SUBACUTE SPINAL SUBDURAL HEMATOMA ASSOCIATED WITH INTRACRANIAL SUBDURAL HEMATOMA

A. SARI (1), B. SERT (1), H. DINC (1), K. KUZEYLI (2)

(1) Department of Radiology, Karadeniz Technical University Faculty of Medicine, Farabi Hospital, Trabzon 61080, Turkey.
(2) Department of Neurosurgery, Karadeniz Technical University Faculty of Medicine, Trabzon, Turkey.

SUMMARY

We describe a subacute spinal subdural hematoma in a patient with post-traumatic subacute intracranial subdural hematoma. CT and MRI demonstrated hematoma within the interhemispheric subdural space and at the lumbar posterior subdural space which extended from the L1 to the S2 level. The lesion showed high signal intensity on both T1 and T2 weighted images. Surgical decompression of the spinal subdural hematoma was performed. The symptoms completely resolved after surgery. Spinal subdural hematoma may be concomitant with or may occur after intracranial subdural hematoma. If a patient with intracranial subdural hematoma complains of low back pain and weakness in both legs; lumbo-sacral MR examination should be performed to exclude spinal subdural hematoma.

Key words: intracranial subdural hematoma, spinal subdural hematoma, Cauda Equina, compression.

INTRODUCTION

Subacute spinal subdural hematoma (SDH) associated with subacute intracranial SDH is an uncommon condition [9-14]. Spinal subdural hematoma is usually associated with trauma, blood dyscrasia, anticoagulation, lumbar puncture and vascular malformation [10-14]. Subacute spinal SDH is also potentially a life threatening condition and cause of spinal cord compression [2, 13, 14]. Most patients with spinal SDH require urgent surgical evacuation, although some cases can be treated conservatively. In this case report, we describe the CT and MRI appearances of a subacute spinal SDH in a patient with an associated subacute intracranial SDH.

CASE REPORT

A 56-year-old man was admitted to the neurosurgery department because of severe head trauma. He had a complaint of headache. Physical examination revealed no focal neurological deficits. Glasgow Coma Scale Score of the patient was 15. Cranial CT showed hyperdense subdural hematoma within the interhemispheric fissure (figure 1a). The patient did not require surgical evacuation of the intracranial SDH. He was treated conservatively. Six days later the patient developed low back pain. The patient had no history of back trauma. Blood tests demonstrated no coagulation abnormality. Physical examination revealed paraspinal muscular spasm, marked tenderness of the lumbar spinous processes and bilateral L4, L5 and S1 radiculopathy. Radiographs of the lumbar spine revealed no abnormality. Eleven days after trauma, computed tomography of the lumbar spine was obtained for lumbago. Computed tomography revealed a hyperdense lesion within the dural sac with a density of 55 Hounsfield units (figure 1b). On the same day, cranial and lumbar spine MR examinations were performed. Sagittal T1-weighted MR images revealed an interhemispheric subdural hematoma that had high signal intensity on both T1- and T2-weighted images (figure 2a). Sagittal and transverse T1-weighted and sagittal T2 weighted fat saturated MR images demonstrated a concave shaped lesion extending from L1 to S2 (figures 2b, 2c and 2d). The lesion had high signal intensity on both T1- and fat saturated T2-weighted images and was pathognomonic for a subacute hematoma. The lesion was separated from the posterior epidural fat by the hypointense dura mater. The subdural collection displaced and compressed the cauda equina ventrally. There was no signal intensity of vascular malformation on the MRI. Surgical decompression of the spinal SDH was performed, and macroscopic examination confirmed
the diagnosis. The symptoms completely resolved after surgery. Follow-up examination of the spine was normal. Follow up cranial CT examination showed decrease in size of the intracranial SDH.

