

Prognostic Factors and Treatments Efficacy in Spontaneous Spinal Epidural Hematoma

A Multicenter Retrospective Study

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Abstract

Background and Objectives

Spontaneous spinal epidural hematoma (SSEH) is an uncommon but serious condition with a high morbidity rate. Although SSEH is related to numerous risk factors, its etiology remains unclear. There is a paucity of data on its prognostic factors. We aim to evaluate prognostic factors for SSEH in this study.

Method

A retrospective study was performed on patients who were admitted for SSEH in 3 academic neurosurgical centers from January 2010 to June 2021. Clinical parameters, including clinical condition on admission, anticoagulants use, imaging modality, the timing and type of surgery performed, and outcomes, were collected. Prognostic factors were analyzed. The Frankel scale was used to assess the clinical condition.

Results

A total of 105 patients with SSEH were retrieved from medical records, with a mean age of 51.3 years. Eighty-three patients (79%) complained of acute onset of severe neck or back pain. Eighty-two patients (78%) suffered from moderate to severe neurologic deficits (Frankel scale A–C). Anticoagulation usage was found in 20% of cases. Lower thoracic spine ($p = 0.046$), use of anticoagulants ($p = 0.019$), sphincter function dysfunction ($p = 0.008$), severe neurologic deficits at admission ($p < 0.001$), and rapid deterioration (<1 hour, $p = 0.004$) were found to be associated with poor outcomes. Surgical decompression was performed in 74 (70%) cases. The univariate and multivariate analysis revealed that preoperative severe neurologic deficits ($p = 0.005$) and extended paraplegia time (>12 hours, $p = 0.004$) were independent adverse prognostic factors. The univariate analysis revealed that lower thoracic spine location ($p = 0.08$) and rapid progression (<6 hours, $p = 0.005$) were correlated with poor prognosis, but the multivariate analysis failed to identify them as independent prognostic factors.

Discussion

Adverse prognostic factors for SSEH might include thoracic segment location, use of anti-coagulation, severe neurologic deficits on admission, sphincter dysfunction, and rapid progression. Preoperative neurologic deficit and extended paraplegia time were strongly correlated with the prognosis in the subset of patients who underwent surgical decompression. Timely surgical decompression is recommended for patients with moderate/severe neurologic deficits or progressive neurologic deterioration.

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Glossary

DSA = digital subtraction angiography; IVVP = internal vertebral venous plexus; SSEH = spontaneous spinal epidural hematoma.

Spontaneous spinal epidural hematoma (SSEH) is a relatively uncommon but potentially devastating disorder first described in 1869 by Jackson.¹ Its estimated incidence is 0.1 in 100,000 per year.² Patients generally present with sudden onset acute back or neck pain, followed by progressive motor, sensory symptoms, or sphincter dysfunction, which ultimately leads to complete/incomplete motor deficit caused by spinal cord/nerve root compression or cauda equina. Once the diagnosis is suspected, early surgical intervention should be considered.³⁻⁵ Although there is no consensus on the ideal timing for surgery, most authors consider that surgery performed within 12 hours after the onset of symptoms is associated with better outcomes.³ Owing to the relative rarity of SSEH, many of these reports were gleaned from single-center data with limited sample sizes. The clinical implication of these studies is therefore might be limited. A recent individual patient data meta-analysis from the published literature enrolled 617 patients and identified that severity of preoperative neurologic deficit and use of anticoagulants determine the postoperative outcomes, but not the time interval between symptom onset and surgery.⁶ The patients managed conservatively need to be included in future studies to provide more generalized suggestion for management plan.

In this study, we presented a retrospective case-control study that analyzed the demographic, clinical, treatment, and outcome data from our largest SSEH patient cohort. We aimed to suggest prognostic factors that affect the outcome in patients with SSEH.

Methods

Study Design

We conducted a retrospective multicenter study that evaluated prognostic factors in patients who were diagnosed with SSEH and treated at 3 neurosurgical centers in Hangzhou, China, from January 1, 2010, to June 30, 2021.

