

# Spontaneous Lumbar Subdural Haematoma - A Case Report

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## ABSTRACT

Spinal subdural haematomas are a rare cause of spinal cord or cauda equina compression. These are usually seen in association with lumbar puncture or coagulopathy. Spontaneous spinal subdural haematomas are even rarer. This report presents one such case diagnosed by magnetic resonance imaging (MRI) together with a review of the literature. The patient presented with low back pain of acute onset after minimal trauma. MRI showed high signal intensity on both T1-weighted and T2-weighted sequences in the posterior subdural space indicative of a subacute lumbar subdural haematoma. There was no history of bleeding diathesis. The patient was treated conservatively and recovered spontaneously.

**Keywords:** spontaneous, spinal subdural haematoma, MRI

## INTRODUCTION

Spinal subdural haematoma (SSH) is rare. In 1983, Russell et al<sup>(1)</sup> reported 58 spinal subdural haematomas of which 6 occurred in the lumbar or lumbosacral region. He divided SSH into 3 grades of chronicity depending on their clinical course. Sixty-seven percent of his cases were acute, with sudden onset of severe back pain, followed by immediate or rapidly progressive paraparesis with paraplegia within 24 hours. Eleven percent were subacute, typically presenting with back pain progressing to radicular pain, paraparesis and paraplegia within 2 weeks. Chronic cases accounted for 22% and were characterised by slow progressive cord compression over months to years, with little or no back pain.

Most cases of SSH reported have been in patients with bleeding diathesis or lumbar puncture. However, there have been occasional cases in patients with no predisposing factors. Recently, there have been a few reports of spontaneous SSH diagnosed by MRI<sup>(2-4)</sup>.

## CASE REPORT

YCT, a 52-year-old Chinese male taxi driver, presented with a 2-week history of low back pain. This began suddenly whilst he was changing a car tyre. The pain was spiking in nature and associated with bilateral sciatica. It was aggravated by exertion, coughing and sneezing. It was worse in the morning and relieved by lying down. There was no paraesthesia, bladder or bowel symptoms. There was no history of hypertension, fever, bleeding diathesis, recent spinal injection or lumbar puncture.

On physical examination, he was afebrile, pulse rate was 80 beats per minute and blood pressure was 110/70 mmHg. There was limitation of spinal flexion. Straight leg raising was 30° on the right and 45° on the left. Limb power, sensation and reflexes were normal. A clinical diagnosis of a prolapsed intervertebral disc was made.

Blood investigations showed a total white count of 10,400 per mm<sup>3</sup> and a normal differential count. The prothrombin time was 10.9 seconds (control 12 seconds) and the partial thromboplastin time was 26 seconds (control 25.5 seconds).

Magnetic resonance imaging (MRI) was performed and this showed high signal intensity in the subdural space on both sagittal T1-weighted and T2-weighted sequences extending from the level of L2 down to S2 (Figs 1 and 2). This showed as a crescentic or biconvex area of bright signal on the axial T1-weighted scans (Figs 3a and 3b). Findings were that of a subacute spinal subdural haematoma.

In the ward, the patient was treated with intramuscular pethidine 75 mg 6 hourly for 24 hours, a single dose of intramuscular diazepam, hot pack, short wave therapy and lumbar traction. His symptoms improved dramatically and straight leg raising was 70° bilaterally the next day. He was treated

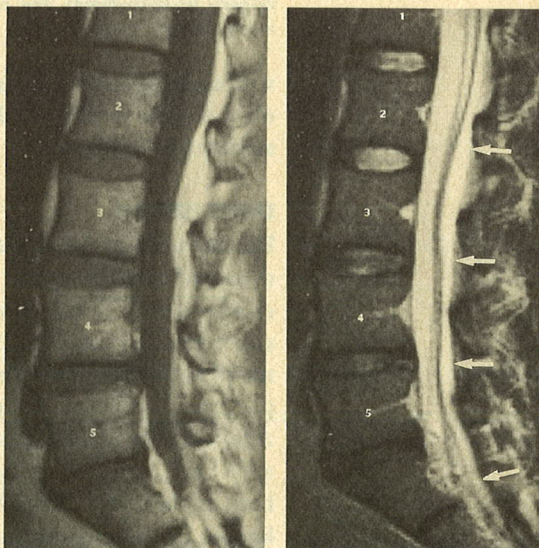


Fig 1

Fig 2

**Fig 1 and Fig 2** - Sagittal T1-weighted (TE 17ms, TR 850ms) and T2-weighted fast spin-echo images (TE 90ms, TR 5000ms) respectively. There is a high signal intensity collection in the posterior aspect of the spinal canal extending from the L2 to the S2 (arrowheads). This is indicative of subacute haemorrhage. There is also desiccation of the lower 3 discs with an annular tear at L4-5.

