

A Rare Case of Spinal Subdural Hematoma Complicating Minimally Invasive Lumbar Micro Discectomy and Decompression for Recurrent Lumbar Disc Herniation

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Abstract

Spinal subdural hematomas is a very rare and unusual complication of spinal interventions. We present a case of subacute SSDH in the lumbar region of a 60 year-old woman following microdiscectomy for recurrent lumbar disc herniation. By presenting this rarely seen case of postoperative subacute SSDH, we want to bring attention to the possible postoperative complications like spinal hematomas in the differential diagnosis of failed back surgery syndrome in patients who do not respond to conservative treatment or develop neurological deficits and to the importance of radiological imaging in such cases.

Keywords: Postoperative Spinal Subdural Hematoma, Magnetic Resonance Imaging, Computerized Tomography, Failed Back Surgery Syndrome

Introduction

Spinal hematomas can be epidural, subdural, subarachnoid or intramedullary depending on etiology. Being one of them, spinal subdural hematoma (SSDH) is a rare pathology and develop usually in patients with coagulation disorders or spinal vascular malformations following spinal interventions like anesthesia procedures, lumbar puncture, spinal surgery or after spinal trauma [1-6]. Diabetes mellitus, chronic renal failure, hypertension and alcoholism are the other reasons accused in the etiology [7]. The first case of lumbar SSDH developed after repair of a dural rupture during microdiscectomy [8]. In this report, the presentation, radiological findings and treatment of SSDH developed in a patient who was reoperated due to recurrent lumbar disc herniation was discussed.

Case Presentation

A 60 year old woman who had been operated for lumbar disc herniation 8 years ago and left lower extremity vascular pathology 4 years ago in other hospitals, applied to our clinic with complaints of lumbar and left leg pain, which was present for the last 4 months and did not respond to conservative treatments. Our patient had used oral antiaggregant treatment for a long time until 6 months ago. She were using oral antihypertensive and antidiabetic drugs for hypertension and type-II diabetes mellitus. The clinical examination findings were as follows; left lasague test (+), left leg lift test (30 degrees +) and 30 % strength loss in left foot dorsal flexion. The blood biochemistry was normal except hyperglycemia (216 mg/dL). In lumbar magnetic resonance imaging (MRI), recurrent disc herniation at L4-5 level with compression on L5 nerve root was detected. Following anesthesia preparations, left microdiscectomy at L4-5 level was performed. The operation lasted approximately 1 hour and no complications were observed. The next day, she

was discharged with recommendations. After that, she presented with spasm and pain in the left leg beginning on the postoperative day 14 and was evaluated with lumbar computed tomography (CT). At level of L4-5, laminectomy defect on the left side with accompanying paracentral and neural foraminal air densities were determined. Conservative treatment consisting of analgesic and anti-inflammatory drugs was planned. 3 weeks later, the patient reapplied due to her ongoing complaints. Contrast-enhanced lumbar MRI performed under sedation and revealed a subdural hematoma that was hyperintense on fat suppressed T1W and heterogenous intensity containing both increased and decreased signal intensities on T2W, at the L3-S1 level, consisted with subacute hematoma with widest thickness of 7x12 mm pushing the fibers of filum terminale towards the front (Figures 1, 2, 3 & 4). In addition, at level of L4-5, near the dural sac and L5 nerve root there was decreased signal intensity on T1W and increased signal intensity on T2W, which was evaluated as granulation tissue. Since there was no neurological deficit, conservative treatment was recommended and her complaints decreased day by day. In the control MRI performed on postoperative day 45, complete resolution of the hematoma was seen with total recovery of complaints of the patient.



Figure 1: a subacute subdural hematoma that was hyperintense on fat suppressed sagittal T1W at the L3-S1 level



Figure 2: a subacute subdural hematoma that was heterogenous intensity containing both increased and decreased signal intensities on sagittal T2W at the L3-S1 level

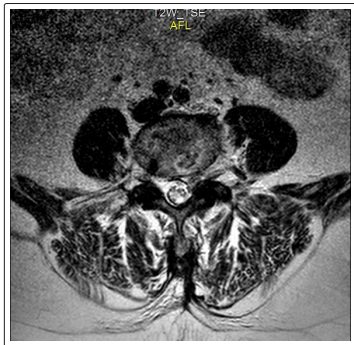


Figure 3: a subacute subdural hematoma that was heterogenous intensity containing both increased and decreased signal intensities on axial T2W



Figure 4: a subacute subdural hematoma that was hyperintense on fat suppressed axial T1W

Discussion

SSDH can be idiopathic, but more frequently develops in patients with coagulopathy or vascular malformations following spinal interventions including spinal surgery, lumbar puncture, spinal anesthesia procedures or spinal trauma [7-9]. In the literature there are reported cases of SSDH following lumbar microdiscectomy, laminectomy with discectomy, only decompressif laminectomy and percutaneous vertebroplasty [10-14]. It was also reported to be developed associated with hypertension, pregnancy, infection or lumboperitoneal shunt administration [6,9,15]. Less often it can develop spontaneously after coughing, sneezing [5].

