# Spontaneous epidural hematomas of cervical spine. Report of our cases

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## **Summary**

In this retrospective study we report four cases of spontaneous cervical spine epidural hematomas (SCSEH) during last twelve years and review the relevant literature. The average age of our patients was 56 years, all had various sensorimotor neurological deficits, and two of them had certain predisposing factors. T1 and T2 sequences of spinal MRI varied on clot characteristics, age, size, and oxygenation of the hematomas. In our series, all SCSEHs located posterolaterally, extending along three to eight vertebral levels, and treated by wide laminectomy, hematoma removal, and cord decompression with or without vertebral stabilization. One case was confirmed pathologically as hematoma due to bleeding from a true dural AVM.

Three patients had gradually significant improvement of the preoperative neurological status, but in one case there was no functionally useful improvement.

Spontaneous epidural hematomas of the cervical spine are uncommon neurosurgical entities which, because of their localization is important to promptly diagnosed and treated appropriately, so that this group of patients to be driven in as more favorable operating results.

Although the cause has not exactly been made clear, most authors have reported that is the result of anticoagulant and antiplatelet therapy, vascular malformations, venous epidural plexus defects, and inherited or acquired bleeding disorders.

These hematomas should always be suspected in patients with acute neck pain followed by neurological deficits or myelopathy in the presence of such risk factors. The diagnostic procedure of choice is MRI, and standard treatment is urgent surgical decompression. The two main predictors of outcome are surgery timing and preoperative neurological status

Though differential diagnosis of this clinical entity is extensive, careful neurological examination combined with modern imaging techniques and early intervention allows patients with spontaneous cervical epidural hematoma have a safe diagnosis, significant neurologic improvement, and good outcome.

**Keywords:** Spontaneous epidural hematoma, cervical spine, treatment

#### Introduction

Spontaneous epidural hematoma of the spine is an extremely rare but important cause of compression of the spinal cord or cauda equina, which requires increased suspicion, prompt diagnosis, and appropriate treatment, otherwise it leads to permanent neurological deficit or death. Since Jackson first described this unusual disease in 1869<sup>11</sup>, 500 cases of patients have been published worldwide. Although the etiology of spinal epidural hematomas is not completely understood, most researchers implicate as the most common etiologic factors anticoagulant and antiplatelet agents, vascular malformations, and congenital or acquired coagulation disorders and blood dyscrasias<sup>2,3,9,19,21</sup>. Tsai<sup>27</sup> named them as idiopathic spinal hematomas, when there is no identifiable etiologic factor, and as spontaneous when there is no bone injury after minor trauma or after normal daily activity.

The usually applied therapeutic approach is the earliest possible evacuation of the hematoma and

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decompression of the spinal cord<sup>10,18</sup>. However, some researchers have reported that conservative treatment is also an option in some selected cases<sup>14,</sup> <sup>26</sup>

Spontaneous spinal epidural hematomas can occur at any level of spinal axis , although the most common site is the thoracic area<sup>8,9</sup>. The purpose of this paper is to present our experience in the treatment of spontaneous epidural hematomas of the cervical spine, and review the recent literature.

### Material and method - Results

During the last twelve years, four patients with spontaneous epidural hematoma of the cervical spine (Spontaneous Cervical Spine Epidural Hematoma, SCSEH), were treated surgically in Neurosurgical Clinic of Asklipeio Hospital of Voula.

1st patient: A 80 years old woman with a history of rheumatoid arthritis and hypertension since 20 years receiving anticoagulation therapy (coumarin) due to atrial fibrillation, presented with sudden neck pain and standing – walking weakness since 12 hours. On neurological examination we found a normal level of consciousness with flaccid tetraparesis mainly right, without sensory or sphincter disorders. Magnetic Resonance Imaging (MRI) revealed a space - occupying lesion of the cervical spine from C5 to C7 with characteristics of an epidural hematoma (Fig. 1, 2). She immediately discontinued anticoagulation therapy, received vitamin K, and after improvement of prothrombin time was subjected to C5, C6 laminectomy and total removal of the hematoma. The postoperative course was excellent with gradual neurological improvement until almost complete recovery.

