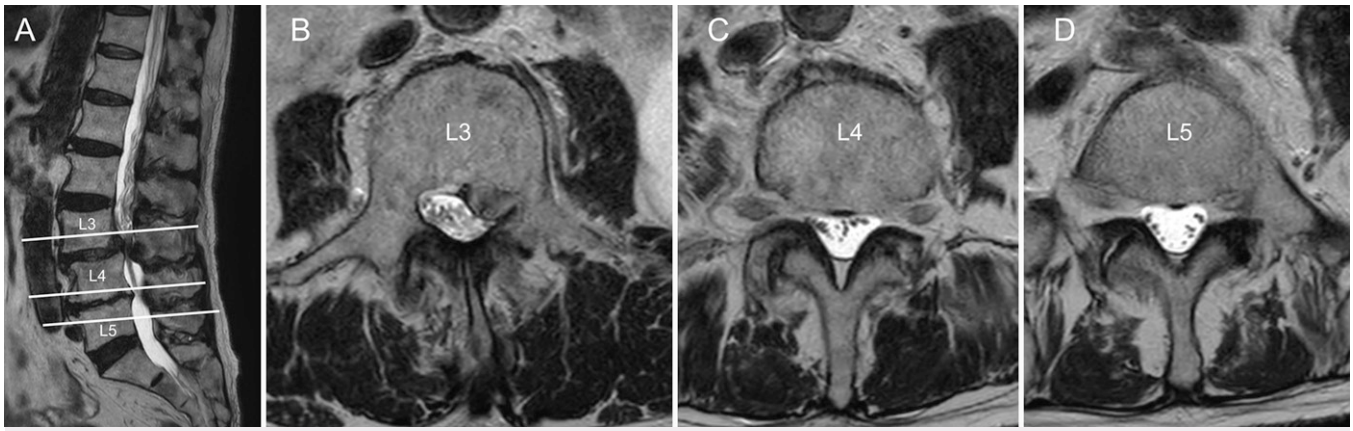

FIG. 1. Initial spinal cord MRI. Sagittal T2-weighted image (**A**) shows severe disc herniation at L3–4 and severe lumbar spinal canal stenosis at L3–4 and L4–5. Axial T2-weighted images (**B–D**) at the levels of the *white lines* in panel A.

legs, or bladder/bowel dysfunction, conservative therapy was performed. One week later, MRI indicated an improvement in the ventral deviation of the spinal nerves and a reduction in SSEH (Fig. 3). Thirty-three days after admission, the patient was transferred to another hospital for rehabilitation.

Discussion

Observations

An SSEH is defined as the accumulation of CSF between the dura and arachnoid space. In previous reports of SSEH with and without intraoperative durotomy, researchers proposed that the mechanism of CSF collection within the subdural space may be caused by the destruction of the arachnoid layer, resulting in a one-way valve that allows CSF to enter the subdural space.[1–6] In this case, durotomy or CSF leakage was not noted intraoperatively; however, arachnoid injury may have occurred due to physical stimulation by the procedure. A possible reason for the unilateral passage of CSF from the subarachnoid space to the subdural space is the effect of pressure changes in the meningeal compartments.[7] Monro-Kellie proposed that the sum volumes of the brain, CSF, and intracranial blood are constant.[8] Severe lumbar canal stenosis causes intradural pressure disparity between the upper and lower levels of canal stenosis.[9,10] This was eliminated by decompressing the lumbar canal stenosis. Consequently, unidirectional changes in the volume of CSF and/or intracranial blood due to the resolution of this disparity may lead to the development of SSEH. Moreover, the denticulate ligaments become stronger in the cervical region and weaker as the spinal cord descends.[11] Tension changes depending on the vertebral level in the denticulate ligaments may also be related to the development of SSEH. In the current case, initially, the outflow of CSF into the subdural space due to the arachnoid injury was unidirectional, yet this flow may have become bidirectional due to physical stimuli, such as walking or pulsation associated with respiration and blood pressure, resulting in spontaneous resolution on MRI. An alternative mechanism is that any change in the CSF absorption pathway from the dura can lead to CSF collection within the dura, resulting in SSEH.[12] CSF may be transported from the subarachnoid space into the interstitial space of the dura through various mechanisms (i.e., along exiting nerves, exiting vessels, transcellular transport, and arachnoid granulations).[7]