In our case, SSDH occurred after microdiscectomy performed due to

recurrent lumbar disc herniation. However, our case had previously been operated for lower extremity vascular pathology and had received oral antiaggregant treatment for a long time, and had type II diabetes.

The pathophysiology of postoperative SSDH is not clear yet. Electron microscopic studies reported that SSDH may have the same developmental principle with intracranial subdural hematoma (ICSDH) and might also develop as an extension of ICSDH as a result of gravity [16-20]. There are various theories about the source of SSDH. Vessels under dura matter occupy as an anastomosis network having a longitudinal course. However, the small calibration of these vessels in the spinal region limit development of an important subdural hematoma [9]. Some authors have stated that lumbar radiculomedullary vessels with larger caliber were injured as a source of hemorrhage. These vessels accompany L4 and L5 nerve roots and pierce the dura matter laterally in order to enter subarachnoid space [14,17,21]. During laminectomy while dissecting adherent dura matter hemorrhages may occur due to tearing of these vessels. In our case, there was lots of granulation tissue due to the previous discectomy procedure making dissection more difficult. It was thought that the same mechanism occurred in our case. The symptoms of SSDH are sudden onset, severe back and lomber pain that may be accompnied by radicular symptoms. Paresis or sensory loss can develop due to the acute compression of spinal cord or cauda equina [10,22]. SSDH is defined as acute when symtoms occured in one week, as subacute if symtoms developed after 1 week and as chronic if symtoms last more than 1 month [9]. Subacute SSDH can present with slowly developing paraplegia in about 1 week [23]. In our case symptoms appeared also 1 week after the operation but there was no neurological deficit. The more frequent type of hemorrhage in spinal region is spinal epidural hematoma (SEDH). In differential diagnosis clinical findings alone may not be helpful without radiological evaluation. Computerized tomography (CT) especially in the subacue period has limited capacity due to the isodensity of subacute blood with thecal sac and spinal cord. Therefore, intrathecal contrast enhanced CT is a better option for diagnosis [22,24]. MRI is the most appropriate imaging modality for diagnosis and treatment planning [11,17,18,25,26]. MRI is also valuable in discrimination of SSDH from other intradural extramedullary pathologies and in the monitoring of the hematoma [4,17,27-29]. Addition of sagittal T2W GRE sequence make easier to establish the level of hematoma with great accuracy [30]. In the differentiation of SSDH from SEDH, axial images are very helpful. SSDH is located more frequently in the anterior part of the spinal cord or semicircularly with a large ventral part and extends in a long segment in the craniocaudal axis. In SSDH the dura matter appears smooth and do not show continuity with bony structures. The characteristic MRI finding is the presence of abnormal signal intensities in the dural sac [31]. In our case, CT was performed firstly due to the MRI phobia of the patient and no pathological findings were found. Following persistant severe radicular pain nonresponsive to conservative treatment, MRI performed with the aid of sedation in postoperative 5th week. Subacute SSDH that was hyperintense on T1W was established. SSDH should not be considered as an indication for revision surgery at first line and conservative treatment should be considered in patients without neurological deficits [3,32]. Most cases of SSDH resorbe spontaneously or can be treated with lomber spinal tap or drainage [24,29]. Steroid treatment can be applied in SSDHs causing radicular pain [1]. When progressing neurological deficits are present, emergent surgical intervention is

necessary [25,27]. Especially in patients with neurogenic deficits hematoma should be discharged immediately by opening dura matter via laminectomy to preserve nerve functions and to gain back the deficits. In our case conservative treatment was applied because of the absence of neurological deficits.

As a result, after a spinal intervention or operation, when a progressing radicular pain and/or progressing neurological deficits appeared, it should be come to mind that this can be due to spinal hematoma as well as a component of failed back surgery syndrome and should be further evaluated immediately. Patients with SSDH without accompanying neurological deficits should be treated conservatively and monitored radiologically at first line.

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