Standard Protocol Approvals, Registrations, and Patient Consents

This observational study was performed following the Strengthening the Reporting of Observational Studies in Epidemiology guidelines.⁷ Ethics approval for this study was obtained from the Institutional Review Board of each participating hospital (the First and Second Affiliated Hospitals of Zhejiang University and the Affiliated People's Hospital of Hangzhou Medical College) (No. 2021QT216). Because this was a retrospective study, an exemption to patient informed consent was granted by hospitals' Institutional Review Board.

Study Population

The inclusion criteria were patients diagnosed with SSEH either by the attending surgeon intraoperatively or by magnetic resonance imaging (MRI) in those managed non-surgical. The exclusion criteria were (1) hematoma related to trauma or spinal surgery; (2) hematoma associated with spinal punctures/catheters, arteriovenous malformation, or tumor; (3) patients with a short-term follow-up (less than 1 month).

The spinal segments involved were divided into cervical (C1-C5), cervical-thoracic (C6-T3), thoracic (T4-T8), thoracolumbar (T9-L1), and lumbar (L2 onward), to describe SSEH distribution. This classification is based on spinal cord vascular distribution, spinal canal anatomic features, and neurologic function relevance.⁶ The Frankel scale was applied to evaluate the neurologic function. Therapeutic strategies were divided into 2 major categories: surgical decompression without fixation and conservative treatment, including osmotic diuretics, steroid therapy, and neurotrophic drugs. Evaluation of prognosis was based on the assessment of neurologic function, which was performed 6 months after treatments. Clinical data and follow-up data were collected from sources including medical records, follow-up phone calls, or out-patient clinic visits with the patient's physicians.

Study Variables

Factors related to outcomes included (1) clinical characteristics, (2) location and involved vertebral levels, (3) coagulopathy, (4) severity of spinal cord injury (motor/sensory symptoms, pain level at onset, sphincter dysfunction, and Frankel scale), (5) progression interval, (6) preoperative interval, and (7) paraplegia interval. The progression interval was defined as the time interval from the onset of the symptoms to the emergence of severe neurologic deficits, such as paraplegia. The preoperative interval was defined as the time interval from the onset of the disease to the operation. The paraplegia interval was defined as the time interval from the emergence of severe neurologic deficits, such as paraplegia, to the operation.

A good functional outcome was defined recovering from Frankel A/B to at least Frankel C at the latest follow-up. If patients started with the Frankel score of C/D, then they need to have at least 1 class improvement to be classified as good. Patients with a Frankel scale of E before and after treatment would also be defined as good functional outcomes. The rest of the scenario was defined as poor functional outcomes.

Statistical Analysis

The χ^2 test was used to compare the categorical variables. For continuous variables, the normality of the data sets was assessed using the Shapiro-Wilk normality test. If the

Table 1 Patient Demographics and Clinical Characteristics (n = 105)

	Value, n (%)
Sex	
Male	64 (60.9)
Female	41 (39.1)
Age, y, mean (range)	51.3 (16–88)
Segmental location	
Cervical (C1–C5)	22 (20.9)
Cervico-thoracic junction (C6–T3)	45 (42.8)
Thoracic (T4–T8)	16 (15.2)
Thoraco-lumbar junction (T9–L1)	18 (17.1)
Lumbar (L2–L5)	4 (3.8)
No. of segments involved	
1–2 segments	37 (35.2)
3–4 segments	49 (46.7)
>4 segments	19 (18.1)
Location hematoma	
Posterior/posterolateral	95 (90.5)
Anterior/circumferent	10 (9.5)
Anticoagulants usage	
Anticoagulants or antiplatelet	21 (20.0)
No anticoagulants	84 (80.0)
Hypertension	
Yes	19 (18.1)
No	86 (81.9)
Pain at onset	
Yes	83 (79.0)
No	22 (21.0)
Frankel scale	
A	33 (31.4)
B	23 (21.9)
C	26 (24.8)
D	7 (6.7)
E	16 (15.2)
Treatment strategy	
Surgery	74 (70.5)
Controversy	31 (29.5)

normality hypothesis was rejected, the differences between groups were calculated using the nonparametric, unpaired Mann-Whitney *U* test. Although $p < 0.05$ was used to define

statistical significance for all inferential statistics, a p value threshold of ≤ 0.1 was used in univariate analysis to include potential risk factors into the multivariate model. All analyses were performed with SPSS (version 25; IBM Corp., Armonk, NY).

