Clinical Case Report

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Spontaneous spinal epidural hematoma management with minimally invasive surgery through tubular retractors

A case report and review of the literature

Chao-Feng Fu (MD), Yuan-Dong Zhuang (MD), Chun-Mei Chen (PhD)*, Gang-Feng Cai (MD)*, Hua-Bin Zhang (MD), Wei Zhao (MD), Said Idrissa Ahmada (MD), Ramparsad Doorga Devi, Md Golam Kibria

Abstract

To report a minimally invasive paraspinal approach in the treatment of a case of spontaneous spinal epidural hematoma (SSEH). We additionally aim to review the relevant literature to enhance our knowledge of this disease. SSEH is an uncommon but potentially catastrophic disease. Currently, most appropriate management is emergence decompression laminectomy and hematoma evacuation. An 81-year-old woman was admitted to the neurology department with a chief complaint of bilateral numbness and weakness of the lower limbs and difficulty walking for 4 days with progressive weakness developed over the following 3 days accompanied with pain in the lower limbs and lower back. No history of trauma was reported. Magnetic resonance imaging of the thoracolumbar spine demonstrated an epidural hematoma extending from T-12 to L-5 with thecal sac and cauda equina displacement anterior. The patient was treated in our department with a minimally invasive approach. This operation method had been approved by Chinese Independent Ethics Committee. Three months following the operation, the patient had regained the ability to walk with the aid of a cane and myodynamia tests revealed normal results for the left lower limb and a 4/5 grade for the right limb. Importantly, no complications were exhibited from the surgical operation. The minimally invasive paraspinal approach through tubular retractors is demonstrated here as an effective alternative method for the treatment of SSEH.

Abbreviations: CT = computed tomography, MRI = magnetic resonance imaging, SSEH = spontaneous spinal epidural hematoma.

Keywords: minimally invasive surgery, spontaneous spinal epidural hematoma, tubular retractors

1. Introduction

Spontaneous spinal epidural hematoma (SSEH) is an uncommon condition with an uncertain etiology that results in permanent neurological deficit or death when not treated appropriately.[1,2] This disease represents 0.3% to 0.9% of all epidural space-occupying lesions.[3]

It is characterized by the initial manifestation of sudden-onset acute back or neck pain around the affected vertebra followed by the rapid progression of neurological dysfunction, which is caused by compression of the thecal sac and cauda equina.[4] Many cases can be diagnosed upon detailed neurological examination and magnetic resonance imaging (MRI).[5] Despite this, the etiology of SSEE remains elusive in many cases, with no definitive inciting event. Numerous risk factors have been reported including anticoagulant therapy, aspirin, coronary thrombolysis, lumbar puncture, hypertension, and ankylosing spondylitis.[6,7]

Most surgeons advocate aggressive and prompt surgical intervention for SSEH cases, interventions which may cause thecal sac and cauda equina compression and neurological dysfunction. Today, the most widely used surgical approach is laminectomy.[8–11]

Here we reported a case of SSEH partial recovery from neurological dysfunction after minimal invasive laminectomy through tubular retractors. In our department, we have generally used this approach to treat disc herniation, intraspinal tumors, and spinal stenosis. This procedure is similar to partial laminectomy with the exception of using a tubular retractor instead of an open approach (Fig. 1).

2. Case

An 81-year-old woman was admitted to the neurology department experiencing persistent mild frequent urination and dysuria for 1 year without any reported inducing factors. The symptoms developed included numbness and weakness of the bilateral lower limbs and difficulty walking (more severe in the right lower

Editor: Helen Gharaei.

CF-F is the first author, and YD-Z contributed equally to this work and should be considered co-first author.

The authors declare no conflict of interest.

Department of neurosurgery, Fujian Medical University Union Hospital, Fuzhou, Fujian, China.

* Correspondence: Chun-Mei Chen, Department of neurosurgery, Fujian Medical University Union Hospital, NO.29, Xinquan Road, Fuzhou, Fujian, 350000, China (e-mail: cmchen2009@hotmail.com); Gang-Feng Cai, Department of neurosurgery, Chinese people's liberation army 180 hospital, Quanzhou 362000, China (e-mail: 261992047@qq.com).

