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LETTER TO THE EDITOR

Active cerebrospinal fluid leakage after resolution of postdural puncture headache

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To the Editor:

The clinical course of a postdural puncture headache (PDPH) is generally benign and most cases resolve spontaneously within 1 week, or within 48 h after appropriate treatment. However, imaging findings from cases of PDPH have not been adequately reported. Herein, we report imaging findings and clinical symptoms during the recovery phase of PDPH after accidental dural puncture in a patient in whom active cerebrospinal fluid (CSF) leakage was revealed, despite resolution of PDPH symptoms.

A 54-year-old woman had been treated with an epidural block for sciatic pain. However, she began to complain of headache, nausea, and vomiting from the day after that treatment (day 0). At the time of referral to our hospital on day 5, she was nearly bed-ridden because of an intolerable orthostatic headache and nausea. Results of a neurological examination were normal, except for the orthostatic headache, and previously existing painful numbness in the right L5 and S1 dermatome distributions. The patient did not report tinnitus or hearing disturbance. Cranial magnetic resonance imaging (MRI) on day 6 revealed pachymeningeal gadolinium enhancement (PMGE) and enlargement of the venous sinuses (Fig. 1a). We made a diagnosis of PDPH after accidental dural puncture. By day 8, the symptoms had improved with bed rest and hydration at 1500 ml/day. Lumbar MRI on day 8 revealed spondylolisthesis with a narrow canal at the L4–5 level. In addition, an abnormal hyperintensity back to the same level was noted by T2-weighted imaging (Fig. 1b, arrow). CSF accumulation associated with accidental dural puncture, meningeal diverticulum and juxta-facet cysts were considered for differential diagnosis. Computerized tomography (CT) myelography was performed for further evaluation on day 15 when the symptoms were nearly resolved. The opening pressure upon lumbar puncture in a lateral decubitus position at L2–3 was 160 mmH_2O. Neither early nor delayed CT images of the entire spine after 20 ml intrathecal injection of contrast medium (Isovist® Inj. 240; Bayer Yakuhin, Limited, Osaka, Japan) revealed any cystic lesions. However, CSF leakage on the left side extending to the paraspinal muscle was found (Fig. 1c, d). We concluded that the abnormal findings obtained by lumbar MRI indicated CSF accumulation after accidental dural puncture. Cranial MRI performed 1 month later showed disappearance of PMGE and enlargement of the venous sinuses. Lumbar MRI 5 months after discharge confirmed the disappearance of abnormal hyperintensity (Fig. 1e, f). At a 1-year follow-up examination, she was free from PDPH symptoms.

Imaging findings of severe PDPH may be similar to those of spontaneous intracranial hypotension, including PMGE, engorgement of the venous system, CSF accumulation, and subdural hematoma, as revealed by both cranial and spinal MRI [1–3]. In this case, despite resolution of the clinical symptoms, active CSF leakage was revealed by CT myelography and the opening pressure of CSF upon lumbar puncture was normal on day 15. Furthermore, lumbar MRI on day 8 revealed abnormal signal intensity associated with CSF accumulation due to the accidental dural puncture. Discrepancies between imaging findings and clinical symptoms during the clinical course of intracranial

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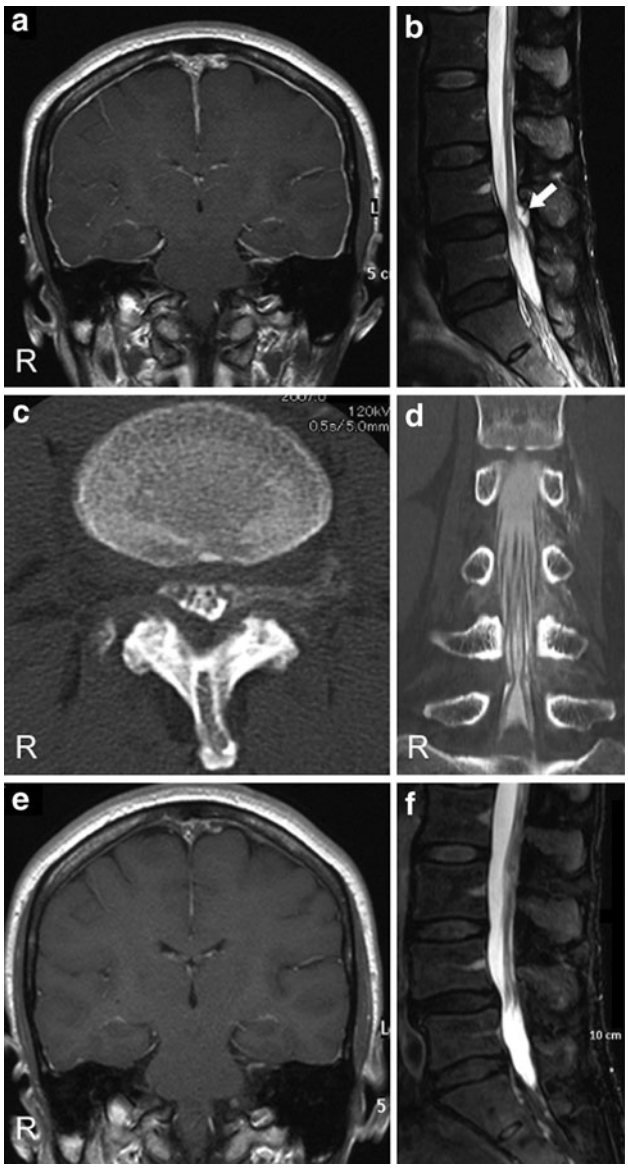

Fig. 1 **a** Coronal gadolinium-enhanced T1-weighted cranial MRI revealing PMGE and enlargement of the venous sinuses. **b** Sagittal T2-weighted lumbar MRI showing L4–5 spondyloisthesis. An abnormal hyperintensity is noted back to the same level (*arrow*). **c** Axial CT myelogram at the L4–5 level showing CSF leakage on the left side. **d** Coronal reconstruction image of CT myelogram findings of the lumbar region showing extensive CSF leakage. **e** Coronal gadolinium-enhanced T1-weighted cranial MRI 1-month later revealing disappearance of PMGE and enlargement of venous sinuses. **f** Sagittal T2-weighted lumbar MRI 5 months after discharge showing the disappearance of abnormal hyperintensity. *R* right side

hypotension have been reported [4, 5]. We considered that the CT myelography findings in this case that did not reflect the clinical symptoms could be similarly explained. In addition, it is also possible for occult CSF leakage to be present even after resolution of PDPH symptoms, with inappropriate treatment possibly causing recurrence. It is important for clinicians to recognize that patients with PDPH may have imaging findings that suggest persistent CSF leakage associated with accidental dural puncture. Therefore, careful observation and appropriate treatment should be conducted for severe cases of PDPH even after resolution of clinical symptoms.

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