Data Availability

Anonymized data not published within this article will be shared on reasonable request to the corresponding author.

Results

Patient Demographics

Patient demographics are presented in Table 1. A total of 105 patients diagnosed with SSEH were identified with a mean age of 51.3 years (range 16–88 years). Sixty-four (60.9%) patients were male, and 41 (39.1%) were female (including a pregnant woman), with a sex ratio of 1.5:1. The age showed a bimodal distribution, with a peak incidence in the second and sixth decades of life (Figure, A). Twenty-one patients were on anticoagulants (warfarin $n = 12$ and rivaroxaban $n = 1$) or antiplatelet medications (aspirin $n = 8$).

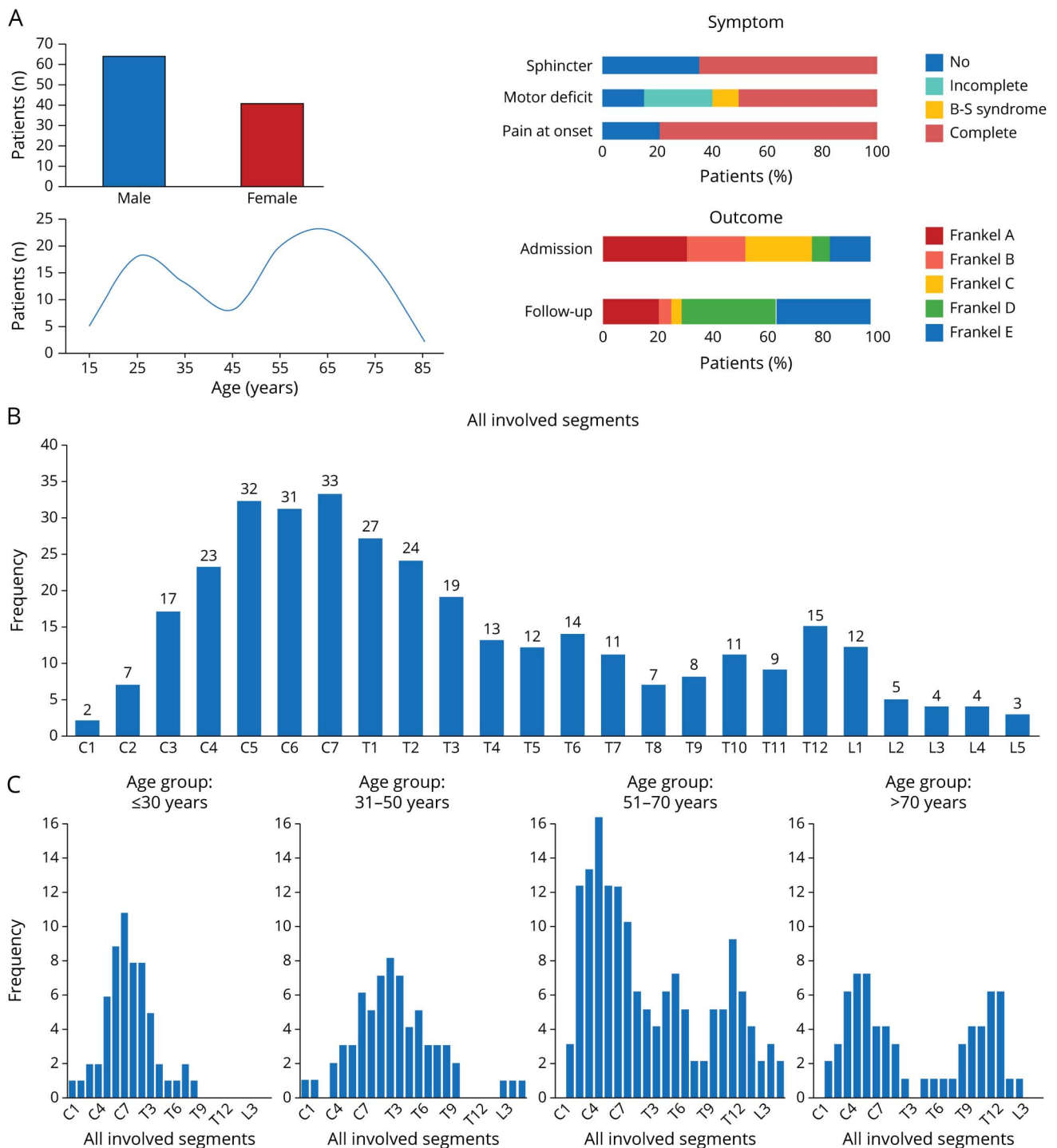
Hematoma Spinal Levels

All patients underwent MRI examinations (3.0 or 1.5 T) within 3 days from the onset of symptoms. On T1-weighted images, the signal intensity of hematoma was isointense and isointense to hypointense on T2-weighted images. Any spinal cord levels could be affected. The hematoma distributions were as follows: 22 cases in the cervical spine (20.9%), 45 cases in the cervicothoracic spine (42.8%), 16 cases in the thoracic spine (15.2%), 18 cases in the thoracolumbar spine (17.1%), and 4 cases in the lumbar spine (3.8%). The cervical and thoracic spines were the most commonly affected segments, corresponding to two-thirds of cases (Figure, B). The length of the hematoma ranged from 1 to 7 spinal levels, and on average, hematoma extended over 3.3 vertebral segments. The SSEH most commonly occurred in the dorsal or dorsolateral part of the vertebral canal ($n = 95$, 90.5%), with the rest in the ventral epidural space ($n = 10$, 9.5%). The distribution age relevance analysis showed that younger age (0–50 years) correlated with the cervicothoracic area, while older patients (51 years or older) showed a bimodal distribution, with 2 peaks at the cervicothoracic and the thoracolumbar area (Figure, C).

