



Spontaneous Spinal Epidural Hematoma: Correlation of Timing of Surgical Decompression and MRI Findings with Functional Neurological Outcome

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OBJECTIVE: Spontaneous spinal epidural hematoma (SSEH) is a rare and morbid entity, with the prognosis affected by delayed diagnosis and surgical intervention. The correlation between the timing of the intervention and neurological recovery has not been clearly reported. We present a retrospective study of SSEH to determine the correlation between the timing of surgical intervention and changes in the spinal cord signal on magnetic resonance imaging (MRI) with the neurological outcome.

METHODS: The records of 14 patients who had undergone surgical decompression of SSEHs during a 10-year duration were reviewed. The diagnosis was established from the MRI, intraoperative, and histopathological examination findings.

RESULTS: We identified 14 patients from both centers, 6 of whom were male. Their mean age was 54.1 years. The onset was spontaneous, and 2 patients were receiving anticoagulant therapy. The most common presentation was paraplegia ($n = 8$), followed by paraparesis ($n = 3$) and quadriparesis ($n = 2$). Spinal cord signal changes were demonstrated on all T2-weighted MRI studies. The response to surgery was favorable for 13 of our patients at the 6-month follow-up examination. The spinal cord changes had persisted in 5 patients on the 6-month post-operative MRI scan. All 14 patients, except for 1, had a favorable neurological outcome at the last follow-up examination.

CONCLUSIONS: Significant neurological recovery after surgical decompression of SSEHs can be achieved, despite

the significant preoperative neurological deficits, spinal cord changes on MRI, and delayed timing of intervention.

INTRODUCTION

Spinal epidural hematoma (SEH), first described in 1869 by Jackson,¹ is a rare condition. An underlying etiology can be identified in nearly 70% of cases.^{2,3} The list of possible etiologies include preceding trauma, coagulopathy, arteriovenous malformation, and tumor apoplexy. SEHs can also be iatrogenic, with reports of it occurring after lumbar punctures, epidural catheters, and spinal surgeries.³ Less frequently, no underlying organic pathology will be identified, and the hematoma will be attributed to a spontaneous rupture of the epidural vasculature.^{3,5} The annual incidence of spontaneous SEH (SSEH) has an estimated incidence of <0.1 in 100,000 of the general population. SSEH has been associated with serious morbidity, in part owing to its late diagnosis.^{3,4}

Just as occurs with other causes of spinal cord compression, the duration of compression is a valuable factor in determining the postoperative outcomes.⁶ One area that has been poorly described in the management of such a rare entity is the outcome to be expected with varying durations of compression. Several reports have advocated decompression within variable time windows, ranging from within 12 hours to ≤ 2 days as the maximum delay acceptable for satisfactory outcomes.⁶⁻⁸

We report the cases of 14 patients presenting with a rapidly progressive neurological deficit after SSEH who had undergone surgical decompression. We also attempted to correlate the overall outcome to the timing of the intervention and spinal cord changes demonstrated on the MRI scan at presentation.

Key words

- MRI
- Outcome
- Spontaneous spinal epidural hematoma
- Surgical decompression

Abbreviations and Acronyms

AIS: American Spinal Injury Association impairment scale

IVVP: Internal vertebral venous plexus

MRI: Magnetic resonance imaging

SHE: Spinal epidural hematoma

SSEH: Spontaneous spinal epidural hematoma

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METHODS

Design

We performed a retrospective longitudinal cohort study.

Setting

The medical records database was retrospectively reviewed for the diagnosis of SSEH from July 2004 to December 2014 at King Abdulaziz University Hospital (Jeddah, Saudi Arabia) and Montreal General Hospital (Montreal, Quebec, Canada).

Patients

A total of 14 patients were identified, 7 from each center. Their medical records were retrieved for review after both hospital's institutional ethics review board had approved the present study. The data collected for these patients concerned their demographic data, clinical presentation, coagulation parameters, radiological characteristics, and operative intervention. The MRI findings supported the diagnosis in all SSEH cases and was confirmed by the operative findings. The hematoma signal characteristics, size, and location were recorded, in addition to the presence of the spinal cord signal changes on T2-weighted MRI scans. An underlying cause was sought in all cases. Specific attention was given to document the presence of trauma, spinal fractures, previous spinal interventions, vascular malformations, and history of malignancy. Exclusion of these epidural hematomas was determined by examination of clinical and radiological records and histopathological examinations for any suspicious clots or tissue found during surgery. The documented absence of all these causes would label the hematoma as an idiopathic SSEH.

