Spinal Epidural Hematoma Caused by Extradural Arteriovenous Malformation: A Case Report and Review of the Literature

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Abstract

About 330 cases of spinal epidural hematoma have been reported in the literature but few cases had pathologically proven extradural arteriovenous malformation. The authors report a case of spinal epidural hematoma caused by extradural arteriovenous malformation. The patient presented with a sudden onset of back pain followed by rapidly progressive neurological deficit. MRI was the procedure of choice for diagnosis of this lesion. Treatment was emergency surgical decompression. Prognosis depends on the preoperative neurological deficit, operative interval and localization of the hematoma.

Key word : Spinal Epidural Hematoma, Extradural, Arteriovenous Malformation, Case Report

Spinal epidural hematoma (SEH) has rarely been seen in clinical neurosurgery with an estimated incidence of approximately 0.1 patients per 100,000 patients per year[1]. Since 1869, when Jackson first reported a case of idiopathic spinal epidural hematoma[2], about 330 cases have been reported[3] including all causes of SEH such as coagulopathy, anticoagulant therapy, blood dyscrasia, trauma with or without fracture and dislocation, vascular anomaly, neoplasm, postoperative spinal surgery and iatrogenic causes. SEH may be associated with minor trauma or straining. Vascular causes made up 5-10 per cent of all SEH[4-8] and a few reports had pathologically proven extradural arteriovenous malformations[4,9-13]. SEH can occur in patients of any age but is most common in patients over 50 years[5,14,15]. It is more common in males than females[3,5,8,16-20]. The onset of symptoms is primarily acute, however, some patients present with subacute or chronic onset. SEH occurs in any level of the spinal cord but is most common in the thoracic area[20]. Diagnosis includes careful history taking, physical examination and investigations such as myelography, CT, CT myelography, and MRI.

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MRI is the modality of choice for diagnosis of spinal cord lesions. Treatment is emergency surgical decompression[21,22] but a few patients benefit from conservative management[23]. Neurological outcome depends on the severity of preoperative neurological deficits, operative interval and localization of the hematoma[3,10,16,20,24-27]. We report a case of SEH caused by extradural arteriovenous malformation and review the correlating literature.

CASE REPORT

A 47 year old male presented with sudden onset of interscapular pain and progressive paraplegia occurring within 1 hour. He had experienced low back pain 10 months prior to the episode with symptoms disappearing 9 months later. Cause of pain was suspected to be lumbar herniated nucleus pulposus and was treated with medical and physical therapy. He had no history of bleeding diathesis, medication and genetic syndrome. Physical examination revealed a normal general appearance. Neurological examination revealed good consciousness, normal cranial nerve function, however, paraplegia was found. Upper extremity had normal muscle power. All sensory modalities were lost below T5 level. Deep tendon reflexes were absent. Laboratory analysis revealed slight leukocytosis, normal liver function test and only slightly high blood sugar.

MRI of the thoracic spine revealed a posterior extradural mass at the T3-4 level. (Fig. 1-4) It was an isosignal intensity on the T1 weight image and a heterogeneous signal intensity on the T2 weight image. There was an abnormal signal intensity the same as the mass, along the posterior aspect of the thecal sac extending from the upper portion of the mass to the C2 level. This signal abnormality was compatible with acute blood. T3-5 laminectomy was performed 8 hours after onset and an epidural blood clot along the posterior aspect of the

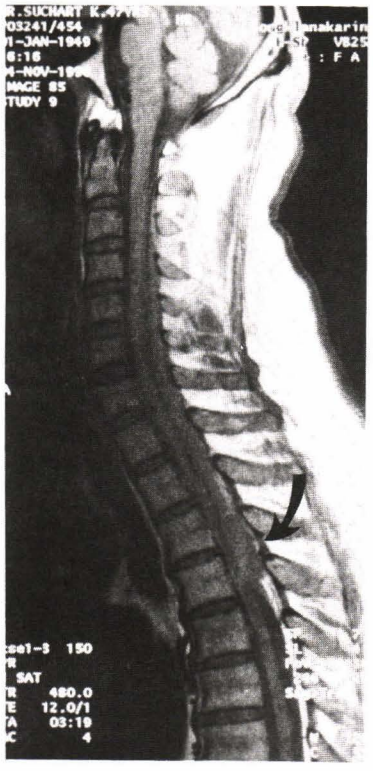


Fig. 1. MRI, sagittal T1 weight image show an isointense signal intensity mass at posterior aspect of the T4-5 level. (arrow)