Medicine (2016) 95:26(e3906)

Received: 17 November 2015 / Received in final form: 14 May 2016 / Accepted: 18 May 2016

http://dx.doi.org/10.1097/MD.0000000000003906

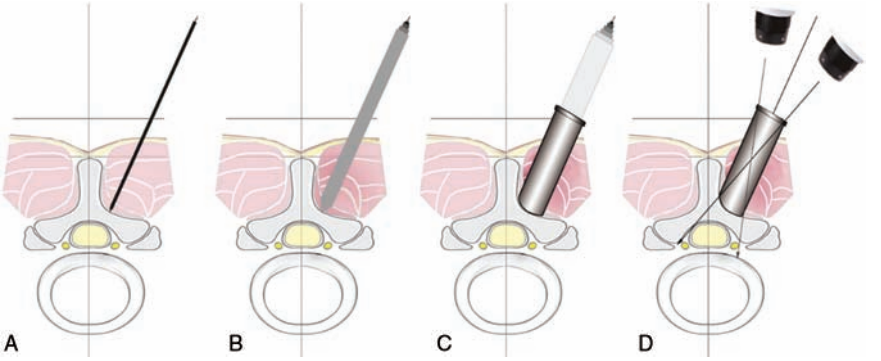

Figure 1. Schematic diagram of this approach.

extremity) 4 days before admission. Progressive weakness developed over the next 3 days accompanied with pain in the lower limbs and lower back.

Upon neurological examination, weakness was noted in the right lower and left lower extremities at grades of 0/5(R) and 3/5 (L), respectively, according to the Medical Research Council grading scale. The bilateral lower-limb exhibited normal muscle tension though deep-tendon reflexes appeared absent and Babinski sign was negative. A digital rectal examination indicated that rectal tone was normal for the patient. The remainder of the physical examination was normal. During sensory examination, this patient was not cooperative. The patient's medical history did not reveal any notable events. At the moment of admission, the patient's blood pressure was 158/78 mmHg and initial laboratory tests (including complete blood count, chemistry panel, erythrocyte sedimentation rate, C-reactive protein, and coagulation evaluation) revealed no remarkable contributions.

After initial consultation, this patient was admitted to our department. The day after admission, MRI of the thoracolumbar spine demonstrated an epidural hematoma extending from T-12 to L-5 with thecal sac and cauda equina displacement anterior (Fig. 2). Neurological deficits did not show any improvement during the course of the following 3 days. We next explained the condition of the disease and the operative method to the patient and her family. She was taken to the operating room for an emergency surgical decompression after the patient provided informed consent. After general anesthesia, the patient was placed in prone position. On the left side of the skin which is distant from midline 2 cm in T12, L3 level and right side of the skin which is distant from midline 2 cm in L2, L5 level, we made 4 longitudinal incisions about 2 cm length (Fig. 3E). Separation of subcutaneous tissue reaching the muscle fascia and the paraspinal muscle was bluntly separated to lamina from the space of the multifidus and longissimus. A tubular retractor device was inserted in this space and the appropriate position was ascertained under x-ray. High-speed burr was used to grind off the lower edge of the T12, L2, L3, and L4 lamina and the upper edges of the L1, L2, and L5 lamina (Fig. 3C), preventing damage to the zygapophysial joints and then dissected the ligamenta flava. The intraspinal dark red hematoma compressing the thecal sac and cauda equina were visible (Fig. 3A) and an aspirator and detacher were used to evacuate the hematoma. The hematoma in the vertebral canal, which is adjacent to the tube, could then also be eliminated via adjustment of the angle of the tube. After evacuation all the hematoma, hydrogen peroxide and povidone–iodine was used to wash the operative cavity until we could clearly observe the dural thecal sac expansion and cerebrospinal fluid pulsation (Fig. 3B). Drainage catheters were positioned at the intraspinal extradural after operation (Fig. 3D). The duration of the operation was about 3 h and the total blood loss was about 50 mL. No complications related to the surgical procedure were observed.

