ORIGINAL ARTICLE

Acta Orthop Traumatol Turc 2014;48(4):437-442
doi: 10.3944/AOTT.2014.13.0133

Acute postoperative cervical spinal epidural hematoma

Guodong YIN[1,2], Bin NI[1]

[1]Department of Orthopedic Surgery, Changzheng Hospital, Second Military Medical University, Shanghai, China;
[2]Department of Orthopedic Surgery, 422 Hospital of PLA, Zhangjiang, China

Objective: The aim of this study was to assess the clinical presentation, causes or risk factors, treatment and outcomes of symptomatic acute postoperative cervical spinal epidural hematoma (APCSEH).

Methods: The study retrospectively reviewed all patients who underwent open cervical spinal surgery between January 1, 2004 and October 31, 2008. Perioperative coagulation parameters, past medical history, age, gender, time to decompression, pre- and postoperative (decompression operation) neurological status, as well as the interval of hematoma formation and level of segments were reviewed. Potential risk factors for extradural hematomas were examined. Patients who were diagnosed with APCSEH underwent immediate surgical drainage of the hematoma.

Results: Over 2,338 cervical spinal surgeries were performed by the Orthopedic Spinal Disorder Group in our hospital between 2004 and 2008. Twelve (0.5%) cases of APCSEH were identified. Two-thirds of the patients had history of hypertensive disease or transitory hypertensive status after surgery. No patients had abnormal preoperative coagulation parameters, bleeding diathesis or developed intraoperative coagulopathy. All patients were treated with hematoma evacuation within 2 hours of the ultimately definitive diagnosis of hematoma.

Conclusion: Acute postoperative cervical spinal epidural hematoma is a rare cause of postoperative neurological deterioration. Hypertensive disease and the method of multilevel cervical surgery appear to increase bleeding and predict formation of hematoma. Early diagnosis and evacuation of the hematoma can result in resolution of the neurological deficit.

Key words: Cervical spinal surgery; risk factor; spinal epidural hematoma; treatment.

Spinal epidural hematoma is a rare condition. It was first described by Jackson in 1869,[1] and only 350 cases were reported in the literature between 1869 and 2003.[2] Although asymptomatic spinal epidural hematoma has been identified by CT and MR imaging studies in 33% to 100% of patients who underwent lumbar disc or decompression surgery, most cases are asymptomatic and spinal epidural hematoma only rarely becomes symptomatic. Once neurological deterioration due to spinal epidural hematoma develops, emergency evacuation is required. In cases in which emergency evacuation is not performed rapidly enough, neurological sequelae such as paralysis may persist.[3] Coagulopathy, hypertension, increased venous pressure and vascular malformation were once thought to play an important role in the formation of spinal epidural hematoma. Others have proposed a constellation of factors believed to predispose postoperative epidural hematoma, including revision surgery, multilevel procedures, systemic illness and bleeding.[4]

Correspondence: Guodong YiN, MD. Second Military Medical University Changzheng Hospital, Department of Orthopedic Surgery, Huangpu 200040, Shanghai, China.
Tel: (+86-021) 8188 5623 e-mail: ygd0810@126.com

Submitted: October 10, 2013 **Accepted:** March 18, 2014

Available online at
www.aott.org.tr
doi: 10.3944/AOTT.2014.13.0133
QR (Quick Response) Code

Operation is an important cause of spinal epidural hematoma.[5,6] While few cases have been focused on acute postoperative cervical spinal epidural hematoma (APCSEH), such cases often result in more extensive and permanent neurological loss than others. The cause, risk factors, characteristics, treatment options and prognosis of APCSEH remain a problem.

We report a series of APCSEH in a subset of patients who awoke from surgery neurologically improved, but then deteriorated within 24 hours. The aim of this study was to call attention to the clinical features of this uncommon entity and to determine if any common elements exist that predispose the development of APCSEH.

Patients and methods

The study reviewed consecutive patients who underwent cervical spinal surgery at the hospital between January 1, 2004 and October 31, 2008. Patients with cervical punctures, steroid use and/or local anesthetic injections were excluded.

The diagnosis of APCSEH was made on the basis of progressing paralysis, numbness of both lower extremities and requirement of further surgery to evacuate the hematoma. Theater logbooks were searched for all cases of 'evacuation of hematoma'. All patients who unexpectedly returned to the operating theater within 2 weeks of the initial operation were also identified. A 2-week period was determined to be sufficient as the reported maximum period was 8 days after surgery.[2]

Mean follow-up period was 14 (range: 4 to 25) months. Medical records, perioperative coagulation parameters, history, age, gender, time to compression, pre- and postoperative neurological status and the interval of neurological deficit were reviewed. Potential risk factors for APCSEH, including medications and underlying hematological conditions that predispose bleeding coagulation studies, the use of wound drains and history of previous surgeries were also examined. Neurological function was assessed using the Frankel scale[7] preoperatively and after evacuation of the hematoma.