Outcome Measures

The medical records were reviewed, and the neurological findings were graded according to the American Spinal Injury Association impairment scale (AIS), reported in 1997 by Maynard et al.⁹ A follow-up MRI scan was acquired for all patients at ~6 months postoperatively to document spinal cord decompression and any persistent structural damage; this was possible in all cases. We defined a "favorable response" to surgery as either >2 grades of improvement on the AIS or a score of E at the first 6-month and last follow-up examinations.

Statistical Analysis

The demographic data and baseline characteristics of the study participants were summarized using the mean \pm standard deviation or proportions, as appropriate. One patient was found to be an outlier, based on their interval to surgery of 10 days. We performed analyses with and without the data from this patient.

The association between the interval to surgery and change in outcome was evaluated using scatterplots and linear regression models with a change in AIS score as the response and adjusted for various covariates. Separate models were considered for a change in AIS score at 6 months and after 1 year, also with and without the outlier. All statistical analyses were performed using R, version 3.3.1, and RStudio, version 1.0.136 (R Foundation, Vienna, Austria).

Spearman's correlation coefficient was used to assess whether the patient and hematoma characteristics affected the extent of improvement on the AIS and the speed of recovery, our 2 operative

outcome measures. The patient characteristics evaluated were age, extent of the deficit on presentation, and the interval from deficit to surgery. The hematoma characteristics assessed were its extent (measured in vertebral body length), degree of T2-weighted signal change on the preoperative MRI scan, and whether the SSEH was idiopathic. We also correlated the magnitude of the signal change on the postoperative MRI scan acquired at 3 months postoperatively. The nonparametric Mann-Whitney U test was used to test the association between these 2 outcome measures and sex. P values <0.05 were considered to indicate statistical significance.

RESULTS

Demographic Data

Our series (Table 1) included 6 men and 8 women (female/male ratio, 1.3:1), with a mean age of 54.1 years. Of the 14 patients, 12 had previously been healthy and 2 had been receiving anticoagulation therapy and had presented with an elevated international normalized ratio >5.

The presenting symptoms for all our patients were sharp interscapular pain and neck pain that preceded the sudden onset of a rapidly progressing neurological deficit. The most common neurological deficit in our series was paraplegia ($n = 8$), followed by paraparesis ($n = 3$) and quadriparesis ($n = 3$).

Radiological Findings

The SSEH was located in the cervical region in 3 patients, the cervicothoracic region in 6 patients, the thoracic region in

Table 1. Summary of Demographic and Medical Characteristics

Covariate	Mean \pm SD or n (%)
Country	
Saudi Arabia	7 (50)
Canada	7 (50)
Age (years)	54.1 \pm 22.8
Male sex	6 (42.9)
Cause	
Idiopathic	12 (85.7)
Warfarin use	2 (14.3)
Time to surgery (hours)	21.8 \pm 10.2
Preoperative AIS score	
A	5 (35.7)
B	5 (35.7)
C	3 (21.4)
D	1 (7.1)
Change in AIS level	
At 6 months postoperatively	2.3 \pm 1.2
At >1 years postoperatively	2.5 (1.1)
AIS, American Spinal Injury Association impairment scale.	

3 patients, and the thoracolumbar region in 2 patients. The hematoma extent spanned a mean 4.29 vertebral levels (range, 2–9) and was located exclusively posterior to the spinal cord in 13 patients; the remaining patient had a situated posteriorly thoracic SSEH with an additional anterior extension to the lumbar epidural space.

The hematomas had similar MRI characteristics, including isointensity on T1-weighted images and hyperintensity on T2-weighted images. No lesion or hematoma enhancement was manifested after intravenous gadolinium administration (Figure 1).

Spinal cord signal changes on T2-weighted images were noted in all 14 cases, spanning an average 2.14 vertebral levels (range, 1–4). The length of signal changes was increased among those patients who had presented with a complete neurological deficit (AIS score, A; mean vertebral levels, 2.5) compared with those showing only partial deficits (AIS score, B and C; mean vertebral levels, 1.6).

