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Clinical Study

Spontaneous spinal epidural hematoma: The importance of preoperative neurological status and rapid intervention

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ABSTRACT

We describe the presentation, management, and outcome of spontaneous spinal epidural hematoma (SSEH) in two tertiary academic centers. We retrospectively reviewed clinical and imaging files in patients diagnosed with SSEH from 2002–2011. Neurologic status was assessed using the American Spinal Injury Association (ASIA) Impairment Scale (AIS). A total of 17 patients (10 females; mean age 54 years, range 10–89) were included. Among patients presenting with AIS A, 5/8 showed no improvement and 3/8 reached AIS C. Among those presenting with AIS C, 5/6 reached AIS E and 1/6 reached AIS D. Of those presenting with AIS D, 3/3 reached AIS E. Mean time-to-surgery (TTS) was 28 hours (range 3–96). TTS surgery in two patients remaining at AIS A was ⩽12 hours; in 4/8 patients recovering to AIS E it was >12 hours, including three patients operated on after >24 hours. In patients remaining at AIS A, a mean of 4.4 levels were treated compared with means of 3.7 and 3.5 in those with AIS C and E, respectively, at late follow-up. In this series, preoperative neurological status had greater impact on late outcome than time from symptom onset to surgery in patients with SSEH.

1. Introduction

Spontaneous spinal epidural hematoma (SSEH), defined as blood occupying the spinal epidural space and causing compression of the neural structures, is an uncommon entity, comprising less than 1% of all spinal space-occupying lesions. SSEH constitutes a neurosurgical emergency, and often leads to devastating sequelae unless it is diagnosed early and managed effectively.

A description by Jackson in 1869 [1] is credited as the first official record of a spinal epidural hematoma. Since that time, there have been numerous reports of SSEH presenting in all age groups, with higher frequency in the fourth and fifth decades of life. The estimated annual incidence of SSEH is 0.1 per 100,000 [2]. There is increasing awareness of the condition, especially in emergency departments. Suspicion of SSEH usually triggers an emergency neurosurgical consultation.

Approximately 40–60% of spinal epidural hematomas are idiopathic [3,4]. SSEH is associated with hypertension [5,6], coagulopathy and anticoagulation or anti-aggregation therapy [7–10], chiropractic spinal manipulation [11,12], and pregnancy and labor [13–19]. In addition, insignificant straining or effort, such as bending, dancing, swimming, sneezing, coughing, vomiting, micturition, or heavy lifting may play a role in the development of SSEH [20,21].

The characteristic presentation of SSEH is sudden or severe back or neck pain with or without radicular radiation that may rapidly progress to paresis and sometimes paralysis [22]. Spontaneous recovery has been reported [23–26] and some authors have advocated conservative management in patients with mild, stable, or improving neurological status [23,26–28]; however, patients with symptomatic SSEH are most commonly managed with emergent surgical decompression and evacuation of the hematoma [6,22,29].

We reviewed our experience in the management of SSEH in two academic tertiary medical centers, with the aim of describing patient presentation, diagnostic challenges, potential risk factors, management, and outcomes.

2. Methods and materials

We retrospectively reviewed the digital patient record systems at the Sheba Medical Center in greater Tel Aviv, and the Hadassah-Hebrew University Medical Center in Jerusalem, Israel,

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to identify all patients treated for SSEH from 2002–2011. We included patients with spinal epidural hematoma that was apparently spontaneous in nature; patients with hematoma due to traumatic injury (including any minor trauma or chiropractic manipulation) or secondary to surgery or after invasive procedures, such as epidural analgesia or lumbar puncture were excluded from the study. Institutional Review Boards for the two centers approved the study, and waived the requirement for informed consent.

Medical records, including symptoms at presentation, findings at physical and neurological examination, and considerations regarding the differential diagnosis were noted. Patient history and risk factors, including any recent activity involving a straining effort, hypertension, coagulopathy, anticoagulation or anti-aggregation, or recent pregnancy, as well as significant comorbidities, were noted in the study data. Details of surgical treatment were also included in the research file. Presentation on admission CT scan as well as MRI were recorded. The spinal level and extent of the epidural hematoma were noted. Operative reports and histopathology were reviewed for all patients. The intervals from symptom onset to hospital admission and symptom onset to surgical decompression were recorded.

Neurological status at admission, discharge, and long-term follow-up was classified based on the American Spinal Injury Association (ASIA) Impairment Scale (AIS), a five-part classification system for localization and assessment of the degree of motor impairment in individuals with spinal cord injury (Table 1) [30].