After identifying all cases of APCSEH, a control group was randomly selected from patients undergoing a procedure of similar complexity at the same section of the spine by the same surgeon within 6 months of the initial operation to match the study group and increase the power of the study. A detailed review of subjects' medical records was performed and a variety of perioperative possible risk factors were determined.

Statistical analyses were performed using the statistical package SPSS for Windows. As the continuous variables in this study exhibited an abnormal distribution, the non-parametric Mann-Whitney U-test was used to test the strength of the association between each continuous variable and spinal epidural hematoma. The Fisher exact test was used to test the significance of associations between the categorical variables and spinal epidural hematoma. Factors associated with spinal epidural hematoma in univariable analyses ($p<0.05$) were entered into a multivariable logistic regression model to identify whether the factors were independently associated with spinal epidural hematoma. The Spearman's rank correlation was used to test the strength of the association between two independent continuous variables and the relationship between the interval from symptom onset to surgery and neurological outcome. P values of less than 0.05 were considered significant.

Results

A total of 2,338 patients who underwent cervical spinal surgeries between January 1, 2004 and October 31, 2008 were identified from their medical charts. Median age of the patients was 49.2 (range: 31 to 82) years and the female to male ratio was 1:2.9. Of the 2,338 patients, 313 (13.4%) were treated with cervical decompression by a posterior approach, 1,889 (80.8%) by an anterior approach and 136 (5.8%) by both approaches. History of hypertension was present in 628 patients (26.9%).

Over the 4 year review period, 12 patients (0.5%) were identified with APCSEH due to a clinical deterioration occurring within 24 hours after index procedure. The median age was 48 (range: 35 to 68) years and the female to male ratio was 1:2. Eleven patients underwent anterior procedures (corpectomy and fusion with instruments) and 1 with a posterior approach (laminectomy and fusion with instruments) (Table 1). Medium Hemovac drains were used in all cases after the index procedure. Drains were maintained for the first postoperative 24 to 48 hours.

Table 2 presents an overview of possible perioperative risk factors. Factors found to be significantly associated with spinal epidural hematoma in the univariable analysis were hypertension ($p<0.01$) and bi-level/multilevel procedure ($p<0.03$). No other factor was found to be significantly associated with increased risk. A multivariable logistic regression model was used to determine if significant individual risk factors were independently associated with spinal epidural hematoma. Analysis confirmed that hypertension ($p=0.010$) and bi-level/multilevel procedure ($p=0.029$) were significant independent predictors of spinal epidural hematoma. All patients

Table 1. Basic statistics.

Patient No.	Age/ Sex	MH	Surgery	Frankel score Pre-/Postoperative	Improvement in Frankel score (number of Grades)	Duration of symptoms (hrs)	BP (mmHg)
1	57/F	Hypertension	C6 (ACCF)	C/E	2	0.6	145/85
2	53/M	None	C5 (ACCF)	B/D	2	0.6	140/86
3	38/M	None	C3/4 (ACDF)	E/E	0	1.2	134/79
4	50/M	None	C6/T1 (ACDF)	A/A	0	2.7	100/60
5	68/M	None	C5 (ACCF)	B/D	2	0.25	150/80
6	43/M	Hypertension	C5 (ACCF)	C/D	1	1.3	155/85
7	42/F	Hepatitis C	C5 (ACCF)	C/D	1	0.8	130/90
8	35/M	None	C5/6 (ACDF)	D/E	1	0.6	130/70
9	40/F	Hypertension	C4-C7 (posterior approach)	C/D	1	0.7	156/88
10	51/F	Hypertension	C4/5 (ACDF)	C/D	1	0.3	150/90
11	43/M	None	C5/6 (ACDF)	D/E	1	0.8	150/106
12	56/M	None	C5 (ACCF) + C6/7 (ACDF)	D/E	1	1.3	150/116

ACCF: Anterior cervical corpectomy and fusion; ACDF: Anterior cervical discectomy and fusion; BP: Blood pressure (measurement of blood pressure has been undertaken twice between the index surgery and hematoma evacuation surgery and the mean value of both was adopted); MH: Medical history.

had normal coagulation function tests at the time of the index surgical procedure (Table 3). Two-thirds of the patients had history of hypertensive disease or in transitory hypertensive status after operation (Table 1). None of the patients suffered from excessive blood loss (Table 2). No patients had previous spinal surgeries at the site of the acute spinal epidural hematoma. Five patients received irregular anti-inflammatory medication for pain control.