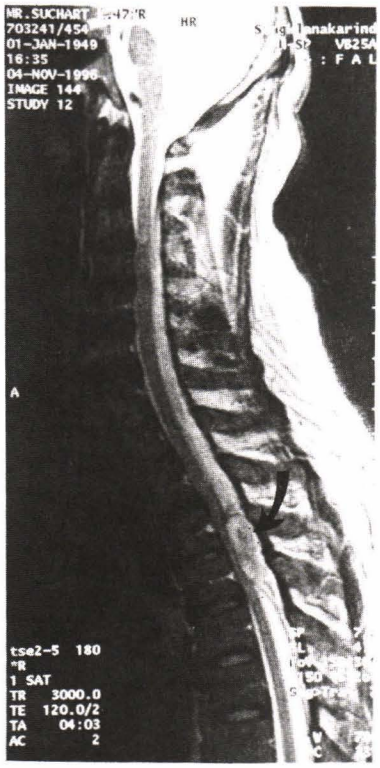
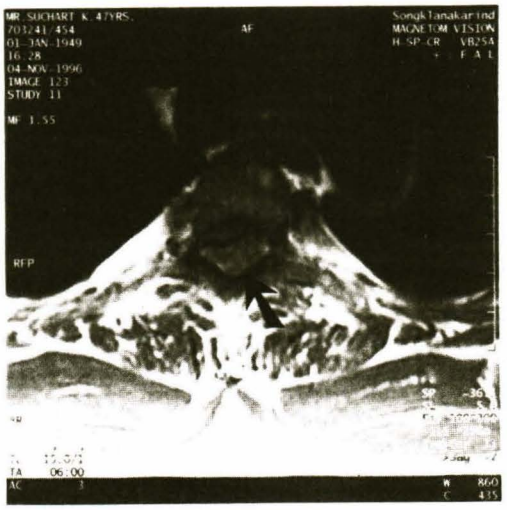


Fig. 2. MRI, sagittal T2 weight image show a heterogeneous signal intensity at the same level. (arrow)



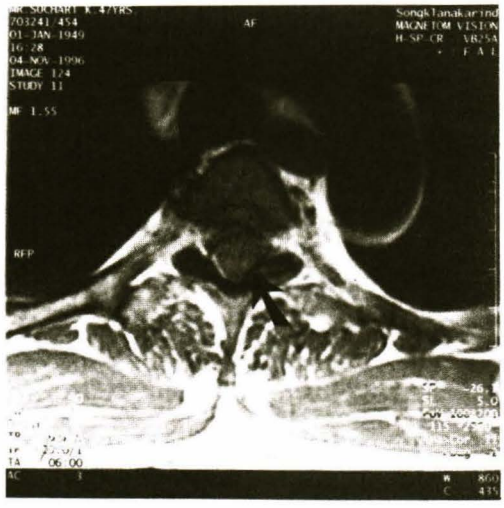
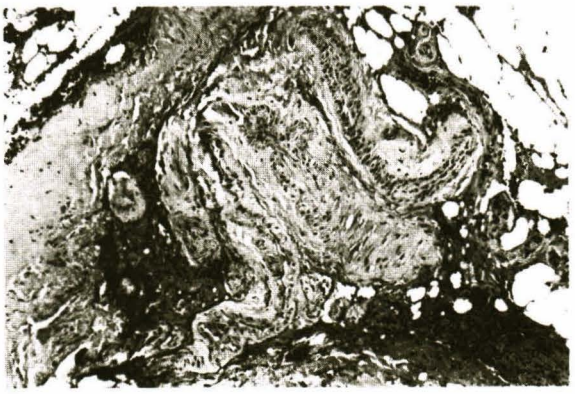


Fig. 3 & 4. MRI, axial T1 weight image show the mass (arrow) displaced thecal sac anteriorly.



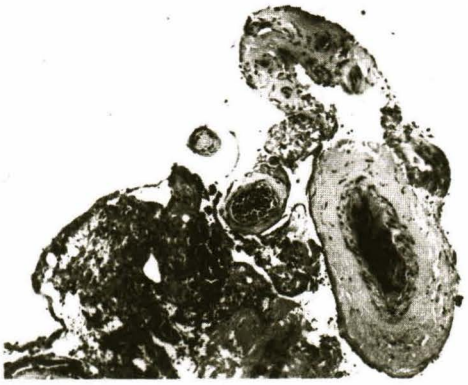


Fig. 5-7. The figures show various structurally abnormal vessels typical of arteriovenous malformation. The vessels are abnormally large and tortuous (Fig. 5 & 7). Thick-walled vessels due to collagenous hyalinization are seen in Fig. 6. The muscular media varies in thickness (Fig. 7).

thecal sac was found. There was a 2-cm mass at the T3-4 level, well circumscribed and highly vascularized. It was confined to the extradural space and not attached to the dura. After removal of the mass, intradural exploration was completed with no abnormality. Pathological report on the mass showed an arteriovenous malformation. (Fig. 5-7) The patient had no return of motor power and slight sensory improvement to T10 level after 16 months of follow-up.