2nd patient: A 74 - year - old woman with a history of coronary heart disease and arterial hypertension receiving antiplatelet therapy was hospitalized elsewhere because of sudden neck pain, headache, stiff neck, and right hemiparesis with a good level of consciousness. By the suspicion of a subarachnoid hemorrhage she was subjected to computed tomography of the brain and lumbar puncture that were normal. A digital subtraction angiography of the brain was also negative for a cerebral aneurysm or other vascular

malformation. After 48 hours the patient showed dramatic neurological deterioration with flaccid quadriplegia and respiratory failure. She was taken under mechanical ventilation in the ICU and the next day underwent cervical spine MRI, which revealed an extensive epidural hematoma of cervicothoracic spine from C3 to T3 (Fig. 3, 4). She was transferred to our Neurosurgical Clinic and underwent an extensive laminectomy C4 - T2, total evacuation of the hematoma, and posterior lateral masses fusion of C4 up to C7 (Fig. 5, 6). The postoperative course was accompanied by a slight improvement in muscle strength without significant functional equivalent, the patient remained with a permanent tracheostomy, and transferred to the Physical Medicine and Rehabilitation Clinic.

**3rd patient:** A 49 years old male without medical history had a sudden neck pain with radiation to the back and shoulders since 30 hours and progressively worsening left hemiparesis with sphincter disorders. He underwent urgent MRI which revealed a fresh epidural hematoma expanding from C3 to C5 vertebra (Fig. 7). He was operated urgently and underwent laminectomy of C3, C4, and C5. With the removal of the hematoma and washing of blood clots, we found the cause of the hematoma, a small arteriovenous malformation of the dura mater at the C4 level (dural arteriovenous fistula), which also treated by thermocoagulation with bipolar diathermy The postoperative course was excellent with full recovery of the initial neurological deficits.

4th patient: A young man aged 21 years without medical history or preceding injury was admitted because of intermittent headache and neck pain since twelve hours. Cranial computed tomography scan was normal. During hospitalization he developed a serious bilateral upper limb paresis, and urinary retention. MRI of the cervical spine showed an extensive epidural hematoma from C4 to T2 (Fig. 8, 9). He underwent a C4 to T1 laminectomy, removal of the hematoma, and posterior cervicothoracic spinal fusion. The postoperative period was without complications, and after an intensive physiotherapy programme his muscle strength returned almost full (4/5).

#### **Discussion - Conclusion**

SCSEH is a rare neurosurgical entity. Due to its critical localization is very important to be diagnosed early and treated adequately with the promising prospect of permanent and non – reversible cervical myelopathy. These hematomas usually affect patients between 50 - 80 years (mean age of our group: 56 years), with a relative superiority of men over women (1.4 - 2:1)<sup>9</sup>.

The exact cause of these hematomas remains unknown in the majority of patients and is probably multifactorial. The most common risk factor is the congenital or acquired hematological disorders, coagulation disorders, intravenous or oral anticoagulants, antiplatelet agents or subcutaneous administration of high doses of low molecular weighted heparin (used most widely, prophylactically or therapeutically, in everyday present medical practice), hypertension, diabetes mellitus, pregnancy, preeclampsia, childbirth, thrombolysis, using of cocaine, vasculitis, non - Hodgkins lymphoma, spinal hemangioma, and epidural vascular malformations<sup>3</sup>, 4,9,13,19,20,23. Minor injuries of the cervical spine in patients with ankylosing spondylitis have also been implicated as a predisposing factor<sup>22</sup>. In 40% of cases the cause of SCSEH remains unknown<sup>6</sup>. In our small series, two patients had a predisposing factor such as hypertension and the use of anticoagulants and antiplatelet agents.