In the initial postoperative examination after the index operation, all patients had free use of all four limbs after operation. The initial presenting symptom which heralded the onset of neurological deterioration consisted of progressing motor weakness and sensory loss, or even disappearance of the lower limb function. This was often followed by tendon reflex disappearance. Frankel grades at the time of presentation were A in 1 patient, B in 2, C in 5, D in 3 and E in one. The average time to neurological deterioration was 5.0 (range: 0.6 to 15.8) hours. Patients' Frankel grades improved by a mean of 1.08, in terms of neurologic deficit at the time of discharge compared with the maximum deficit before evacuation surgery. Improvement in Frankel grade was significantly higher in patients who underwent evacuation surgery in a shorter period of time ($p<0.01$). There was a negative correlation between the improvement in Frankel grade and duration of symptoms (Spearman's rank correlation coefficient: -0.705).

Table 2. Possible perioperative risk factors.

Possible risk factors	Patients with postoperative SEH (n=12)	Patients without postoperative SEH (n=24)	p
Number of males	8	10	.289
Median age (range)	48.0 (35.0-68.0)	46.9 (36.0-68.0)	.710
Smoker	3	6	1.000
Diabetes	3	6	1.000
Hypertension	8	4	.007
NSAID	5	10	.194
Trauma*	2	2	.588
Previous spinal surgery	0	0	N/A
Bi-level/multilevel procedure†	7	4	.020
Length of operation (min) (range)	78.3 (53.0-96.0)	78.6 (53.0-104.0)	.973
Blood loss (ml) (range)	88.5 (75.0-110.0)	85.6 (53.0-104.0)	.468

N/A: Not applicable; NSAID: Non-steroidal anti-inflammatory drug; SEH: Spinal epidural hematoma.

*Trauma within 6 weeks leading to the operation.

†Bi-level/multilevel procedure defined as a procedure that involved more than one intervertebral disc.

Table 3. Possible perioperative hematological risk factors.

Possible hematological risk factors	Patients with APCSEH (n=12)	Patients without APCSEH (n=24)	p
APTT (s) (range)	28.3 (25.1-31.7)	29.7 (26.5-34.0)	.752
PT (s) (range)	11.0 (10.1-11.9)	10.6 (10.0-11.2)	.485
INR	0.9 (0.9-1.2)	1.0 (0.9-1.1)	.597
Platelets (10^9/L) (range)	186 (158-275)	204 (177-295)	.488
Hb (g/L) (range)	146 (137-168)	142 (137-168)	.594

APCSEH: Acute postoperative cervical spinal epidural hematoma; APTT: Activated partial thromboplastin time; Hb: Hemoglobin; INR: International normalized ratio; PT: Prothrombin time.

At the first attack, a high dose of methylprednisolone (30 mg/kg, by intravenous infusion within 15 minutes) was used to preserve the cervical spinal cord. Once the diagnosis of APCSEH was confirmed, emergent surgical evacuation of the hematoma was carried out as soon as possible. The original site of the surgery was re-explored and the clot evacuated. Spinal dura mater was washed repeatedly with cold saline in order to lower cellular metabolism and cease bleeding. The average interval from symptom onset to evacuation surgery was 0.93 hours. On subsequent re-exploration, all of the cases had drains during the immediate postoperative period.

Surgical evacuation of APCSEH resulted in neurological improvement in 11 patients. Mean follow up was 14 (range: 4 to 25) months. One patient had no improvement in neurological function after evacuation of the hematoma. Three patients returned to baseline neuro-examination status within 2 hours of their surgical decompression and 2 had right leg weakness, which mildly improved in the early postoperative period. One patient had residual right hand and leg weakness, which mildly improved at the 4th month follow-up and an additional patient had no change in neurological function even with treatment of hyperbaric oxygen and neurotrophic drugs at the 25th month follow-up.

Discussion

Postoperative spinal epidural hematoma is an uncommon complication of spinal surgery and occurs in 0.1%[6] to 0.22% of cases.[2] The incidence of APCSEH is higher than that of epidural hematoma and was 0.5% in our series. To our knowledge, no previous series on APCSEH has been published in the English literature.

Several factors have been implicated in the development of postsurgical spinal epidural hematoma, including preoperative use of non-steroidal anti-inflammatory medication, multilevel procedures (greater than 4 levels), surgical drains, blood loss of over 1 liter and postoperative use of anticoagulation.[8] However, others have argued that the use of postoperative anticoagulation is not associated with a higher risk of postsurgical spinal epidural hematoma. As of yet, none of these factors have been scientifically demonstrated to be significant in a large series. It remains unclear whether these factors play a role in the formation of APCSEH.