DISCUSSION

Spinal epidural hematoma (SEH) has rarely been seen in clinical neurosurgery. Since Jackson first reported it in 1869,[2] about 330 cases have been reported in the literature[3]. SEH occurred more commonly in males than females with the male-to-female ratio of 1-2:1[3,5,6,8,16-20]. Patients varied in age from 7 months to 90 years [3,5,8,16] with the mean age about 42-50 years [3,5,8,16,17,20,28,29]. SEH usually occurred in patients who were more than 50 years old[5,14,15]. Symptoms, in more than 50 per cent of cases, were acute onset of severe localized back pain and radicular pain[15,18,30]. These complaints were followed by neurological deficits such as complete or incomplete cord lesions. There were some reports with Brown-Sequard syndrome[31-36], anterior cord syndrome[10], Horner's syndrome[35,37] and Cauda Equina syndrome[38,39]. There were a few cases presenting with sudden severe localized back pain and no neurological deficit. Sphincter disturbance occurred in many patients. These neurological deficits presented most often within 3-12 hours after the onset. In traumatic SEH, the symptoms consisted of progressive neurological deterioration after trauma[40]. Children presented with meningeal signs, irritability, mild fever, ataxia, abdominal discomfort and whooping cough[41]. Laboratory tests should be completed for coagulogram. Plain film was non-diagnostic for SEH. Diagnostic procedures of SEH include myelography[42], CT with or without myelography[35,43-45] and MRI[1,35,46-50]. Myelography demonstrated only CSF blockage. MRI is the procedure of choice for spinal cord lesions. It is a noninvasive, sensitive, specific method to evaluate the lesions. In the first 24 hours, SEH produced an isosignal intensity on T1 weight image and heterogenous signal intensity on T2 weight image. After 24 hours, it produced a hypersignal intensity on both T1 and T2 weight image.

The etiology of SEH was unknown in 40-50 per cent of the cases[6,8,16,18] but this could not exclude cryptic vascular malformation[41]. The other etiology were anticoagulant therapy (30-50 per cent)[19,24,26,45], bleeding diathesis from congenital syndrome such as hemophilia[47,51,52], factor IX[53] and factor XI deficiency[33]. Vascular anomalies were found in a few patients (5-10 per cent) [4,7,8], especially arteriovenous malformations, a few reports were pathologically proven[4,9-13]. Some literature has shown that trauma[40], neoplasm and iatrogenic causes such as postoperation, LP or epidural anesthesia can cause SEH[20,54-56]. However, there were some factors associated with the occurrence of SEH; such as minor trauma[8], sneezing[57], coitus[58], whooping cough[59], pre-existing hypertension[5], pregnancy[60,61], Paget's disease[38,62,63], liver disease[64], excessive garlic ingestion[65] and SLE[66].

The most common location of SEH was the thoracic area[20,45]. Lumbar and cervical SEH occurred with the same frequency[20]. In children, the most common locations were the cervical and upper thoracic areas[8]. In males, the most common locations were the lower cervical and thoracolumbar areas in contrast to the lower thoracic area in females[50]. Most SEH occurred at the posterior aspect of the spinal cord (99 per cent), however, a few patients with ventral SEH were reported (1 per cent)[5]. These may be due to many vascular channels in the posterior aspect of the thecal sac. SEH usually involved 2-3 spinal segments[16,57], but it sometimes extended along the thecal sac also[67].

Differential diagnosis included acute ruptured disc, epidural neoplasm, acute transverse myelitis, epidural abscess or vascular accident such as occlusion of anterior spinal artery, ruptured intradural malformation, cord ischemia, hematomyelia, subdural hematoma, dissecting aneurysm, congenital cyst, spondylitis and torticollis[28,44,49,68-70].

Pathophysiology of bleeding in SEH was controversial. Most authors suggested that bleeding was from the venous system because of a weakened or fragile epidural venous plexus, thin wall and valveless vein[3,11,60,71-73]. These occurred with sudden increased intravenous or thoracoabdominal pressure. However, the pressure from venous bleeding should not be higher than intrathecal pressure. It should not produce cord compression. Arterial bleeding may cause neurological deterioration in SEH[56,68] especially with hemangioma, AVM or aneurysm[1]. There was some discussion about cryptic vascular malformation in idiopathic SEH[74].

The generally accepted treatment of SEH was emergency surgical decompression[21,22]. There were, however, a few reports of nonoperative management of SEH. Some authors suggested nonoperative management in patients who had no neurological deficits[23] or who had earlier experienced neurological recovery with or without radiological confirmation[75]. They suggested close fre-

quent neurological observation[26,49] or monitoring somatosensory evoke potential (SEP) every 6-8 hours for 24-36 hours[51]. Spontaneous recovery without surgical decompression has been reported (6,9,21,31,58,75-78).