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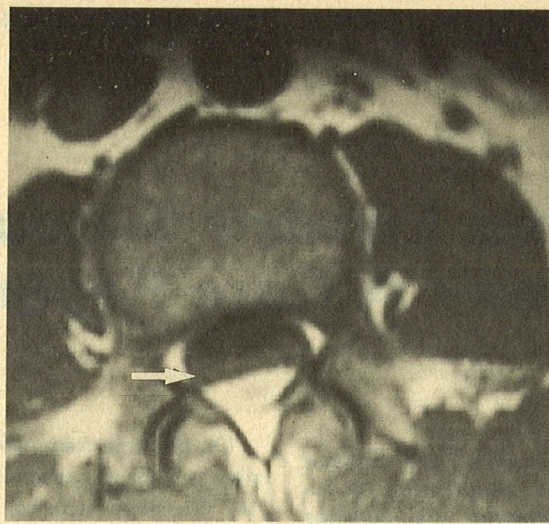


Fig 3a

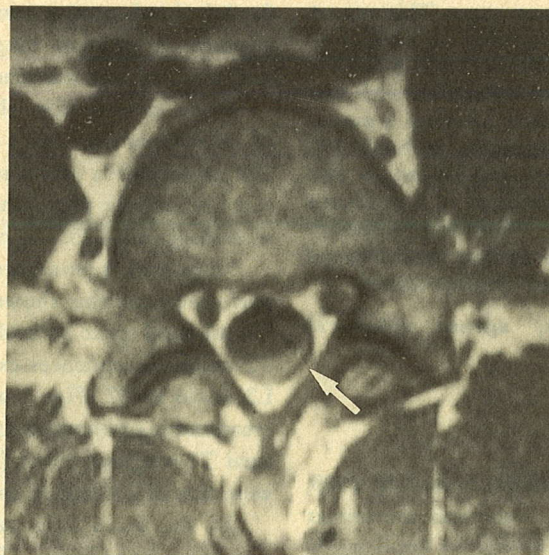


Fig 3b

**Fig 3 (a,b)** - Axial T1-weighted spin-echo images (TE 15ms, TR 740ms) demonstrate crescentic or biconvex high signal posteriorly. It is displacing the cauda equina anteriorly (arrow). It is separated by a thin low signal line from the high signal in the epidural fat (arrowhead). This confirms that the haemorrhage is in the subdural space.

with analgesics and oral diazepam and was discharged on the sixth day after admission. He was well on subsequent follow-up.

#### DISCUSSION

Haematomas in the spinal canal are known to be a cause of sudden spinal cord and cauda equina compression. They have been associated with ruptured vascular malformations, underlying neoplasms, hypertension, coagulopathy, trauma, pregnancy, old age, infection and spinal surgery. Anticoagulant therapy, especially in combination with spinal punctures or epidural anaesthesia, has often been implicated<sup>(5-7)</sup>. Spinal haematomas have also been known to occur spontaneously or after only minor activities such as sneezing or coughing. Amongst the spinal haematomas, epidural haematomas are by far the most common. Spinal subdural haematomas are infrequent and spinal subarachnoid haematomas are even rarer. For this reason, imaging of these lesions has received little attention until recently.

Many workers believe that spinal subdural haematomas often arise within the subarachnoid space, rupturing the leaves of the arachnoid mater and organising into a solid mass in the subdural space<sup>(1,8)</sup>. Prompt recognition of SSH is important. Clinically, it presents with sudden backache with radicular pain followed by signs and symptoms of spinal cord dysfunction clinically indistinguishable from an acute intervertebral disc herniation. Surgical drainage is usually necessary to preserve bladder and bowel functions. Percutaneous drainage has been performed successfully with relief of symptoms and signs<sup>(2)</sup>.

In the past, most cases have been diagnosed by myelography. However, it may sometimes be difficult to differentiate epidural and subdural haematomas on myelogram. MRI can effectively demonstrate whether the haematoma is in the subdural or epidural space. In addition, blood can be distinguished from soft tissue masses. It is also non-invasive. It is better than computed tomography (CT) for detecting subacute and chronic bleeds which may be missed by CT as they become progressively isodense with cerebrospinal fluid. Therefore, MRI where available, should be the investigation of choice in suspected SSH.

On MRI, the appearance of spinal haematomas varies with the age of the clot as in the brain. The features of evolving epidural haematomas have been described by Rothfus et al<sup>(9)</sup>. Although the MR appearance of blood in the epidural and intramedullary spaces has been described, there has been no description of the appearance of evolving spinal subdural haematomas except for a few case reports of the MR appearance of acute and subacute subdural haematomas.