Uribe et al.[2] reported a higher incidence of delayed postoperative spinal epidural hematoma (DPOSEH) in patients undergoing previous spinal surgery at the same site at which the hematoma developed. The higher incidence was attributed to the formation of scar tissue throughout the various tissue layers of the posterior spinal structures, including the epidural space, bone, ligaments, muscle, subcutaneous fat and skin after surgery, reducing the ability to absorb blood and blood products and predisposing DPOSEH. In our reported cases, however, none of the patients in the APCSEH group received previous cervical spinal surgery or had a history of preoperative coagulopathy from medical illness or anticoagulation therapy before operation for anesthesia needs. Perioperative coagulation parameters and intraoperative blood loss (<150 ml) were within the normal range. We believe that patients who require bi-level/multilevel cervical procedures and have the history of hypertensive disease are possibly at a higher risk for developing an acute postoperative epidural hematoma.

We postulate that the intraspinal epidural venous plexus is the site of bleeding.[9-11] The theorized mechanism is that the valveless, thin-walled venous plexus is vulnerable to rupture with abrupt changes in expanded venous pressure.[11] Others have argued that the source is more likely to be a hemorrhage from the epidural arteries as pressure from arterial bleeding would be more likely to cause acute spinal cord compression. In fact, arterial bleeding is easy to find and stop in operation. If the venous plexus is indeed the source of bleeding, the relevant question of why the bleeding does not stop when an epidural hematoma increases the pressure in the epidural space enough to stop the bleeding, when the

epidural venous pressure should be much lower than the cerebrospinal fluid pressure arises.[12] We believe that the hypertensive disease and bleeding from the cervical vertebrae body easily increases the epidural venous pressure which goes beyond the normal cerebrospinal fluid pressure and leads to continuous bleeding.

Cullen et al. reported that patients do not develop signs and symptoms of acute spinal cord compression after therapeutic blood patch when a relatively large amount of blood is injected into the epidural space for a short period of time.[13] In the rare event that a postsurgical spinal epidural hematoma becomes symptomatic, prompt surgical intervention is warranted to prevent significant and irreversible neurological consequences. Sokolowski et al. found that after lumbar decompression, 58% of patients developed epidural hematoma of sufficient magnitude to compress the thecal sac beyond its preoperative state at one or more levels while none developed new postoperative neurological deficits.[14] In our study, epidural hematoma was often observed after cervical surgery. However, high blood pressure and bilevel/multilevel procedure will inevitably result in greater volume of blood, decreasing the epidural space.

In the present study, the most common presenting symptom before neurological deterioration in all patients was numbness in the lower limbs spreading upward. This was often followed by motor weakness or paralysis and disappearance of tendon reflex. Signs and symptoms in our cases varied in onset from 0.6 to 15.8 hours.

Acute spinal epidural hematoma should be suspected in patients with progressive pain or neurological deficit after spinal surgery. MRI is the best diagnostic method when APCSEH is suspected. MRI can provide differential diagnoses regarding pathologies that most frequently present with spinal pain and neurological deficit.[15] In our experience, hysteria was easily misdiagnosed as APCSEH. In our study, while 2 patients who complained of progressive paralysis below the operative spinal level had been previously misdiagnosed with APCSEH, epidural hematoma was ultimately definitively excluded. Hysteria is usually associated with high intensity and over-respiration.

In cases of APCSEH with severe paresis, rapid decompression surgery is the most suitable treatment and the severity of preoperative status influences recovery. Kebaish et al. reported an incidence of postoperative spinal epidural hematoma requiring evacuation ranging between 0.1 and 3% and that neurological recovery correlated with the time from the onset of neurological symptoms to evacuation of the hematoma.[16] In our research, there was statistically significant difference in the degree of neurological recovery if hematoma evacuation was performed within 24 hours. However, Kebaish et al. reported that when hematoma evacuation is delayed beyond 24 hours, neurological recovery is less likely to occur.[16] Groen and van Alphen showed that the critical factors for recovery after spontaneous spinal epidural hematoma were the level of preoperative neurological deficit and the operative interval.[17] Liao et al. reported that in patients with a complete deficit, good neurological recovery can be achieved after timely surgery when the interval between the initial ictus and surgery is less than 48 hours and when the duration of the complete neurological deficits is less than 12 hours.[18] In this study, all patients underwent emergent decompression surgery within 2 hours. Eleven of the twelve patients who underwent surgery recovered fully. However, one suffered from permanent residual symptoms, such as urinary dysfunction and motor weakness for poor preoperative neurological function.