Beatty and Winston<sup>2</sup> believe that the combination of non-steroidal anti-inflammatory drugs (NSAID), anticoagulants, and chronic alcohol intake is very dangerous for development of a spontaneous epidural or subdural hematoma of the cervical spine from the rupture of small epidural arteries. Spontaneous spinal epidural hematoma is more often than subdural, because of plenty of blood vessels in the epidural versus subdural space. Gundry and Heithoff<sup>12</sup> suggest that an acute prolapsus of intervertebral disc can tear the fragile venous plexus of Batson and this leads to epidural hemorrhage. Variations in intrathoracic or intra-abdominal pressure after intense physical exertion, seems to be one of the possible mechanisms of rupture of a vessel of epidural venous plexus, whose veins lack valves and have thin wall. This explains why the great majority of these hematomas are located on the dorsal (posterior) surface of the dura mater, which has a complex well developed venous plexus<sup>4</sup>. SCSEHs usually extend along two or three vertebral levels<sup>7,25</sup>. The usual presence of SCSEH at the dorsal epidural space facilitates the surgical access. In our series hematoma always involved the posterior spinal canal and thus typically accessed by a posterior approach. The greater length of the SCSEH covered eight vertebral levels, and the lower three levels.

In contrast to older studies that epidural venous plexus is responsible for causing SCSEHs, most recent data suggest that bleeding has an arterial origin<sup>4</sup>. In general, the pressure within the cervical epidural veins is lower than the intradural veins, thus a venous bleeding cannot strongly compress the cervical spinal cord. Moreover, in some cases an arteriovenous malformation or angioma<sup>9,21</sup> have been implicated as the cause of the epidural hematoma. We encountered one case of dural arteriovenous communication that was dealt simultaneously with the removal of the hematoma.

Neurological findings usually include sudden intense neck pain with a possible extension to the back, and rapid radiation to the upper extremities. Gradually, within minutes, hours, or days, a cervical spinal cord pressure appears with various degrees of neurological deficits and myelopathy below the level of injury (transverse medullary sensorimotor quadraparesis or semi-transverse medullary syndromes Brown - Sequard with hemiparesis)<sup>1,4,10,24</sup>.

Differential diagnosis is not always easy, requires a high degree of suspicion, and involves a wide spectrum of diseases. In cases of an acute paresis differential diagnostic includes acute prolapse of a cervical intervertebral disc, spinal cord ischemia (anterior spinal artery syndrome), extradural or intradural tumors, epidural abscess, hematomyelia, and polyradiculoneuropathy or transverse myelitis<sup>1,20</sup>.

In past decades, diagnosis could be made only with Computed Tomography in combination with myelography1. Nowadays, the gold standard in the diagnostic approach of SCSEH is MRI. It clearly highlights the hematoma and its exact location, the size and age of the hematoma, the compression and swelling of the spinal cord, and the post-operative visualization of the region. Hematoma is illustrated with equal to the spinal cord signal intensity in T1 sequences within the first 24 hours (oxyhaemoglobin and deoxyhaemoglobin), and as high signal on both T1 and T2 sequences after 36 hours (extracellular methaemoglobin). Thus, the display of a superacute SCSEH may resemble that of an epidural abscess or tumor. The contrast enhancement with paramagnetic substance is extremely rare. Generally, in cases of uptake of the contrast a strong suspicion of epidural tumor or infection should be raised<sup>5,7</sup>. Selective spinal angiography is indicated on suspicion of a vascular malformation.

Preoperative neurological status is perhaps the most important factor in relation to the degree of neurological recovery<sup>6,7,17</sup>. Patients with incomplete sensorimotor deficit have a better neurological outcome, and this is in agreement with our series. In contrast, patients with complete sensorimotor paralysis and sphincter disorders have the worst prognosis. Another important predictor is the interval from the onset of symptoms until operation. According to the literature the surgical hematoma evacuation within 8 - 36 hours of onset of symptoms increases significantly the chances of complete recovery 15,17,18. In our series, the second patient operated too late with fully installed spinal cord compression, and unfavorable eventually had an outcome. Furthermore, in experimental models of spinal cord

injury in animals, it was confirmed that as greater the time length of spinal cord pressure then more severe pathological changes are identified in the nerve fibers, in the vascular endothelium, and microcirculation. Other factors that seem to affect prognosis are age, the extent and location of the hematoma, and the speed of neurological deterioration<sup>20</sup>.

