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Case Report

A calvarial acute subdural hematoma migrating into the spinal canal in a young male

Abstract

It is not common for an acute subdural hematoma (SDH) in the supratentorial region to show rapid resolution or migration during the clinical course. In this report, we present a rare case where the SDH in the supratentorial region was observed to rapidly migrate into the lumbar spinal canal, leading to severe radiculopathy. A 20-year-old male patient was admitted to the emergency department with severe headache after head trauma. The patient's overall condition was good, whereas his Glasgow Coma Scale score was 15 and blood pressure was normal. He had vomited 3 times after the onset of pain. No stiff neck was found, and the computed tomography showed an ASDH over the outer layer of the right hemisphere, causing a 7- to 8-mm shift. During the follow-up, the headache regressed and eventually resolved after 12 hours; however, another severe pain occurred in the lumbar region and in both legs. The pain worsened over time, progressing to sciatica in both legs. Acute SDH associated with a minor head trauma may migrate from the supratentorial compartment into the spinal canal by the help of elastic cerebral tissues in young adults and children.

Traumatic acute subdural hematoma (ASDH) is a neurosurgical emergency with a high mortality rate (60%-80%). Generally, it requires rapid surgical intervention [1]. The rapid resolution of traumatic ASDH was first reported in 1986 [2-4]. The spontaneous resolution of cerebral subdural hematoma (SDH) occurring particularly after cranial fractures in children and young adults, both patient groups having relatively high cerebral expansion capacities, is encountered during monitoring in the clinical practice of neurosurgery [5,6]. However, it is noted that the intracranial SDH cannot migrate into the spinal canal shortly after the trauma [1-5]. Nonetheless, this event, reported by few studies, is still recognized as a phenomenon [2,7-9].

In this study, we discuss a rare case of supratentorial ASDH, migrating rapidly into the lumbar spinal canal and leading to severe radiculopathy, in combination with the radiographic images.

A 20-year-old male patient was admitted to the emergency department because of severe headache associated with a minor head trauma. The neurologic examination revealed no pathology, and the blood pressure was normal. The patient had vomited 3 times after the onset of headache. However, his overall health status was very good. No stiff neck was found and the head computed tomography (CT) displayed an ASDH of 0.92 cm thickness over the outer layer of the right hemisphere, causing a shift of 0.91 cm (Fig. 1a-c). Because his overall health status was good, we decided to monitor the patient for a short time and repeat the head CT after 8 hours. The first head CT had shown a hypodense thin band between the hematoma and the calvarium, at the parietal border of the hematoma. The second head CT assessment showed no SDH in the parietal area, whereas the cortical surface at the level of right temporal lobe inferior gyrus, tentorium, and the adjacent tentorial free margin showed areas of hyperdensity. Moreover, the cerebral midline shift was observed to be resolved (Fig. 1d and e). The patient was continued to be monitored.

During the monitoring period, the headache resolved after 24 hours. The patient developed severe pain in the lumbar area and in both legs. The pain was of radicular character, and the Lasegué test was 30° positive in both legs. There was no pathologic reflex. The pain in the legs was a typical sciatica, and the complaints of the patient increased over time. The spinal magnetic resonance imaging (MRI) showed a hypointense lesion starting from the Th 10-11 level and extending to the lower areas intradurally, while filling the canal like a column which was consistent with ASDH (Fig. 2a-d). T2 sagittal magnetic resonance (MR) image showed that the hematoma was in the arachnoid layer by displaying a band of cerebrospinal fluid (CSF) between the hematoma and the neural tissue. Moreover, cranial MR angiography was applied. Because no vascular pathology was determined, digital substrate angiography was not performed. The entire spine was scanned with MRI; however, no additional lesion was found. All the complaints of the patient resolved after 1 month, and he began ambulation without any pain. The follow-up MRI showed the resolution of the hematoma in the lumbar region (Fig. 3a and b). Approximately 1 year after the treatment, he still has normal neurologic findings.

Radiographically, ASDH cases associated with a major trauma and presenting with a thickness greater than 10 mm or those of 5 to 10 mm thickness accompanied by impaired consciousness, hemiparesis, and pupillary changes mandate emergency surgery [1-4,10]. Nonetheless, there are cases in the literature, albeit very few, reporting spontaneous resolution within 2 to 72 hours after the diagnosis of SDH [8,11-17]. There are various theories aiming to explain the spontaneous resolution of acute cranial SDH.

In the first theory developed by Makiyama et al [15], there is a meningeal tear resulting from a trauma. Because of the cerebral edema associated with the meningeal damage, the subdural coagulated blood is forced to flow out by the increased intracranial pressure (ICP) [15]. First, a massive cerebral edema forms 20 to 60 minutes after the severe head trauma [18]. Coupled by the pulsations of the cerebral parenchyma, acute cerebral edema can force the SDH to migrate to an extradural or extracranial area [15]. Niikawa et al [17] reported the spontaneous resolution of an SDH by acute cerebral edema in 1 case. In our patient, there were temporary mild clinical symptoms suggesting elevated ICP. The presence of symptoms associated with acute ICP increase and rapid resolution of the SDH appears to be supportive of this theory. Moreover, in cases of linear cranial fractures, SDHs may exhibit a migration to the extracranial area, followed by a resolution