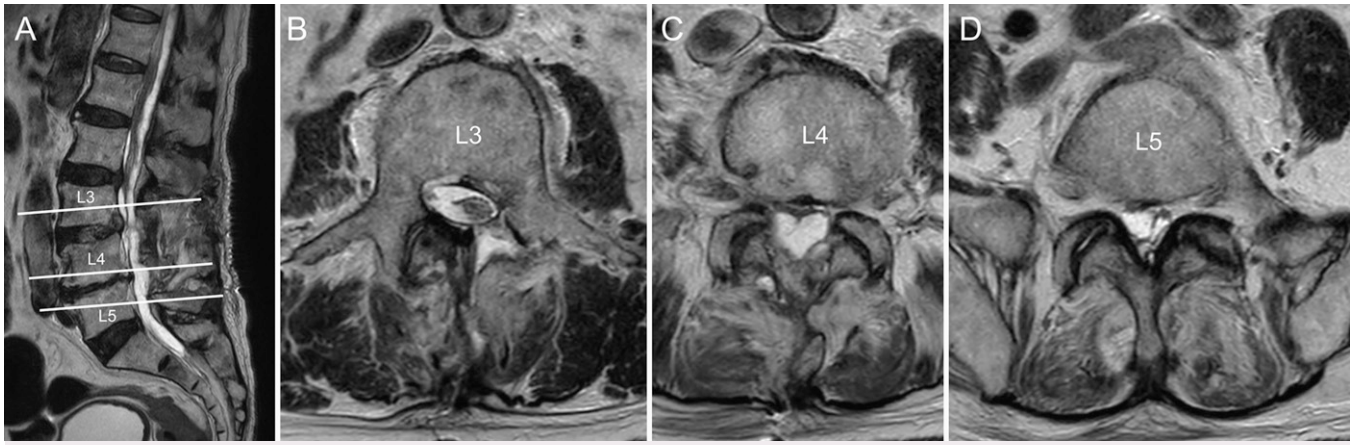

FIG. 2. One-week postoperative sagittal T2-weighted (**A**) and axial (levels of the *white lines* in panel A) T2-weighted (**B–D**) MRI shows spinal nerve deviation to the ventral side due to SSEH from T12 to S1.

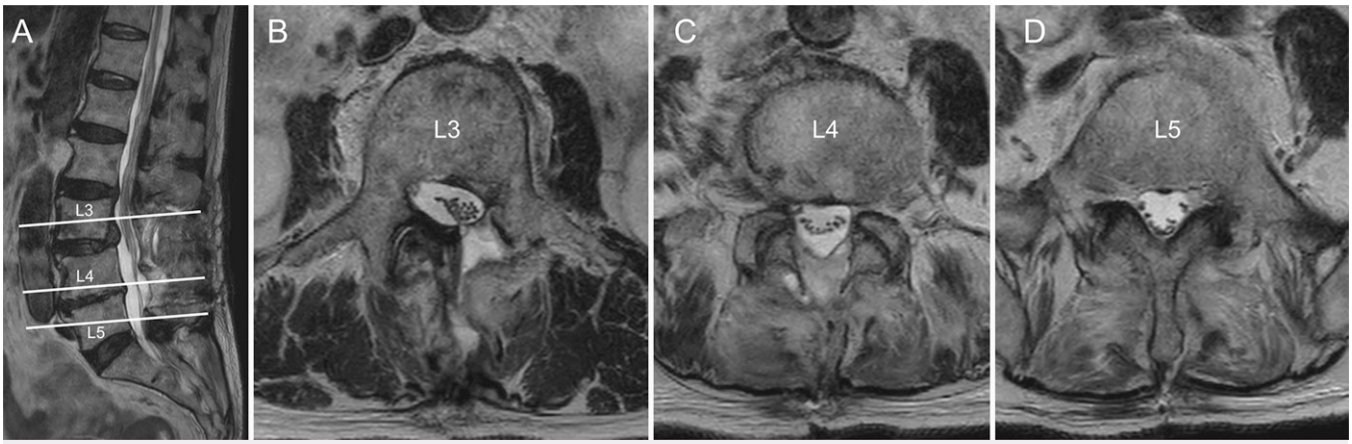

FIG. 3. Two-week postoperative sagittal T2-weighted (**A**) and axial (levels of the *white lines* in panel A) T2-weighted (**B–D**) MRI shows improved spinal nerves that are deviated to the ventral side by SSEH.

Several cases of SSEHs with cauda equina syndrome after lumbar surgery have been reported.[3–6] However, to the best of our knowledge, this study is the first to report an asymptomatic SSEH with improved imaging (Table 1). MRI is suitable for the diagnosis of SSEH. MRI shows that the spinal nerves are flattened and compressed on axial images because of lateral anchoring by the dentate ligament. If progressive cauda equina syndrome is observed due to SSEH postoperatively, treatments to decompress SSEH tension by laminectomy and standard durotomy and repair the arachnoid should be performed immediately.[3–6] However, for asymptomatic SSEHs, as in the current case, conservative treatment can be selected.

Lessons

SSEHs after posterior decompression without durotomy are extremely rare. Asymptomatic SSEHs may resolve with conservative treatment. However, surgery should be performed to decompress hygroma in patients with symptomatic SSEH.

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TABLE 1. Summary of spinal subdural epi-arachnoid hygroma following lumbar posterior decompression

Authors & Year	Age (yrs)	Sex	Procedure	Durotomy	Location of Hygroma	Symptomatic (S) or Asymptomatic (A)	Treatment
Singleton et al., 2012[3]	71	M	Lumbar microsurgical decompression	+	L4 to S2	S	Decompression & arachnoid & dural layers closed together
Singleton et al., 2012[3]	31	F	L5–S1 microdiscectomy	+	L5 to S1	S	Decompression
Elder et al., 2017[4]	53	F	lt L5–S1 discectomy & lt L4–5 laminoforaminotomy	–	Lower thoracic spine to S1	S	Decompression, dural suture, & fibrin glue
Mak et al., 2021[5]	63	F	L3–4 & L4–5 laminectomy	–	L1–5	S	Decompression & dural suture
Nentwig et al., 2019[6]	34	M	L4–5 decompression & posterolateral instrumented fusion of L4–S1	–	T12 to L2	S	Decompression
Present case	84	M	L3–4 & L4–5 laminectomy & L3–4 discectomy	–	T12 to S1	A	Conservative therapy

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Disclosures

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

Conception and design: Kawasaki, Ioroi. Acquisition of data: Kawasaki. Analysis and interpretation of data: Kawasaki. Drafting the article: Kawasaki, Kobayashi. Critically revising the article: all authors. Reviewed submitted version of manuscript: Takayama, Maki. Approved the final version of the manuscript on behalf of all authors: Kawasaki. Study supervision: Takayama.

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