Surgical Intervention

Multilevel decompressive laminectomy and hematoma evacuation were performed in all patients. None of the patients experienced any complications and had an uneventful postoperative period. Only 1 patient required a single-level laminectomy for decompression, with the remaining 13 requiring multilevel procedures. One patient required decompression of 8 levels owing to the presence of >1 site of significant compression of the spinal cord and cauda equina by distinct collections. Six patients required 4-level laminectomies and 6 only needed 2-level laminectomies. The interval to surgery was longer in our series mainly owing to a delayed presentation by patients who erroneously believed their symptoms to probably be transient.

Further delay was required for 2 patients to correct their coagulopathy because the activated prothrombin complexes were unavailable (patient 2 developed a transfusion reaction to fresh-frozen plasma). The mean interval from presentation of the

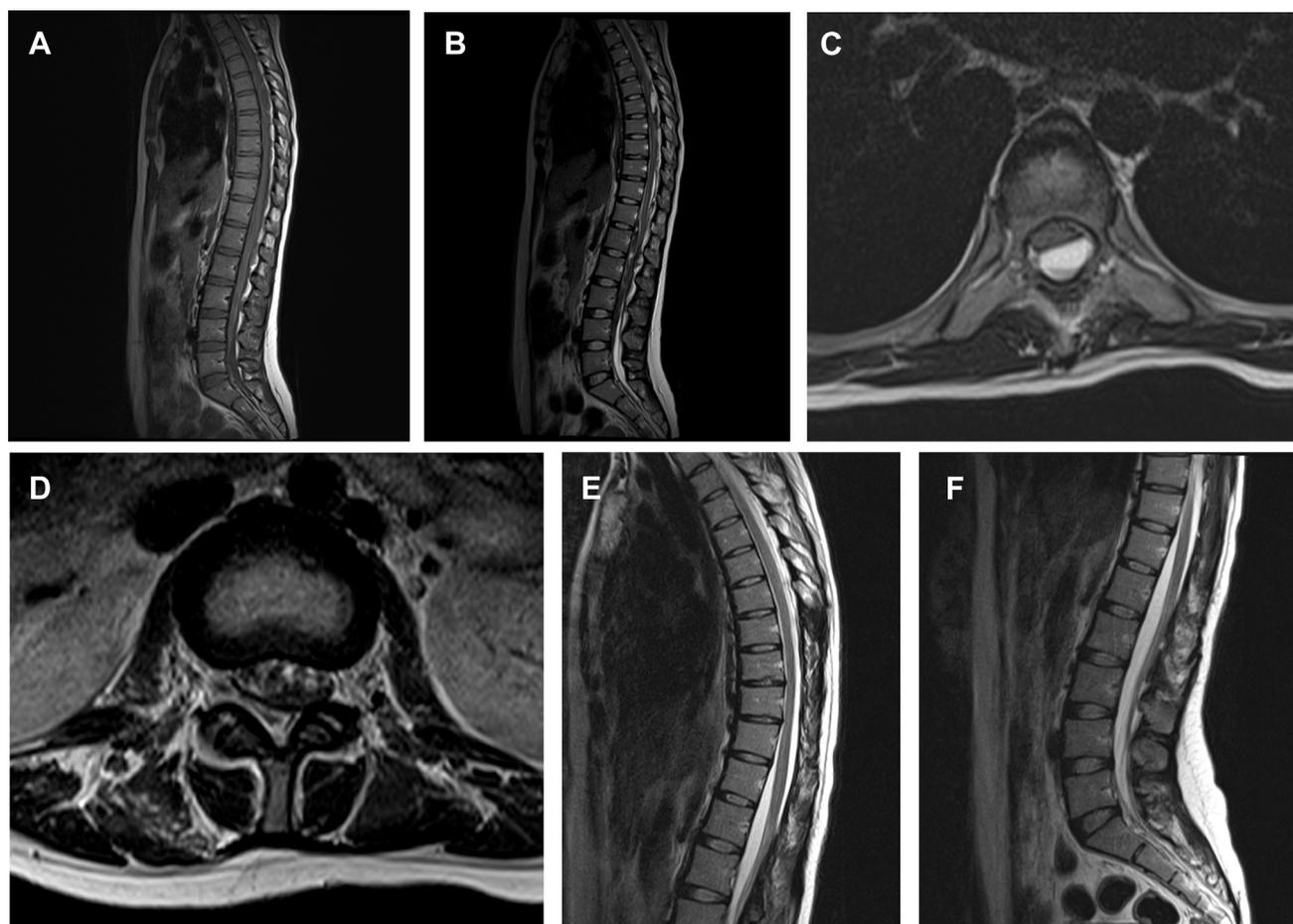


Figure 1. Case illustration of a spontaneous spinal epidural hematoma (SSEH) in a healthy 32-year-old woman who had presented with paraplegia and sphincter disturbance. (A–D) Magnetic resonance imaging (MRI) of the spine revealed an extensive posterior thoracic and anterior lumbar epidural hematoma causing significant spinal cord and cauda equina compression with high T2-weighted signal changes in the spinal cord. The patient underwent thoracic lumbar laminectomies and evacuation of the SSEH with a good clinical outcome. (E, F) Her follow-up MRI scan revealed complete resolution of the SSEH and no spinal cord abnormalities.

deficit to surgery was 21.29 ± 10.2 hours (range, 10–48), except for an outlier case, which required 10 days to be prepared for surgery. The histopathologic findings from any suspicious masses were negative for any structural lesion and typical for the findings of a hematoma.

Outcomes and Follow-Up

All 14 patients improved after surgery during the long-term follow-up period (Table 2). The response to surgical intervention was favorable in all 14 patients (AIS score E in 9 and AIS score D in 4), except for the patient whose case was an outlier. The AIS score for that patient had not improved even at 1 year postoperatively. No patient presented with hematoma recurrence. The mean improvement at the 6-month follow-up examination of the patients' AIS was 2.3 degrees, despite the poor functional status on presentation (AIS score, A or B for 10 of 14 patients) and decompression