In the first 24 postoperative hours, there was no significant improvement in neurologic function. Physical examination similarly showed no change compared to preoperative results. The lower back and bilateral lower limb pain was reportedly relieved on the third postoperative day; moreover, there was an obvious improvement in the tendon reflexes of bilateral lower

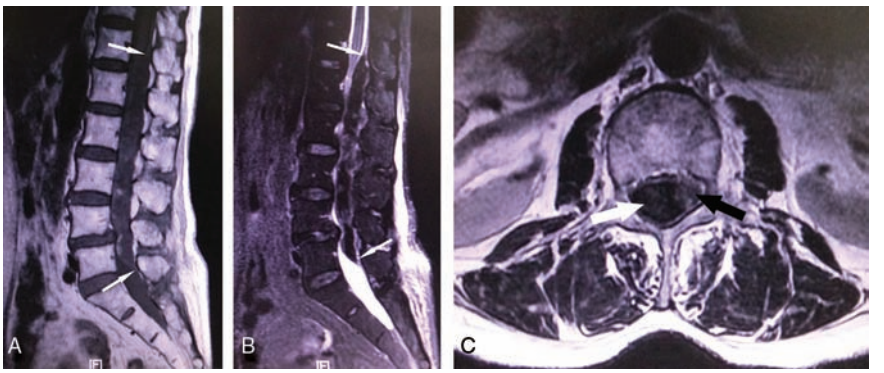

Figure 2. A, Sagittal T1-weighted MRI revealed a hematoma (isointense and slight hyperintense) located posterior to the thecal sac and cauda equine, extending from T12 to L5 (white arrows at the extremities of the mass). B, Sagittal T2-weighted MRI revealed a hematoma (isointense and hypointense, white arrows at the extremities of the mass). C, Axial T2-weighted MRI showed the hematoma (hypointense, white arrow) compressed the thecal sac and cauda equina (black arrow).

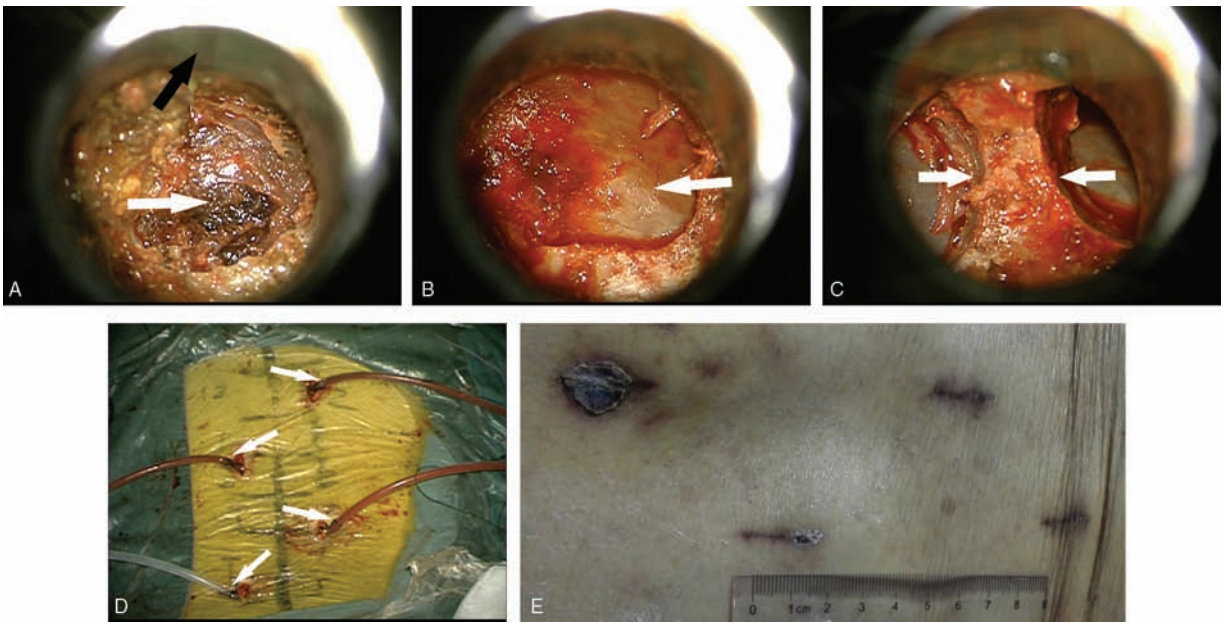

Figure 3. A, Dark red hematoma (white arrow) compressed the thecal sac. B, Thecal sac expanded and recovered (white arrow). C, The upper and lower edges of L2 vertebral plate were ground off (white arrows); zygapophysial joints were integrated. D, After surgery, drainage catheters (white arrows) were positioned in the intraspinal extradural. E, Each surgical incision was about 2 cm length (1 month postoperative).