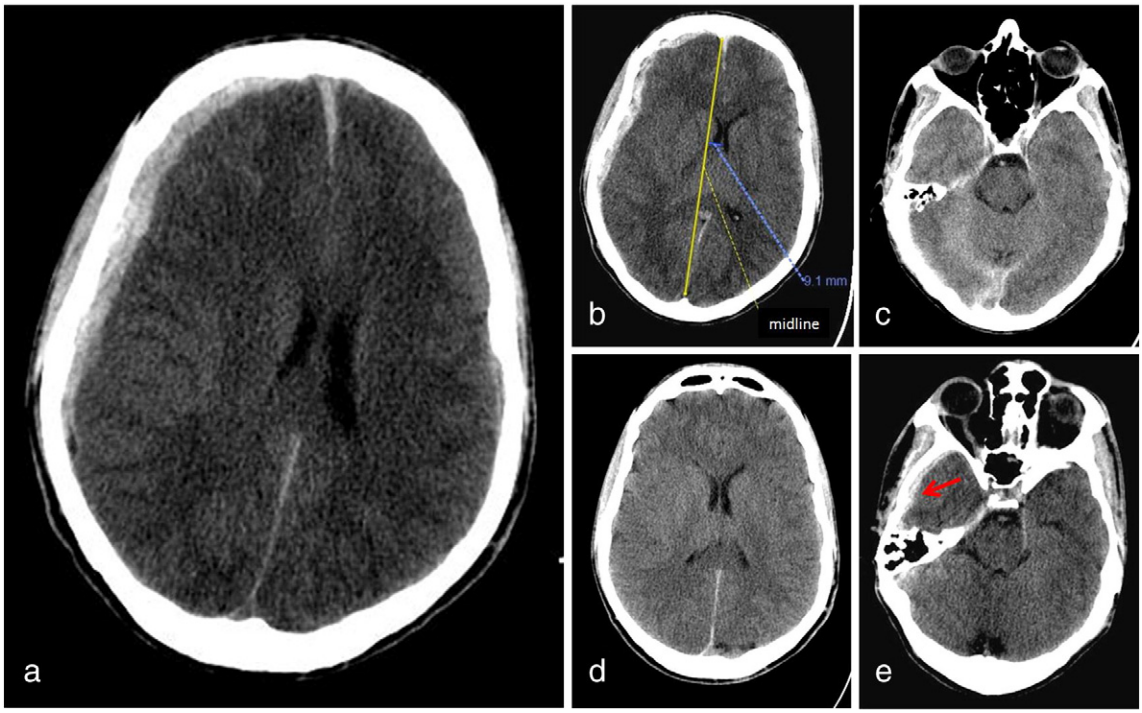

Fig. 1. a to c, CT images on admission. a and b, Axial CT section at convexity level. Acute subdural hemorrhage in the right hemisphere and a midline shift is apparent. c, Simultaneous axial CT section at tentorial level. d and e, Computed tomographic images at 8 hours. Axial sections taken from the same level used in Fig. 1. d, Subdural blood is resolved at convexity level. e, Vague areas of hyperdensity on the cortical surface of the right inferior temporal gyrus at tentorial level, on the left at tentorial level, and on the tentorial free margin facing the pons.

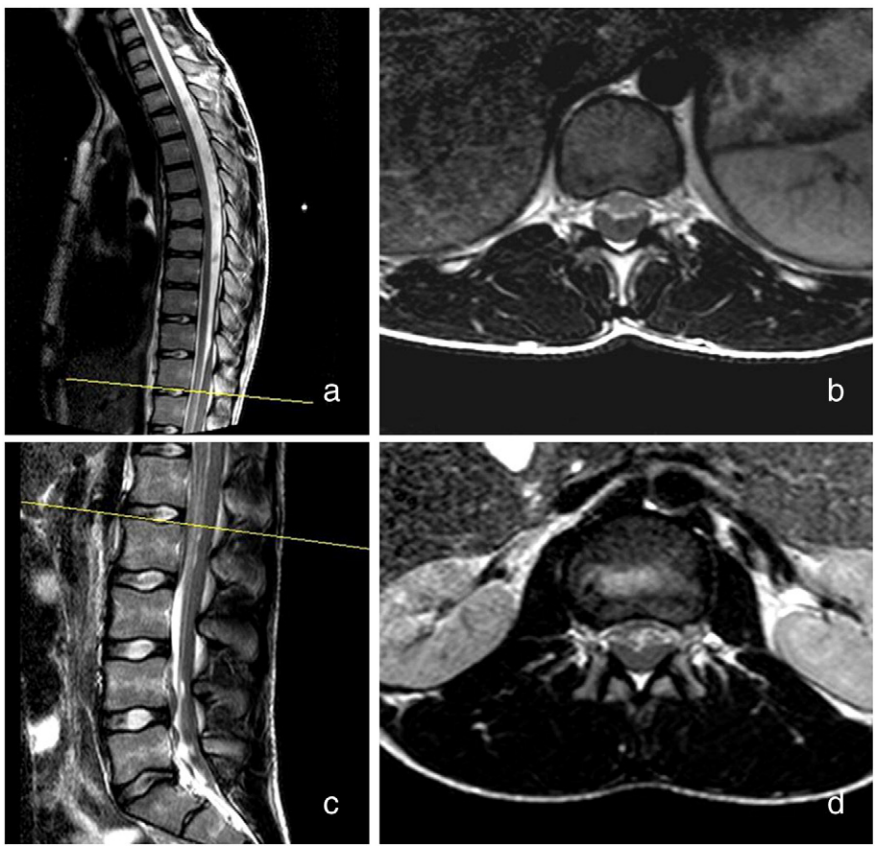

Fig. 2. a to d, Spinal MR images at 48 hours showing the presence of spinal SDH. a and b, T2 sagittal and axial MR images displaying an intradural hypointensity originating from the T10-11 level, filling the canal diffusely and having a thin CSF column between itself and the neural elements. This appearance was diagnosed as acute spinal subdural hemorrhage. c and d, Lumbar sagittal and axial T2 MR images.