performed beyond the optimal 12-hour limit. The time course to reach maximal improvement in neurological status was somewhat prolonged in the patients from Saudi Arabia. In that group, we attempted to correlate the patient and hematoma characteristics with the extent of improvement and the speed of recovery using the 1-tailed Spearman correlation coefficient. However, we failed to find any statistically significant correlation, with 1 exception. The length of the postoperative signal changes correlated negatively with extent of maximal improvement (correlation coefficient, -0.746 ; $P = 0.044$).

A follow-up MRI scan of the decompressed region was obtained for all 14 patients. All studies documented improvement in the initially noted spinal cord signal changes. The average change among those with both preoperative and postoperative MRI scans available was a decrease of 1.5 vertebral levels. The average extent of residual spinal cord signal changes among those experiencing a complete recovery was 0.25 vertebral level compared with those with some residual disability. Two patients had signal changes spanning 1 level at the first 6-month follow-up examination. Of the 2 patients receiving preoperative anticoagulation, 1 had to resume anticoagulation therapy 6 weeks postoperatively because of protein S deficiency.

As demonstrated in the scatterplots of the time-to-surgery versus the change in AIS score, we found a weak negative relationship between the time-to-surgery and change in AIS score at 6 months, without including the outlier (Figure 2). For the change in the AIS score after 1 year, we only found a weak positive correlation. When including the outlier, the relationship between our variables appeared moderately negative (Figure 3). Because this single data point influenced our analysis to such a degree, we removed this outlier from analysis.

In 3 of 4 of our linear regression models, the interval to surgery did not have a significant effect on the change in AIS score, after adjusting for age, sex, and country. However, in the model with the outlier present, the interval to surgery was a statistically significant predictor of the change in the AIS score after 1 year, after adjusting for the other variables (Table 2). The effect size was minuscule, possibly owing to the significant follow-up period for our observations when the outlier is included. This coefficient can be interpreted as follows. For each additional hour before surgery, we can expect, on average, a decrease of -0.009 in the AIS score 1 year postoperatively.