limbs as blunt tendon reflexes were observed. On the tenth postoperative day, the patient returned to normal urination and the catheter was removed. The left lower extremity showed improvement during postoperative recovery and muscle strength examination was normal by the tenth day after surgery. The condition of the right lower extremity was not apparently improved as muscle force examination received a grade of 1/5 until the patient was discharged. By the 3-month follow-up examination, however, the patient could walk slowly with the help of a cane and myodynamia testing of right lower limb received a grade of 4/5.

3. Discussion

The pathogenesis of SSEH remains controversial. Patel et al considered that the bleeding is of venous origin. He supposed the mechanism that reversal of blood flow in the valveless epidural venous plexus caused by activities such as evacuation, sneezing, bending, coughing, and coitus could result in rupture of a fragile vein. Those activities could lead to the change of intrathoracic and intra-abdominal pressure. Beside this, traction on nerve roots during operations lead to disruption of the tortuous arterial plexus was suggested to be another source of epidural hematoma. Taking into consideration that the pressure in the epidural veins is less than intrathecal pressure (confirmed by the common observation that the normally expanded dural sac may tamponade epidural venous bleeding encountered during surgical procedures), Beatty and Winston[13] hypothesized that arterial source bleeding is the main cause of acute SSEH. However, Groen[14] has proposed that the majority of SSEH actually results from a rupture of the posterior internal vertebral venous plexus, in a review of 199 cases of SSEH. He has also considered that the rupture of the epidural artery might be responsible for some cases of SSEH. The anatomical structure of human vertebra canal usually restricts epidural hematomas to the dorsal aspect of the epidural space with possible extension to the lateral gutters.[12] In these cases, epidural hematoma is presented in the dorsal aspect, where the definitive cause of SSEH cannot be defined.

Various factors had been reported in the etiology of SSEH, such as anticoagulant therapy, aspirin, coronary thrombolysis, lumbar puncture, hypertension, ankylosing spondylitis, and vascular malformation.[10,15,16] Patient age also appears to highly affect the level distribution of SSEH as people who are less than 40 years old commonly experience SSEH localized to the cervicothoracic region, whereas patients over 40 years old usually experience SSEH in the cervicothoracic and thoracolumbar regions.[14] Our patient, who is 81 years old, presented a hematoma in the T12–L5, in accordance with the literature. The patient, however, presented no obvious causal factors for SSEH.

The typical symptoms of SSEH include a sudden onset of neck or back pain at the involved vertebral level with radiating pain, followed by rapid and progressive signs and symptoms of thecal sac and cauda equina compression.[17,18] MRI, myelography and computed tomography (CT) are commonly used to diagnose spinal epidural hematomas; however, CT cannot be used to determine when bleeding occurred and as such presents a limitation in terms of differential diagnosis. On the contrary, MRI affords a superior diagnostic capability and has replaced CT and myelography as the main diagnostic tool.[5,12] MRI can inform the precise position of the hematoma, the extent and time frame of the bleeding, detection of thecal sac and cauda equina compression, and spinal cord edema.[2] The patient presented here received MRI examination on the sixth day after the initial presentation of symptoms. In this case, we could see an obvious long-segmented SSEH had compressed and displaced thecal sac and cauda equina extending from the T12 to L5 (sagittal view by T2-weighted MRI). This SSEH demonstrated isointensity and slight hyperintensity by T1-weighted MRI and isointensity and hypointensity by T2-weighted MRI; this is in accordance with the subacute manifestation of hematoma in MRI. In axial T2-weighted MRI, we could clearly observe thecal sac and cauda equina compression and pushing to the left side of spinal canal.