Limitations of the study include the small groups, which decreases the ability to detect the possible real differences that might exist between the two groups. In addition, results may also be inevitably influenced by the experience and personal bias of the surgeons or data collectors and they may be less reflective of the risk factors itself, especially blood pressure measurement.

In conclusion, APCSEH is a rare cause of postoperative neurological deterioration. Hypertensive disease and the method of multilevel cervical surgery appear to increase bleeding and predict formation of hematoma. Early diagnosis and evacuation of the hematoma can result in resolution of the neurological deficit.

Conflicts of Interest: No conflicts declared.

References

1. Jackson R. Case of spinal apoplexy. Lancet 1869;2:5-6.
2. Uribe J, Moza K, Jimenez O, Green B, Levi AD. Delayed postoperative spinal epidural hematomas. Spine J 2003;3:125-9.
3. Ikuta K, Tono O, Tanaka T, Arima J, Nakano S, Sasaki K, et al. Evaluation of postoperative spinal epidural hematoma after microendoscopic posterior decompression for lumbar spinal stenosis: a clinical and magnetic resonance imaging study. J Neurosurg Spine 2006;5:404-9.
4. Swamy G, Tay B, Walker JC, Hu S, Berven S, Ames C, et al. Postoperative spinal epidural hematoma – management and neurological outcome. Proceedings of the NASS 20th annual meeting. The Spine Journal 2005;5:1-189.
5. Lawton MT, Porter RW, Heiserman JE, Jacobowitz R, Sonntag VK, Dickman CA. Surgical management of spi-

nal epidural hematoma: relationship between surgical timing and neurological outcome. J Neurosurg 1995;83:1-7.
6. Scavarda D, Peruzzi P, Bazin A, Scherpereel B, Gomis P, Graftieaux JP, et al. Postoperative spinal extradural hematomas. 14 cases. [Article in French] Neurochirurgie 1997;43:220-7. [Abstract]
7. Frankel HL, Hancock DO, Hyslop G, Melzak J, Michaelis LS, Ungar GH, et al. The value of postural reduction in the initial management of closed injuries of the spine with paraplegia and tetraplegia. I. Paraplegia 1969;7:179-92.
8. Awad JN, Kebaish KM, Donigan J, Cohen D, Kostuik JP. An analysis of risk factors and incidence of postsurgical spinal epidural hematoma. Proceedings of the NASS 18th annual meeting. The Spine Journal 2003;3:67-171.
9. Groen RJ, Ponssen H. The spontaneous spinal epidural hematoma. A study of the etiology. J Neurol Sci 1990;98:121-38.
10. Hentschel SJ, Woolfenden AR, Fairholm DJ. Resolution of spontaneous spinal epidural hematoma without surgery: report of two cases. Spine 2001;26:E525-7.
11. Patel H, Boaz JC, Phillips JP, Garg BP. Spontaneous spinal epidural hematoma in children. Pediatr Neurol 1998;19:302-7.
12. Henderson RD, Pittock SJ, Piepgras DG, Wijdicks EF. Acute spontaneous spinal epidural hematoma. Arch Neurol 2001;58:1145-6.
13. Cullen DJ, Bogdanov E, Htut N. Spinal epidural hematoma occurrence in the absence of known risk factors: a case series. J Clin Anesth 2004;16:376-81.
14. Sokolowski MJ, Garvey TA, Perl J 2nd, Sokolowski MS, Cho W, Mehbod AA, et al. Prospective study of postoperative lumbar epidural hematoma: incidence and risk factors. Spine (Phila Pa 1976) 2008;33:108-13.
15. Gelabert M, Iglesias M, González J, Fernández J. Spontaneous spinal epidural hematomas: review of 8 cases. [Article in Spanish] Neurologia 2003;18:357-63. [Abstract]
16. Kebaish K, Matteri R, Campbell J. Correlation between neurological recovery and timing of surgical decompression of postoperative spinal epidural hematoma. Proceedings of the NASS 21st annual meeting. The Spine Journal 2006;6:1-161.
17. Groen RJ, van Alphen HA. Operative treatment of spontaneous spinal epidural hematomas: a study of the factors determining postoperative outcome. Neurosurgery 1996;39:494-509.
18. Liao CC, Lee ST, Hsu WC, Chen LR, Lui TN, Lee SC. Experience in the surgical management of spontaneous spinal epidural hematoma. J Neurosurg 2004;100(1 Suppl Spine):38-45.