Spontaneous acute spinal subdural hematoma in patient on oral anticoagulant therapy. A rare case report

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ABSTRACT

Introduction: Spontaneous spinal subdural hematoma as a result of coagulation defect is a very rare and serious condition, with an overall incidence of less than 1%. Symptoms can vary according to the level of the bleed. Prompt recognition of the presence of spinal subdural hematoma is crucial for treatment. MRI is the investigation of choice for diagnosis as well as for planning the surgery. The differential diagnosis includes abscess, lipomatosis, significant discal hernia and tumors. There are no definite guidelines for the management of spinal subdural hematomas. In cases with serious neurological deficits, prompt surgical evacuation may lead to the resolution of symptoms and complete neurological recovery.

Case report: We present a case of a 75-years-old man with bilateral lower limb radiculopathy, paresthesias, and spinal claudications for four days, progressing to bilateral paraplegia with urine incontinency. The patient was diagnosed with cauda equina syndrome due to spontaneous spinal subdural hematoma in the region from Th12 to L2. He was immediately operated and the subdural hematoma was evacuated. Minimal recovery was achieved and the patient was referred for further rehabilitation with severe paraparesis and urine incontinence.

Conclusion: Early diagnosis of spinal subdural hematoma is essential for treatment. Any delay of correct diagnosis can lead to a devastating neurological deficit. Spontaneous spinal subdural hematoma is one of the rare conditions where an emergency MRI is indicated and crucial for diagnosis.

INTRODUCTION

Spinal subdural hematoma (SSDH) is exceedingly uncommon condition with incidence lower than 1%. Even rarer finding is spontaneous SSDH. Spinal subdural hematomas are usually associated with trauma,
lumbar puncture, anticoagulant therapy (cause of spontaneous SSDH) or spinal surgery. The prevalence of subdural hematoma in the thoracic and lumbar region as a result of coagulation deficit is a rare cause of spinal cord compression. Symptoms can vary according to the level of the hematoma including pain, lower limb weakness, radiculopathy, paresthesias, paraplegia, and urine incontinency. Prompt recognition of the presence of SSDH is essential for successful treatment (1, 2). MRI is the diagnostic modality of choice for diagnosis as well as for planning the surgical procedure. The differential diagnosis includes severe discus hernia, lipomatosis, abscess, and tumors (1).

We present the case of 75-years-old man with bilateral lower limb radiculopathy and paresthesias lasting for four days and progressing to cauda equina syndrome due to spontaneous subdural hematoma at thoracolumbar junction level.

CASE REPORT
A 75-years-old man presented with progressive bilateral lower limb radiculopathy, paresthesias and lower back pain progressing to paraplegia and urine incontinency in 4 days, after which he was admitted to our institution with complete cauda equina syndrome. There was no history of trauma. However, the patient had aortic valve prosthesis inserted 5 years prior to admission to our institution and was on anticoagulant therapy (Figure 1). His initial international normalized ratio (INR) was 6.020. An initial thoraco-lumbar computed tomography (CT) was performed in another institution two days after the onset of symptoms, and except for L1 vertebral body compression fracture without signs of dislocation, no other pathological finding was described (Figure 2). After neurological deterioration to cauda equina syndrome, patient was referred to our institution. Upon admission MRI of thoracolumbar junction as well as lumbosacral spine was performed and revealed a subdural lesion at the thoraco-lumbar junction level which was hyperintense on T1W imaging and hypointense to spinal cord on T2W image. The STIR sequence showed hyperintensity to spinal cord, so presumptive diagnosis of subdural hematoma was made (Figure 3). Patient was immediately operated and Th12, L1 and L2 laminectomies were performed. The underlining dura was blue, tense, and without pulsations. Upon opening the dura with linear incision semi-liquid, dark blood clot came out under high pressure (Figure 4). The subdural space was irrigated after which flow of liquor was obtained. The dura was sutured watertight and the operation was finished in usual fashion. Postoperatively the patient was painless with minimal improvement of lower extremities strength. No improvement of urine incontinence was obtained. Patient was referred for further rehabilitation 7 days after the operation. Unfortunately, no significant improvement was achieved 2 months after the operation.
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**Figure 3.** Spine MRI performed 4 days after the onset of symptoms. (A) Sagittal T1W image showing Th12-L2 isointense subdural hematoma; (B) Sagittal T2W image showing Th12-L2 hyperintense subdural hematoma; (C) Sagittal STIR sequence showing hyperintense signal of SSDH; (D) Axial T2W showing the “Y” shaped sign of the SSDH at the level of L1; (E) Axial T2W showing SSDH at the level Th12 causing absolute spinal stenosis.