Clinical Presentation

The chief complaints in most cases (79.0%) were sudden onset of neck/back pain. Twenty-two (21.0%) cases suffered from severe neurologic deficits without significant pain at the onset. Most patients presented with complete or incomplete spinal cord compression symptoms in several minutes, hours, or even in days. Eighty-two (78.1%) patients had a Frankel scale of A or B or C at admission. Only 16 cases had no neurologic deficits with a Frankel scale of E. Ten (9.5%) cases were of the Brown-Sequard type.

Figure Overview of the Clinical Characteristics, Neurological Deficits, and Segmental Distribution of Hematomas



(A) Overview of the clinical characteristics and Frankel scale before/after treatment in all 105 patients with SSEH. (B) Segmental distribution of hematomas in all 105 patients with SSEH (all affected segments are counted). (C) Segmental distribution according to age category. SSEH = spontaneous spinal epidural hematoma.

Outcome

A total of 64 (61%) patients received surgical decompression, all of whom suffered from severe neurologic deficits (Frankel scale A or B) before operation. Forty-one (39%) patients received medical therapy such as systemic corticosteroids and mannitol at admission. Of those who initially on conservative treatment, 10

patients (9.5%) required operation eventually because of clinical deterioration at an average of 7.5 days (5–14 days) after the injury. Among the rest 31 patients in the conservative group, 19 were Frankel scale D or E, 11 were Frankel scale C (5 of them showed improvement in the early period), and only 1 patient suffered from severe neurologic deficits (Frankel scale A).