Post et al<sup>(3)</sup> described 3 cases of acute spinal subdural collections on MRI. All cases were imaged within 24 hours of onset of symptoms. On T1-weighted images, the acute SSHs were heterogeneous in signal intensity with isointense to slightly hyperintense signal and sometimes also hypointense signal. On T2-weighted and gradient-echo images, they demonstrated areas of hypointensity, isointensity and hyperintensity with striking low signal intensity in a significant portion of the haematomas thought to be due to the presence of deoxyhaemoglobin.

Johnson et al<sup>(6)</sup> reported a case of a subacute SSH (imaged approximately 9 days after symptom onset) which appeared hyperintense on T1-weighted images and slightly hypointense on T2-weighted images. Levy et al<sup>(2)</sup> had 2 cases of subacute SSHs presenting 10 days and 2 weeks after symptom onset. The first case was high signal intensity on gradient-echo images and low signal intensity on the T2-weighted images. The low signal on T2-weighted images was thought to be due to acute rebleed as it was also hyperdense on CT. The second case showed high signal intensity on both T1-weighted and T2-weighted sequences.

In our case, the subacute SSH was imaged about 2.5 weeks after the onset of symptoms. It showed extensive high signal intensity on T1-weighted and T2-weighted sequences lining the posterior aspect of

the spinal canal. The high signal enveloped the low signal intensity cerebrospinal fluid on T1-weighted images. On axial images, the subdural location was confirmed by a thin line of demarcation between haemorrhage and dorsal epidural fat<sup>(10)</sup>. This contrasts with spinal subarachnoid haematomas which surround the cord and nerve roots. Epidural haematomas can be localised or extend over several segments but they do not tend to be as extensive as SSH. A component of the epidural haemorrhage may extend into the neural foramina.

Based on the above reports, the author has found that the MR appearance on high-field MR of acute and subacute SSH closely follows the evolutionary pattern seen with intracranial haematomas (ICHs)<sup>(11)</sup> as described by Gomori et al. In order to associate the MR characteristics of SSHs with ICHs, the author has re-classified the SSHs according to the classification of ICHs rather than using the clinical classification proposed by Russell et al. Haematomas are considered to be acute when less than 1 week old, subacute between 1 week and 1 month old and chronic when more than 1 month old. Acute SSHs tend to be either isointense to slightly hyperintense on T1-weighted sequences and mixed on T2-weighted sequences but with a significantly low signal component. This is probably due to deoxyhaemoglobin in intact red blood cells<sup>(11)</sup>. Subacute SSHs are high signal on T1-weighted images. On T2-weighted images, they can be low or high signal intensity. The low signal intensity is probably seen in early subacute haematomas due to intracellular methaemoglobin and the high signal intensity in late subacute haematomas due to extracellular methaemoglobin. There have been no reported cases of chronic SSHs. Johnson et al<sup>(9)</sup> described an inverted "mercedes sign" as characteristic of spinal subdural haematoma on MR. However, in the literature, this does not appear to be a common finding.

The differential diagnosis of SSH is a spinal subdural abscess. This is also a very rare entity. To the author's knowledge, there are only 48 cases in the medical literature. The MR findings have been described in only 2 cases, one in the Japanese literature<sup>(12)</sup> and one in the English literature<sup>(13)</sup>. Although they did not state specifically, both reports seem to suggest that the subdural abscess is hypointense or isointense on T1-weighted images. The latter case report by Sathi et al showed diffuse enhancement after Gadolinium administration with a central area of non-enhancement presumably due to necrosis. Subdural abscesses can usually be differentiated from SSH by the presence of fever and leucocytosis. The MR appearances of spinal epidural abscesses are well described. On MR, the epidural abscess can be isointense or hyperintense on T1-weighted images and hyperintense on T2-weighted

images<sup>(14,15)</sup>. If subdural abscesses behave similarly to epidural abscesses on MR then it is conceivable that subdural abscesses may mimic SSHs on MR.

Our patient's symptoms and signs resolved spontaneously. This is rather unusual in SSH. It does however emphasize the importance of making a definitive diagnosis. Patients with less severe involvement can be spared unnecessary surgery and be treated either conservatively or by percutaneous drainage.

## CONCLUSION

Spontaneous SSH is a rare entity. MRI provides an accurate and non-invasive means of confirming the subdural location. Based on the findings of this case and the previous reports of high-field MR findings in acute and subacute SSH, the evolution of clot in the subdural space appears to correlate well with the evolution of intracranial haematomas. MR can therefore also help to estimate the age of the haematoma.

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