Results of treatment for SEH consisted of 0-21 per cent mortality rate[3,8,20,68], 30-50 per cent complete recovery[8,79] and 10-24 per cent not improved[3,8,80]. Mortality rate was 6.9 per cent of patients with an incomplete sensorimotor lesion and 22.6 per cent of complete sensorimotor lesion[16]. 35 per cent of surgically treated patient were able to walk[27]. Complete recovery occurred in one half of the patients who were operated on within 24 hours after onset. The percentage dropped to 30 in the patients who received surgery longer than 24 hours after onset[50] and no expected recovery if surgery was delayed longer than 30 hours[58]. McQuarrie IG[27] reviewed 32 surgical cases of spinal epidural hematoma and found that the probability of recovery was less than 50 per cent when surgery was delayed more than 36 hours. In the patients who received surgery within 8 hours after onset, 84 per cent of them experienced remission[18]. So, early diagnosis and treatment should be completed.

Prognosis after treatment depended on many factors. The most significant factors were the severity of preoperative neurological deficits[3,9,16,20,24,25,28], the time interval between onset of neurological dysfunction and surgery (operative interval)[3,10,20,24,26-28] and the localization of the hematoma[3]. Foo D, et al[16] collected 158 cases of spontaneous spinal epidural hematoma treated surgically. Complete sensorimotor recovery occurred in 41.9, 26.1 and 11.3 per cent of the patients with incomplete sensorimotor, incomplete sensory but complete motor and complete sensorimotor lesion, respectively. Motor function after surgery improved in 95.3, 87 and 45.3 per cent of the patients with incomplete sensorimotor, incomplete sensory but complete motor and complete sensorimotor lesion, respectively. Groen RJM, et al[3] reviewed 330 cases of spontaneous spinal epidural hematoma. They found that the critical factors for recovery were the localization of the hematoma, preoperative neurological deficit and the operative interval. Patients with incomplete preoperative sensorimotor deficit correlated highly with favorable outcome. Recovery was significantly better when surgical decompression was performed within 36 hours in patients with complete sensorimotor loss and within 48 hours in patients with incomplete sensorimotor deficit. Lawton MT, et al[20] reported 30 patients with surgically treated spinal epidural hematoma. They concluded that preoperative neurological status and operative interval correlated with outcome. So, rapid diagnosis and emergency surgical decompression maximized neurological outcome. Younger patients and those with chronic compression and lumbosacral area SEH seemed to have a better prognosis[16,75] but many authors concluded age, hematoma site and size, duration of symptoms and delayed surgery had no influence on patient outcome[3,20,25]. However, operation should be performed in every patient even when there is a complete neurological deficit[3,20].

SUMMARY

We report a case of SEH caused by extradural AVM who presented with acute onset of pain and complete neurological deficit. A few reports could have pathologically confirmed extradural AVM as in our case. MRI was the modality of choice for diagnosis of the spinal cord lesion. After surgery, this patient did not recover from neurological deficits. We, therefore, think that prognosis depends on many factors. Failure of neurological recovery may be due to direct traumatic damage to the spinal cord from epidural hematoma.

(Received for publication on May 7, 1998)

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ก้อนเลือดเหนือดูราที่ไขสันหลังจากหลอดเลือดผิดปกติ: รายงานผู้ป่วยและทบทวนวารสาร

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มีรายงานผู้ป่วยก้อนเลือดเหนือดูราที่ไขสันหลังจากสาเหตุต่างๆประมาณ 330 ราย แต่มีผู้ป่วยจำนวนน้อยมากที่มีผลชิ้นเนื้อทางพยาธิวิทยาพิสูจน์ว่ามีสาเหตุจากหลอดเลือดผิดปกติ ผู้เขียนได้รายงานผู้ป่วยก้อนเลือดเหนือดูราที่ไขสันหลังที่มีสาเหตุจากหลอดเลือดผิดปกติและได้ผลพิสูจน์ชิ้นเนื้อทางพยาธิวิทยา ผู้ป่วยมาพบแพทย์ด้วยอาการปวดหลังทันทีทันใดและมีความผิดปกติทางระบบประสาท การตรวจด้วยคลื่นแม่เหล็กไฟฟ้าเป็นวิธีที่ดีที่สุดในการวินิจฉัยภาวะนี้ การรักษาที่เหมาะสมคือการผ่าตัดเลาะก้อนเลือดออกให้เร็วที่สุดเท่าที่จะเป็นไปได้ การพยากรณ์โรคขึ้นอยู่กับความรุนแรงของความผิดปกติทางระบบประสาท, ระยะเวลาตั้งแต่มีความผิดปกติทางระบบประสาทจนถึงการผ่าตัดและตำแหน่งของก้อนเลือด

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