Table 2 Outcome-Related Patient Characteristics (n = 105)

Characteristics	Functional recovery		p Value
	Good outcome ^a	Poor outcome	
Sex, n (%)			
Male	46	18	0.695
Female	28	13	
Age, y, mean (range)	50.7 (16–78)	53.9 (21–88)	0.613 (W)
Segmental location, n (%)			0.046 ^b
Cervical (C1-C5)	19	3	
Cervico-thoracic (C6-T3)	33	12	
Thoracic (T4-T8)	9	7	
Thoraco-lumbar (T9-L1)	9	9	
Lumbar (L2-L5)	4	0	
No. of segments involved			0.250
1–2 segments	25	12	
3–4 segments	38	11	
>4 segments	11	8	
Location hematoma			0.488
Posterior/posterolateral	66	29	
Anterior/circumferent	8	2	
Anticoagulants usage			0.019 ^b
None	62	22	
Anticoagulants	5	8	
Platelet inhibitor	7	1	
Hypertension			0.771
Yes	13	6	
No	61	25	
Pain at onset			
Yes	60	23	0.429
No	14	8	
Sphincter dysfunction			0.008 ^b
Yes	42	26	
No	32	5	
Motor symptoms			<0.001 ^b
Normal	16	0	
Incomplete transverse	25	1	
Complete	26	27	
Brown-Sequard syndrome	7	3	

Table 2 Outcome-Related Patient Characteristics (n = 105) (continued)

Characteristics	Functional recovery		p Value
	Good outcome ^a	Poor outcome	
Sensory symptoms			<0.001 ^b
Normal	18	1	
Incomplete transverse	42	4	
Complete	7	24	
Brown-Sequard syndrome	7	2	
Frankel scale			<0.001 ^b
A	9	24	
B	20	3	
C	24	2	
D	5	2	
E	16	0	
Progression interval (n = 89)			0.004 ^b
<1 h	17	20	
1–12 h	21	4	
<12 h	21	7	
Operative interval (n = 74)			0.266
≤12 h	11	4	
>12 h	34	25	
Paraplegia time (n = 74)			0.037 ^b
≤12 h	20	6	
>12 h	25	23	

^a The patient recovered from Frankel A/B to at least Frankel C at last follow-up.

^b Significant.

The follow-up duration ranged from 6 to 12 months. At the latest follow-up, good functional outcomes were observed in 74 (70.5%) patients while poor functional outcomes in 31 (29.5%) patients (Figure, A). The following adverse prognostic factors were identified: location at lower thoracic spine ($p = 0.046$), use of anticoagulants ($p = 0.019$), loss of sphincter function ($p = 0.008$), severe neurologic deficits on admission ($p < 0.001$), and short progression interval (<1 hour, $p = 0.004$; <6 hours, $p = 0.04$, data not shown). The potential prognostic factors are summarized in Table 2. Seven of 10 patients with the Brown-Sequard syndromes had good outcomes (Frankel scale E). However, there was no significant difference in outcomes between patients with paraplegia and Brown-Sequard type ($p > 0.05$, data not shown). Thus, the Brown-Sequard type

Table 3 Predictors for Good Outcome After Operative Decompression of Spinal Epidural Hematomas (n = 74)

Risk factor	Univariate		Multivariate	
	OR (95% CI)	p Value	OR (95% CI)	p Value
Sex	0.92 (0.35–2.39)	0.86	Not selected	
Age	0.99 (0.97–1.02)	0.70	Not selected	
Segmental location		0.36		0.46
Cervical (C1–C5)	1.0		1.0	
Cervico-thoracic (C6–T3)	0.55 (0.12–2.41)	0.42	0.63 (0.07–5.39)	0.67
Thoracic (T4–T8)	0.25 (0.04–1.44)	0.12	0.56 (0.04–8.10)	0.67
Thoraco-lumbar (T9–L1)	0.23 (0.05–1.18)	0.08	0.11 (0.01–1.23)	0.07
Segment length		0.18	Not selected	
1–2 segments	1.0			
3–4 segments	1.36 (0.47–3.95)	0.57		
>4 segments	0.39 (0.10–1.53)	0.18		
Anticoagulants usage	0.66 (0.22–1.96)	0.45	0.11 (0.01–1.14)	0.06
Sphincteric dysfunction	0.42 (0.13–1.31)	0.13	Not selected	
Preoperative Frankel scale		<0.001 ^a		0.005 ^a
A	1.0		1.0	
B	17.04 (4.05–71.73)	<0.001 ^a	91.70 (5.71–1,471.4)	0.001 ^a
C	35.78 (4.08–313.4)	0.001 ^a	270.55 (6.31–11,610)	0.004 ^a
D and E	2.56 (0.31–20.99)	0.38	1.96 (0.07–57.63)	0.70
Progress interval (>6 h)	2.31 (1.28–4.16)	0.005 ^a	1.80 (0.69–4.72)	0.23
Time to surgery (>12 h)	0.49 (0.14–1.74)	0.27	Not selected	
Paraplegia time (>12 h)	0.33 (0.11–0.95)	0.04 ^a	0.03 (0.00–0.33)	0.004 ^a

Abbreviation: OR = odds ratio.