Table 2. Change in American Spinal Injury Association Impairment Scale Scores

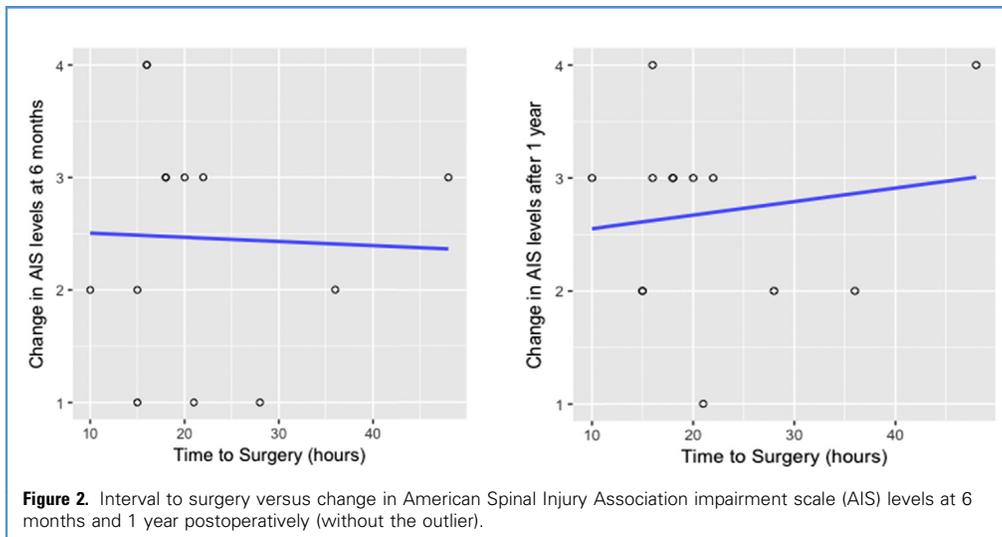
Evaluation Point	Predictor	Estimate	SE	P Value
6 Months	Without outlier			
	Intercept	6.89	2.84	0.04
	Time to surgery (hours)	-0.045	0.04	0.28
	Age	-0.05	0.03	0.12
	Male sex	-0.62	0.71	0.41
	Saudi Arabia	-1.04	1.24	0.43
1 Year	Without outlier			
	Intercept	5.49	2.57	0.07
	Time to surgery (hours)	-0.015	0.04	0.69
	Age	-0.04	0.03	0.19
	Male sex	-0.13	0.64	0.84
	Saudi Arabia	-0.64	1.13	0.58
6 Months	With outlier			
	Intercept	4.87	1.85	0.03
	Time to surgery (hours)	-0.008	0.04	0.12
	Age	-0.04	0.02	0.18
	Male sex	-0.20	0.55	0.72
	Saudi Arabia	-0.50	1.10	0.66
1 Year	With outlier			
	Intercept	5.23	1.60	0.010
	Time to surgery (hours)	-0.009	0.004	0.045
	Age	-0.04	0.02	0.12
	Male sex	-0.08	0.47	0.87
	Saudi Arabia	-0.57	0.94	0.56

SE, standard error.

DISCUSSION

Patients with SSEH will present with severe localized spinal pain, which could have a radicular component.^{10,11} This pain will occasionally be related to minor straining caused by daily activities such as lifting, coughing, defecation, or sneezing. The signs and deficits often progress rapidly to result in paraparesis or quadriplegia eventually.¹¹⁻¹⁷

The pathophysiology of SSEH remains poorly described, and the exact nature and bleeding source of this spontaneous vascular disruption is not known. Clinical observations and anatomical studies have pointed to the fragile valve-less internal vertebral

