Spinal subdural hematoma (SDH) is a rare hemorrhagic condition, which accounts for only 4.1% of all spinal hematomas. Causative factors include lumbar puncture, surgery, trauma, bleeding disorder, and vascular malformation. In 5% of cases, there is no evidence of any potential cause and the spinal hematoma is regarded as spontaneous. Spontaneous spinal SDH following an intracranial lesion is extremely rare, and only 11 cases have been reported, most of them after intracranial surgery or head injury. We report a case of spontaneous spinal SDH following spontaneous intracranial SDH without any causative factor. The clinical presentation was initial headache and dizziness, followed by subsequent lower limb weakness and paraparesis.

**Case Report**

A 35-year-old woman suffered from spontaneous headache and dizziness about 2 weeks prior to presentation. These symptoms progressed and were joined by new symptoms of lower back pain, lower limb weakness, paraparesis and mild fever (about 38°C), which caused her to seek help at the emergency department. Laboratory data revealed normal values for platelet, leukocyte and differential counts. Coagulation parameters were normal, including prothrombin of about 12 seconds, international normalized ratio of 0.95, and activated partial thromboplastin time of about 27 seconds. C-reactive protein (CRP) level was normal at <0.3 mg/dL. Mild fever was categorized as...
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as dehydration fever, and it subsided on the next day after infusion of 1500 mL normal saline. There was no evidence of sepsis. No mention of special travel or traumatic history was obtained. Physical examination revealed lower limb weakness and paraparesis, and walking gait was unstable. No evidence of cranial nerve deficit was demonstrated. Deep tendon reflexes were normal. Brain and spinal magnetic resonance imaging (MRI) was performed with a 1.5T scanner (GE Advantage, Milwaukee, WI, USA). The spinal imaging sequences consisted of spin-echo T1-weighted axial/sagittal images obtained before and after intravenous injection of gadolinium (0.1 mmol/kg), and spin-echo T2-weighted axial/sagittal images. Brain MRI consisted of pre-contrast axial T1/T2-weighted and fluid-attenuated inversion recovery (FLAIR) images, and post-contrast axial/coronal sections. Spinal MRI revealed SDH (Figure 1) distributed from the L3 to S1 level, and surrounded and compressed the cauda equina nerve within the dura sac, with enhanced membrane. In addition, a small continuous SDH that reached the thoracolumbar spine level (thin arrow) was demonstrated. It had a relatively high signal intensity on T1-weighted images, and isointense to slightly hyperintense signals around the paraspinal muscles on T2-weighted images. Subsequent laminectomy and removal of the SSSDH were performed.

Discussion

For spinal subdural hemorrhage (SSDH), the most important differential pathology is spinal epidural hemorrhage (SEDH). The imaging of SSDH is usually less conspicuous than in SEDH, because the blood distribution is semicircular, with subsequent compression instead of displacement of the spinal cord or cauda equina nerve. The majority of SSDHs are located ventral to the spinal cord or circumferentially with a major ventral part, whereas SEDH is usually found dorsal to the dura sac, because of the tight fixation of the dura to the posterior longitudinal ligament. In addition, SSDH does not always have a mass effect on epidural fat, such as displacement or deformation. However, SEDH may exert such an effect. From the imaging point of view, preoperative diagnosis of SSDH in our patient could be made,
because of the circumferential distribution, subsequent compression of the cauda equina nerve, and lack of effect on epidural fat.

SSDH is a rare condition compared with intracranial SDH. There are no bridging veins running in the spinal subdural space. SSDH may be caused by apparent trauma, iatrogenic maneuvers such as lumbar spinal puncture, or hemostatic disorders. The development of spontaneous SSDH (SSSDH) may follow intracranial surgery, but only a few cases have been reported in the literature. To the best of our knowledge, spontaneous spinal and intracranial SDH have not been reported previously. As a result of the clinical presentation of initial headache and dizziness, followed by lower limb weakness, the origin of SSSDH from intracranial SDH is most likely.

From postmortem study, it has long been recognized that air injected within the spinal subdural compartment readily appears in the intracranial subdural compartment, and it seems likely that air in the subdural compartment can flow intracranially when the patient is upright. This suggests that blood may distribute in a similar fashion in the craniocaudal direction after intracranial surgery. The development of SSSDH after intracranial surgery under the influence of gravity can be demonstrated by imaging. The propagation of blood from the cranial to the lumbar subdural spaces and be explained by the anatomic continuity between the subdural spaces.

In the case reported here, no evidence of vascular abnormality was found by MRI or selective angiography. No previous history of lumbar puncture, surgery or trauma could be obtained. There was also no evidence of bleeding or coagulation disorders. Furthermore, the simultaneous appearance of intracranial and spinal SDH, the onset of headache and dizziness, and then lower limb weakness and paraparesis suggested a close relationship between the SDHs. The cause of spontaneous intracranial SDH is unknown, but spontaneous bridging vein rupture is the most likely explanation.

Intracranial SDH can grow or redistribute as time progresses. Spontaneous resolution or redistribution of intracranial SDH seems to be more common in children and young adults. It is hypothesized that a young “elastic” brain forces redistribution of subdural hemorrhage. Thus, the blood can distribute to the spinal subdural space, although this is rare, under the influence of gravity and through anatomic continuity.

Figure 2. (A) Pre-gadolinium (TR/TE/Nex: 550/15/1) and (B) post-gadolinium (500/10/1) axial sections. The intracranial SDH in the left frontoparietal region, with enhanced membrane and adjacent venous engorgement, exerted a mass effect on the adjacent brain parenchyma. Spontaneous intracranial SDH was confirmed surgically.
For an intracranial SDH patient with new complaints of lower back pain or paraparesis, early MRI is necessary to detect SSSDH. The treatment options for SSSDH depend on the clinical presentation. If the clinical symptoms are not severe, close follow-up imaging may be the first strategy. Laminectomy and removal of SDH should be reserved for when follow-up imaging or clinical examination shows progressive deterioration.

References