^a Significant.

itself was not an independent predictor of good outcomes. This is probably due to the differences in the extent of spinal cord compression between hemi-cord dysfunction and paraplegia group.

After univariate logistic regression analysis (Table 3), the variables associated with poor outcome in the subset of the surgery group were (1) hematoma located in the lower thoracic spine ($p = 0.08$), (2) short progression interval (<6 hours, $p = 0.005$), (3) extended paraplegia time ($p = 0.04$), and (4) severe neurologic deficits before operation ($p < 0.001$). After multivariable analysis (Table 3), the severity of preoperative motor/sensory deficits (Frankel scale A/B/C, $p = 0.005$) and extended paraplegia time (>12 hours, $p = 0.004$) were found to be associated with poor outcomes in the subset of surgical decompression group. Hematoma located in the lower thoracic spine ($p = 0.07$), short progression interval ($p = 0.23$), and use of anticoagulants ($p = 0.06$) were not associated with outcome in this subset by multivariate analyses.

Discussion

SSEH is a rare entity with approximately 1,100 cases reported in the literature by 2021. However, most neurosurgeon or neurologist would have experienced at least 1 of these cases in their careers.⁸ In our case series, the incidence of SSEH was higher in male patients and the age profile showed a bimodal distribution, with peak incidences in the second and sixth decades of life (Figure, A). Twenty-one cases were on anticoagulants in our study. Compared with cases with normal coagulation profile, patient with coagulopathy/anticoagulated most probably will have “prolonged clotting time,” leading to a larger hematoma. Because coagulopathy/anticoagulated status is unlikely to be the direct cause of bleeding, we enrolled patients who were on anticoagulants. Age-hematoma distribution analysis showed that younger age (younger than 50 years) correlated with the cervicothoracic area, while older patients (older than 50 years) showed a bimodal distribution, with peaks at the cervicothoracic and the thoracolumbar area (Figure, C). The possible explanation would be, in

both younger and older groups, the posterior internal vertebral venous plexus (IVVP) is particularly prominent at the cervicothoracic area where SSEH frequently occurs.⁹ By contrast, at the thoracolumbar area, the posterior IVVP is more prominent with more advanced age.⁶ Zhang et al. reported positive study findings, that is, spinal epidural arteriovenous fistulas, during the presurgical spinal digital subtraction angiography (DSA) in 15% of SSEH cases (6 of 40 cases).¹⁰ They also reported that, of those with negative DSA findings, 10 cases were found to have abnormal vessels dilations or expansion of the vascular bed during surgical decompression. Thus, the authors concluded that bleeding from the posterior IVVP could be the most likely source leading to SSEH.^{11,12}

There is no consensus on the best SSEH management approach. Patients with mild clinical conditions or significant recovery were more likely to receive conservative management. Decompressive laminectomy is recommended in patients with severe neurologic dysfunction or clinical deteriorations.¹³ The degree of preoperative neurologic deficit and the time interval between symptoms onset to surgery have been suggested as critical factor predicting patient recovery after treatment.⁴ In our study, every 1 in 3 patients were managed conservatively, which was similar to other studies (23%)¹³ in which 73%–84% of patients could recover completely after conservative management.^{13,14}

Patients on anticoagulants (warfarin) were found to have a worse outcome (Table 2). This observation is also in line with the findings from the previous studies.⁶ Anticoagulant therapy may contribute to larger hematoma size and poorer outcomes. However, both univariate analysis and multivariable analysis revealed that the use of either anticoagulants or platelet inhibitor in the surgery subgroup was not an independent risk factor for prognosis (Table 3). Patients taking anticoagulants/antiplatelets medications would receive plasma, cryoprecipitate, and platelet transfusion to correct the coagulopathy before the surgery. Previous study suggested that correcting the coagulation function before surgery may stop the progression of hematoma and potentially lead to improved surgical outcomes.³