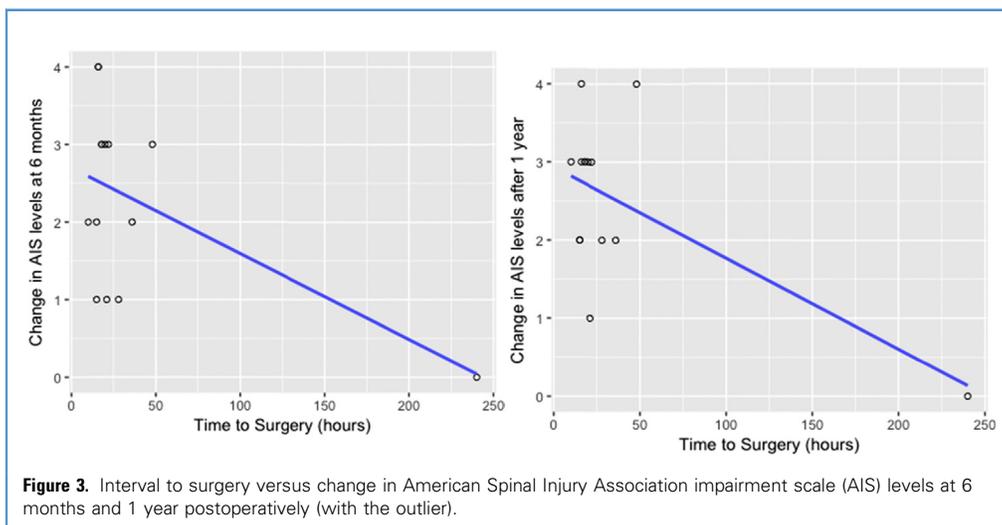


venous plexus (IVVP) as perhaps being the source of bleeding.¹⁸ The IVVP is mainly dorsal to the spinal cord and is sensitive to pressure fluctuations in the abdomen and thorax. This might account for the cases in which hematomas were reported to occur after strenuous activity and Valsalva maneuvers.⁵ Arguments have been made that a possible role for arterial sources must be highlighted, especially considering that the IVVP pressure is less than that of the thecal compartment⁵; however, these have not been widely adopted in the reported studies.

Most reported cases have been in the cervicothoracic or thoracolumbar spinal regions, consistent with our series. A wide range of symptoms and manifestations have been documented according to the location and specific compressive pattern, ranging from minor radiculopathies to complete spinal cord injury.^{8,19-21} The characteristic presenting feature, however, which should raise

clinical suspicion is a sudden onset of unprovoked neck and/or back pain preceding the neurological deficit.^{3,11} Our series showed that the diagnosis might have been discovered at an earlier stage in 1 of our 2 patients receiving anticoagulation therapy had the index of suspicion been greater. That patient had presented to the emergency department with back pain 2 days earlier but had been sent home with only analgesics and no coagulation profile had been ordered.

The spinal MRI scan is the diagnostic modality of choice and will help rule out the differential diagnoses of acute paraplegia. This modality is useful to confirm the diagnosis and can provide information on any underlying etiology and document the hematoma extent, in addition to the spinal cord signal changes, which could influence the prognosis.⁶ Spinal angiography has been routinely performed in some centers when an epidural hematoma is found. Others have reserved spinal angiography for



cases in which the MRI or myelography findings indicate the presence of a vascular malformation.^{10,12,22} Myelography and computed tomography myelography could show an epidural lesion with partial or complete spinal block but these imaging modalities are not specific, are invasive, and could worsen the clinical status. Conventional computed tomography can be used to diagnose an epidural hematoma but can give false-negative results if the hematoma is isodense to the thecal sac or spinal cord and if the image quality is affected by artifacts (often seen in the upper thoracic region).^{1,23,24}

Despite the superiority of MRI, the findings from patient 3 in our series were notable as an excellent example to not rely solely on radiological findings for clinical decision-making. Patient 3 presented with an intact dorsal column on examination despite the dorsally located hematoma. Postoperatively, the patient had maintained her motor deficit with minimal improvement. A repeat MRI scan at 3 months postoperatively revealed spinal cord damage exclusively to the dorsal column despite her exclusively anterior column deficit. We were unable to determine a reason for this puzzling observation and were able to locate only 1 similar case report.²⁵ In that case, however, the MRI scan had shown a corresponding anterior cord signal change and the culprit in the form of a herniated disc. Recently, Endo et al.²² reported the use of apparent diffusion coefficient values, in addition to T2-weighted MRI findings to predict the neurological recovery of 4 patients with SSEH. They concluded that patients with severe neurological deficits with a high T2-weighted MRI signal and low apparent diffusion coefficient values would have limited neurological recovery.²²

Lately, a few investigators have reported significant recovery with conservative therapy for selected patients with a mild to moderate neurological deficit who demonstrated early neurological improvement.¹⁹⁻²¹ Kim et al.²⁶ reported the retrospective comparative results of 15 patients with SSEH who had undergone surgery versus conservative treatment. The surgical group presented with an AIS score of A–D and the conservative group had presented with an AIS score of C–E. They concluded that conservative therapy could be feasible for selected patients with an AIS score of E or patients with an AIS score C or D demonstrating spontaneous early neurological recovery. Although Muñoz González et al.²⁴ reported good recovery, with a score of ≤ 2 using the modified Rankin scale, at the 1-year evaluation of 3 of 4 patients treated with surgery compared with 4 of 8 patients treated conservatively. Raasck et al.²⁷ recently reported a data review of 65 cases from 12 studies. They concluded that conservative management had proved effective, although feasible only if spontaneous recovery had manifested.²⁷ However, decompressive laminectomy should continue to remain readily available, given the inverse correlation between the operative interval and better recovery.²⁷