**Figure 4.** Intraoperative finding of SSDH

**DISCUSSION**

SSDH is a rare condition, and most often these hematomas are associated with trauma, lumbar puncture or spine surgery (1). Spontaneous SSDH is exceedingly uncommon finding. De Beer et al. give classification of SSDH into: (1) posttraumatic, (2) iatrogenic (following spinal surgery or lumbar puncture), and (3) spontaneous (associated with underlying vascular malformations or coagulation deficits) (3). We presented a case of spontaneous SSDH due to coagulation deficit with INR 6.020, since the patient was on anticoagulant therapy because of heart valve replacement.

Clinical presentation of spinal subdural hematoma is not specific (1). Severe back pain with a radicular component is often the first complaint. The pain followed by the development of weakness and numbness progressing to a complete sensorimotor paraplegia over a few hours to days is typical clinical finding since most of SSDH are located in thoracic or lumbar region (3). According to study of Dampeeer, spontaneous SSDH are most often located in the thoracic region and presenting with paraparesis or paraplegia, sensory level and pain, and more than 40% are due to coagulation deficit (3, 4). In our case patient had typical clinical picture for lesion located in thoraco-lumbar junction level (Th12-L2) with complete cauda equina syndrome.

MRI is the imaging modality of choice for diagnosis SSDH. The most important factor to distinguish SSDH from other spinal lesions is identification of blood products on MRI. Also, MRI gives better visualization of the longitudinal extent and size of the hematoma. Subdural hematoma can be divided into hyperacute, acute and chronic. The hyperacute bleed is iso/hypointense on T1W sequence and hyperintense on T2W sequence. The acute hematoma is hypo-/isointense on T1W images and hypointense on T2W images. In early subacute hematoma the T1W image is hyperintense and hypointense on T2W images. The late subacute hematoma is hyperintense on T1- and T2- weighted images. The chronic hematoma is usually hypointense on T1W and T2W images (5, 6). Our patient had acute SSDH from Th12 to L2 spinal segment. CT is the workhorse for emergency cases and is usually done before. Unfortunately, SSDH can easily be missed in the acute setting. After having identified a subdural hematoma on MRI, it is good practice to revise the CT scan in an attempt to identify the SSDH (7). In our case initial CT scan was performed in another institution, and except for L1
vertebral body compression fracture, no other pathological finding was described. We revised CT scan after obtaining MRI finding that was suggestive of SSDH, however, no consensus has been reached among 9 neurosurgeons in our department. SSDH is one of the rare conditions where emergency MRI is indicated and crucial for diagnosis. It is important to distinguish between SSDH and other spinal subdural space occupying lesions such as empyema, hygroma, lipomatosis, tumors, and arachnoiditis. The “Y” shaped sign we noted, is similar to the “Inverted Mercedes Benz” sign described by Kasliwal et al. This sign is a result of the encasement of blood around arachnoid lined neural structure. This helps to differentiate between an epidural and subdural location of the hematoma (8).

There are no definite guidelines for management SSDH. The location and symptoms are the most important factors for treatment decision. Treatment involves conservative management in cases with preserved neurology or laminectomy and drainage in cases with serious neurological deficit. Cervical and thoracic SSDH mostly require surgical treatment, while SSDH below the conus medullaris can be treated conservatively. In general SSDH at the cervical or thoracic level are associated with poor outcome. Apart from the location, the duration of symptoms is one of important prognostic factors (9). In cases of spontaneous SSDH due to coagulation deficit, anticoagulant therapy should be stopped immediately (10). Our patient had complete cauda equina syndrome, so laminectomy and evacuation of SSDH was performed immediately after obtaining the diagnosis with MRI. However, due to long duration of symptoms, functional recovery was minimal. In our case we can modify famous neurological maxima “the time is brain“ into “the time is spine“.

**CONCLUSION**

Early diagnosis of SSDH is essential for treatment. So, in patients with cauda equina syndrome and severe back pain, and especially if there is information and evidence of anticoagulant therapy, spine MRI should be performed. If SSDH is confirmed in patient with any neurological deficit we suggest prompt surgical decompression and hematoma evacuation, although there are no definite guidelines for management SSDH and personalized medical approach is always reasonable. Patient's clinical symptoms, the location of the SSDH, the amount of spinal canal narrowing, and duration of symptoms are the most important factors which should be taken into account when choosing the most appropriate treatment.

**REFERENCES**