Thoracic SSEH has been reported to be associated with the worst clinical outcome.^{6,8} Similarly, in our study, middle and lower thoracic SSEH had worse prognosis (Table 2). The likely reason may be that there are 3 major arterial supplies to the spinal cord, which are divided into cervicothoracic (C1-T2), midthoracic (T3-T9), and thoracolumbar (T10-L2) branches. The midthoracic area seems the most vulnerable to an ischemic insult.¹⁵ Both univariate analysis and multivariable analysis showed that the lower thoracic spine location was associated with worse outcomes in the surgery group; however, the observed differences were not statistically significant (Table 3).

The severity of neurologic deficit at the time of intervention was the most important prognostic factor. Poor neurologic function on admission has been reported to be associated

with the worst prognosis.^{6,8,10} The results of our retrospective analysis supported this association (Table 2). It is worth noting that preservation of sphincter function increased the chances of good outcomes compared with those with complete loss of sphincter function (Table 2). In our study, both the univariate and multivariate analyses showed that severe preoperative neurologic deficit (Frankel scale grade A) was an independent predictor of poor outcome after surgical decompression (Table 3).

The development of early neurologic deficits is most likely caused by direct neural compression.¹⁶ The focal mechanical force could cause conduction blocks and damage to the myelin in the spinal cord.⁶ This direct and severe neural compression seems to be responsible for the short progression interval. Besides that direct neural compression, the impairment of the venous drainage of the spinal cord could lead to venous congestion, white matter edema, and axonal swelling.⁶ These secondary circulatory disturbances, likely to be responsible for the longer progress interval, could be relieved by direct surgical decompression or spontaneous hematoma resolution. The univariate analysis demonstrated that the progression interval was a significant prognostic factor in our study (Tables 2 and 3). These patients with a rapid progression are more prone to a poor prognosis. However, multivariate analysis failed to confirm this. One explanation could be the contributing effects of other possible confounding factors included in our regression analysis such as anticoagulants. We also noticed that anticoagulants were not associated with outcome in univariate and multivariate analyses ($p = 0.06$) in (Table 3).

Groen and van Alphen⁵ demonstrated that preoperative neurologic condition and preoperative time interval were correlated with the outcomes of SSEH. Mukerji and Todd⁴ recently reviewed the literature on the timing and outcome of surgery and concluded that surgery within 12 hours was associated with the best outcome, even in paraplegic patients. Several studies have suggested that early surgical decompression within 12–24 hours is the key to improve patient prognosis.^{5,6,17} Our results only partially confirm the literature findings. For example, the operative interval was not always associated with postoperative outcomes. On the other hand, univariate and multivariable analyses showed that preoperative paraplegic time was an independent predictor of outcome after surgical decompression (Table 3). In this regard, the preoperative interval should be defined as the interval between emergences of severe neurologic dysfunction to surgery. As soon as patients manifest neurologic deterioration, surgery should occur at the earliest possible opportunity.

This study included the largest sample size of patients with SSEH ever studied. Adverse prognostic factors for patients with SSEH include thoracic segment, use of anticoagulation, severe neurologic deficits on admission, sphincter dysfunction, and short progression interval. Preoperative neurologic deficit and paraplegia time were strongly correlated with the prognosis in the subset of patients who underwent surgical decompression. Timely surgical decompression should be

recommended for patients with moderate to severe neurologic deficits or progressive neurologic deterioration. We believe that our study findings will provide new insights and guidance for neurosurgeons to improve SSEH management.

This study is a retrospective analysis and thus bears the inherent limitations of such studies. Another main limitation was the lack of representativeness of the study cohorts to general population. Because of the rarity of SSEH and the various inclusion and exclusion bias, our data may not represent the general population. Therefore, a prospective, multicenter, controlled, parallel study is needed in the future.

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Appendix (continued)

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