A large meta-analysis identified the predictors of a good postoperative outcome as rapid intervention, vertebral levels involved (as a surrogate for extent), and incomplete deficit.⁶ One matter of controversy is what constitutes timely intervention, the meta-analysis of SSEH reported the cutoff for complete deficit should be 36 hours and for incomplete deficits should be 48 hours.⁶ However, that might be an unfair generalization. A more in-depth review of what constitutes a timely intervention by Lawton et al.⁷ provided a subanalysis of all patients who had undergone

surgery within 36 hours, dividing them into groups according to the interval to surgery: ≤ 12 , 12–24, and 24–36 hours. Complete recovery was noted in 52%, 25%, and 23% of the ≤ 12 -, 12–24-, and 24–36-hour groups, respectively, confirming the idea that the chance of improvement diminishes with time. The outcomes for the groups treated between 12 and 36 hours were, in fact, similar to those treated after 36 hours.

Our cohort, therefore, did not undergo surgery within the optimal window, because the mean interval to surgery was ~ 21.8 hours. Our findings are notable, however, for the overwhelmingly favorable 6-month outcomes to surgery, with a mean improvement of 2.85 degrees in the AIS score despite the marked delay to intervention (mean interval, 21.8 hours) and poor functional status on presentation (AIS score A or B in 10 of 14).

The only patient who had undergone surgery within the 12-hour limit (patient 1) was the patient with the poorest postoperative outcome (AIS score A–C at the 6-month follow-up evaluation). That patient was relatively old (50 years) and had had a very short interval between the onset of neck pain and the occurrence of the deficit compared with the other 2 patients. MRI scans showed signal changes spanning 4 vertebral levels, despite a small hematoma that spanned only 2 levels. These factors might have underlined the poor outcome of that patient, and the MRI findings of significant cord signal changes were an indicator.

Thus, we would propose a new marker of prognosis to be evaluated in future studies: the length ratio of the MRI cord signal change to the lesion. We believe a greater ratio would reflect poorer outcomes even with lower absolute values, as suggested by our observation of a single patient.

The younger age group of our series might have been responsible for our overall favorable outcomes, in addition to the propensity of idiopathic SSEH, as opposed to structural causes that can bring into the equation bleeding sources other than low-pressure IVVP. This should serve as a source of encouragement to surgeons and patients alike that surgery should be considered worthwhile for these younger patients, even those with complete deficits and a presentation after the 12-hour window.

Urgent surgical decompression is the treatment of choice for SSEH with a neurological deficit or compromise. Some studies have reported cases in which SSEH was treated nonoperatively with good outcomes, mainly hematomas localized to the cauda equina level and with a mild neurological deficit.^{6,15,23,27} The critical factors for recovery after SSEH are the level of preoperative neurological deficit and the operative interval.^{19,22,25} In patients with complete preoperative sensorimotor loss, surgery within 36 hours of the onset of symptoms will provide better outcomes.²²

Study Limitations

The present study had several limitations. First, because of our small sample size, our study was underpowered to detect effect sizes. Second, our study was performed at 2 very different healthcare systems. Access to rehabilitation is extremely limited in Jeddah where the Saudi patients were enrolled. This is the contrast to the Canadian center, with readily accessible inpatient rehabilitation facilities with intensive physiotherapy and access to orthoses provided. Furthermore, although the last follow-up examination after surgery is 1 year in Canada, it is often much greater than 1 year in Saudi Arabia (range, 1–8 years) because

many patients do not have access to primary care where they can be followed up for late deterioration.

CONCLUSIONS

Although an uncommon diagnosis, a high index of suspicion should be maintained by clinicians for this entity in the appropriate clinical scenario. The onset of spontaneous back pain with a sudden or progressing neurological deficit should place SSEH high in the list of differential diagnoses and is an indication for a spinal MRI examination. Our patients had encouraging results after operative intervention for complete neurological deficits arising from spinal cord compression within 24 hours. This holds especially true for young patients, because even a delayed

presentation of a complete deficit can result in an independent or partially dependent patient with a significant gain in their quality of life. MRI studies and expedient access to surgical decompression should be considered when SSEH is suspected. Our results suggest that a nihilistic approach to patients presenting with complete neurological deficits that have persisted for >12 hours is unwarranted and could hinder access to ambulation-restoring surgery. Despite our inability to demonstrate a correlation between the use of early surgery and improved functional outcomes, we believe that resulted from our small number of young patients, which should not outweigh the considerable body of evidence supporting the benefit of early spinal cord decompression.

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