Preoperatively, it is necessary to discontinue the anticoagulant and antiplatelet therapy, administer fresh frozen plasma or vitamin K, as well as high doses of prednisolone.

Some authors report that in some cases conservative management of SCSEH could be just as effective as surgical evacuation of the hematoma, provided the ability of a close neurological and MRI follow up<sup>14,16, 26</sup>. These authors suggest non — operative treatment in selected patients with clotting disorders in which the hematoma has not been organized in solid clot, but the epidural blood remains in a liquid state. In these patients, usually with hemophilia, the neurological picture was usually mild and radiographic findings improved enough in the next hours<sup>5,14,16,26</sup>. All our patients were in a severe condition with progressive neurological deterioration due to the effects of spinal cord compression, and were all treated surgically.

Intraoperatively, we found that epidural tissue was immersed in both hard blood clots and fresh blood collection. All removed material was sent for full histopathological examination to be examined for any possible cryptic vascular malformation.

Fig 1. Saggital T2 weighted image revealing an epidural hematoma from  $C_5$  to  $C_7$ , slightly hyperintense to cervical spinal cord.



Fig. 3. Sagittal T2 weighted image revealing an epidural hematoma from C<sub>3</sub> to Th<sub>3</sub>. The hematoma is hyperintense to spinal cord with a signal of serious cervical intramedullary ischemia.



Fig. 2. Axial T2 weighted image shows an epidural hematoma on the right posterior epidural space with significant obliteration of epidural fat and cord compression.



Fig. 4. Axial T1 weighted image revealing an epidural hematoma on the right posterior epidural space.

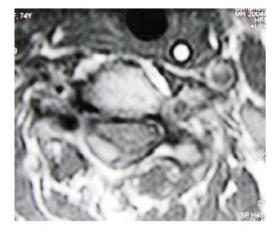


Fig. 5. Intraoperative view of a lateral masses screw fixation from  $C_4$  to  $C_7$ . At the level of  $C_6$  laminectomy the hematoma is appeared at the dorsal spinal canal.

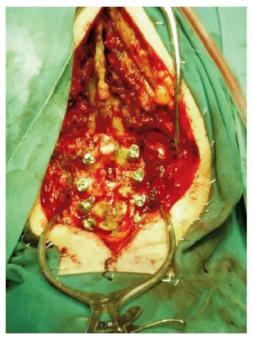


Fig. 7. Sagittal T2 weighted image shows an epidural hematoma from  $C_3$  to  $C_5$ . The 30 hrs hematoma is hyperintense to spinal cord.



Fig. 6. Intraoperative view of a wide C<sub>4</sub> to Th<sub>2</sub> laminectomy, progressive removal of epidural hematoma, and reappearance of epidural fat.

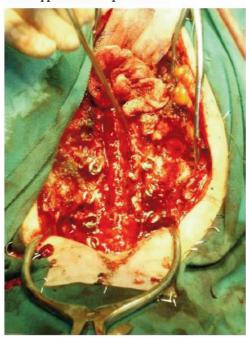
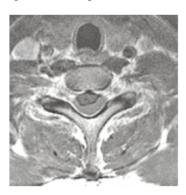


Fig. 8. Sagittal T1 weighted MRI shows large epidural hematoma from C<sub>4</sub> to Th<sub>2</sub> causing severe spinal cord compression. The hematoma is isointense in signal to spinal cord.



Fig. 9. Axial T2 weighted MRI shows an epidural hematoma extended bilaterally in posterior aspect of canal with obliteration of epidural fat and significant spinal cord compression.



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