2.1. Patient management

Patients presenting with back pain or signs of cord compression underwent a thorough neurological examination for diagnosis and assessment of the urgency for intervention. The first imaging study performed for these patients in our centers was CT scan, due to its immediate availability and low cost. MRI was performed in cases where the diagnosis could not be achieved with CT scan. During the study period, once a diagnosis was achieved, coagulopathy was corrected as needed with administration of Vitamin K and fresh frozen plasma to achieve an international normalized ratio <1.5. Surgery was performed urgently in patients suffering a neurological deficit, especially those with progression of deficit. CT angiography of the head and spinal column were performed preoperatively if time allowed, or postoperatively, to rule out vascular causes of spinal epidural hematoma such as vascular malformation. Diagnostic angiography was performed in cases when CT angiography and/or magnetic resonance angiography was not definitive.

All patients in this series suffered SSEH dorsal to the spinal cord, and were operated on using a posterior approach through laminectomy. In cases of SSEH located ventral to the cord, other surgical approaches may be utilized.

3. Results

A total of 17 patients, including seven males (41%) and 10 females (59%), mean age 54 years (range 10–89 years), met inclusion criteria (Table 2). Two patients (ages 76 and 77 years) had a history of hypertension, two had diabetes mellitus, two had ischemic heart disease, one had hyperlipidemia, and one had had a pulmonary embolism 3 weeks earlier. Four patients were on aspirin and three were on warfarin.

One patient reported having strenuously lifted a friend the previous evening, one had lifted weights a week earlier, and one had symptom onset after cervical neck flexion and extension. One patient described a sudden pain radiating to the abdomen that began after he bent over.

All patients presented with sudden onset neck or back pain, followed by onset of progressive neurological deficit. Eight patients presented with AIS grade A (47%), six were grade C (35%), and three were grade D (18%).

In 14 patients (82%), CT scan was inconclusive and the diagnosis was made with MRI. In the two pediatric patients included in our series (Patients 7 and 16) and one 25-year-old who had been weight-lifting a week before pain onset (Patient 15), CT scan was sufficient to achieve the diagnosis, and surgery was performed based on CT scan findings alone. SSEH was seen in the cervical spine in four patients (23%), cervicothoracic in one (6%), thoracic spine in 10 (59%), and thoracolumbar spine in two (12%), with extension for a mean 3.9 levels (range 1–6).

Surgery was performed a mean of 28 hours after symptom onset (range 2.5–96). In four patients (24%) the operation was performed within 12 hours of symptom onset, in four (24%) it was within 13–24 hours, and in six patients (35%) surgery was performed after 24 hours or longer. One patient (Patient 15) had back pain for a full week before onset of progressive neurological deterioration and surgery was performed 5 hours later (5 hours for purposes of analysis). Time from symptom onset to surgery was unavailable for two patients.

Laminectomy of the involved levels was performed in all patients. Spine fusion was performed in two cases (Patient 14 and 17) where the laminectomy included levels above and below the apex of the thoracic kyphosis. There were no perioperative mortality or major complications, and no recurrent hemorrhage after surgery.

A comparison of preoperative neurological status with AIS at late follow-up is shown in Figure 1. At long-term follow-up, the functional status of 5/8 patients presenting with AIS A failed to improve. Of these five, two (Patient 6 and 10) were on aspirin (one with diabetes, hypertension, and ischemic heart disease) and one (Patient 8) was on warfarin following a recent pulmonary embolism. Functional status improved to AIS C in 3/8 patients presenting at AIS A, including one patient on aspirin after percutaneous transluminal coronary angioplasty.

Table 1
American Spinal Injury Association (ASIA) Impairment Scale (AIS) for grading degree of impairment in patients with spinal cord injuries [30]

E	**Normal** – If sensation and motor function as tested in accordance with the International Standards for Neurological Classification of Spinal Cord Injury are graded as normal in all segments, and the patient had prior deficits, then the AIS score is E. An individual without a spinal cord injury does not receive an AIS grade.
D	**Motor incomplete** – Motor function is preserved below the neurological level*, and at least half (half or more) of key muscle functions below the neurological level of injury have a muscle grade >3.
C	**Motor incomplete** – Motor function is preserved below the neurological level*, and more than half of key muscle functions below the single neurological level of injury have a muscle grade <3 (grades 0–2).
B	**Sensory incomplete** – Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4–S5, AND no motor function is preserved more than three levels below the motor level on either side of the body.
A	**Complete** – No sensory or motor function is preserved in the sacral segments.

* For an individual to receive a grade of C or D, i.e. motor incomplete status, they must have either (1) voluntary anal sphincter contraction or (2) sacral sensory sparing (as S4/5 or deep anal pressure) with sparing of motor function more than three levels below the motor level for that side of the body. The Standards at this time allow even non-key muscle function more than three levels below the motor level to be used in determining motor incomplete status (AIS B *versus* AIS C).