DISCUSSION

The spinal subdural area is not a potential space. The space between dura and arachnoid is filled with neurothelial cells [3-12]. If neurothelial cells break up because of pressure caused by mechanical forces, such as fluid collection creates a subdural space [12]. Subacute spinal SDH with intracranial SDH is a rare entity and only few cases have been reported in the literature [1, 4, 14]. Spinal subdural hematoma is often associated with a trauma, coagulation disorders, lumbar puncture, spinal anesthesia, vascular malformation and after intracranial surgery [1, 5-7, 9]. Patients with spinal SDH usually present with acute onset signs of spinal cord or cauda equina compression, and back or radicular pain. Isolated pain is less
common [2, 4, 5, 13]. The prognosis depends on the location of the spinal SDH and duration of symptoms [4]. Prognosis is poor for lesions located at the cervical or thoracic regions and for patients with symptoms lasting over three months [4, 14].

There are no certain mechanisms for the pathophysiology of spinal SDH, but three theories have been discussed in the literature. First, it has been suggested that spinal SDH results from an indirect force on the intraspinal vessels [4, 6, 10, 11, 13, 14], and that a sudden increase in pressure of within the abdominal and thoracic cavities could raise the pressure in the spinal vessels, particularly the valveless radiculomedullary veins which cross the subdural and subarachnoid spaces. If this force is not neutralized by a simultaneous increase in spinal fluid pressure, rupture of the vessels may occur. Second, spinal SDH may be migrated from the intracranial subdural space [1, 6, 9, 14, 15]. This propagation of blood from the cranial to the lumbar subdural spaces is explained by the anatomic continuity between these subdural spaces. Lecouvet et al. showed the anatomic continuity on the MRI within the posterior subdural space of the cervical and thoracic spine [8]. Lumbar accumulation of blood, leading to the symptomatic compression of the cauda equina, most likely results from the effect of gravity. In our patient, we thought that the spinal SDH may have developed as a result of migration of the intracranial SDH to the spinal subdural space. This finding may suggest that the mechanism of spinal SDH may be associated with intracranial events. The third theory states that an increased pressure from the cranial space may amplify shearing forces between spinal subdural and subarachnoid spaces, so the inner dura may tear and bleed [4]. It should be noted that none of these theories has been proven.

For diagnosis of spinal SDH, CT shows a non-enhancing soft-tissue mass, that is often isodense to spinal cord at the subacute stage [14]. In contrast to intracranial SDH, because of the low contrast resolution technique it is difficult to detect subacute spinal SDH in the thoracic spine with CT. Moreover, using CT, it is difficult to determine whether the hematoma is located in the intradural or extradural spaces [13]. The multplanar capabilities of MRI improve detection of the cranio-caudal extension of the hematoma [4, 10, 13, 14]. MRI is also the method of choice for revealing subacute spinal SDHs, allowing surgical planning with regard to the cranio-caudal extension and dorsoventral location of the hematoma [10]. Thereafter, fat-saturated T1-weighted axial images become necessary for reliable differentiation between epidural and subdural location of the hematoma [10, 13, 14]. In the sagittal plane, spinal SDH is more frequently located at the thoracic and thoracolumbar region [7, 13]. The signal intensity depends on the age of the hematoma [13]. Within 3 days of onset, the spinal SDH becomes isointense to the spinal cord and may present areas of high signal intensity on T1-weighted images. After the first week, spinal SDH gives high signal intensity on both T1 and T2 weighted images as seen in our case. A good visualization of the epidural fat confirms the subdural location of the collection. As for spinal epidural hematoma, the recognition of the dura mater indicates the location of the collection [2, 5, 10, 13-15]. The differential diagnosis of spinal SDH includes disc protrusion, spinal tumors, bleeding in a neoplasm, and extra-axial abscess [5, 14].

Spinal SDH can be treated either by surgery or conservatively. The prognosis for functional recovery is good if the condition is treated before the development of irreversible paralysis [2, 4, 5, 13, 15]. In our case, since the hematoma was urgently surgically evacuated, no neurological deficit developed. It is of note that in the absence of severe or progressive symptoms, spontaneous resolution may be observed, but the patient should be monitored using MRI [2, 4, 9, 15].

REFERENCES