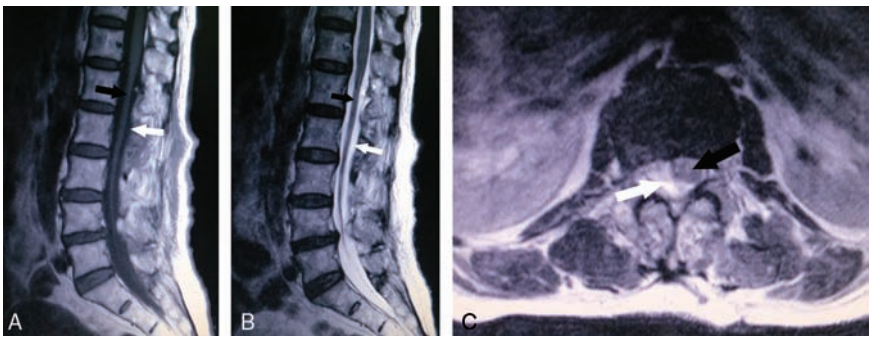

Figure 4. MRI of the patient 9 days postoperative [(A) sagittal T1-weighted, (B) sagittal T2-weighted, and (C) axial T2-weighted) complete removal of the epidural hematoma, the thecal sac (white arrows) and spinal cord (black arrows) expansion and recovery.

Neurological recovery is multifactorial. Preoperative neurological status and the time interval to surgery appear to be 2 critical factors influencing the prognosis of SSEH. A longer interval to surgery is detrimental to a positive neurological outcome,[11,19] on the other hand, Foo et al. hold the opinion that there is no correlation between interval surgery and neurological outcome.[20] Shin et al[2] speculated that early decompression (>12 hours constraining the ischemic change) yields a more favorable recovery. They have also suggested that patients who experience early incomplete motor and sensory loss have a better chance at neurological recovery. Liao et al[18] also reported that patients with preoperative incomplete neurological deficits had a better neurological recovery than patients with preoperative complete neurological deficits. Furthermore, prompt surgery (less than 48 hours from the initial onset of symptoms and less than 12 hours of complete neurological deficits) could also achieve a satisfactory neurological recovery in patients with preoperative complete neurological deficits. In our treatment, the patient was not diagnosed for the first time in our hospital though was admitted to our neurology department for conservative treatment. Patient underwent surgery more than 6 days from the onset of symptoms and complete motor neurological deficit was observed in this patient.

The mainstream treatment of SSEH is emergency decompression and evacuation of the hematoma for prevention of permanent neurological deficits.[19] Some authors have suggested that in patients who demonstrate continual signs of clinical improvement and stability, conservative treatment may be adopted.[5,21] Currently, the standard operation method for SSEH is the open laminectomy method. Iida Y reported that resection of the whole lamina, spinous process, medial part of the facet joint, and flavum ligament seems to play an important role in the occurrence or progress of instability. The author also suggested that in the presence of adequate indications, partial laminectomy should be selected to avoid the occurrence or progress of instability at the operated level.[22] Muscular atrophy is another complication of open laminectomy which may occur due to the damage of the paraspinal muscles.[23] In addition, asymmetry of the paraspinal muscle may also influence the occurrence of postoperative instability,[24] preservation of paraspinal muscle and decrease the incidence of muscular injury—the most important factors associated with a positive long-term clinical outcome.[25,26] Marjan Alimi et al. have also reported that minimally invasive laminectomy has a lower rate of postoperative instability, reduced blood loss, and a shorter hospital stay than the conventional open laminectomy method.[27] In this case report, a minimally invasive surgery was performed to decompress and evacuate hematoma via paraspinal approach through tubular retractors. Through this alternative method, we are able to grind a hole on the skipped lamina with no damage to the zygapophysis. Moreover, through this approach, a smaller trauma of the paraspinal muscles was achieved owing to the tubular retractor system. Smaller intraoperative blood loss and shorter duration of postoperative pain are the other 2 advantages compared with standard laminectomy. In this case study, the intraoperative blood loss was about 50 mL and the

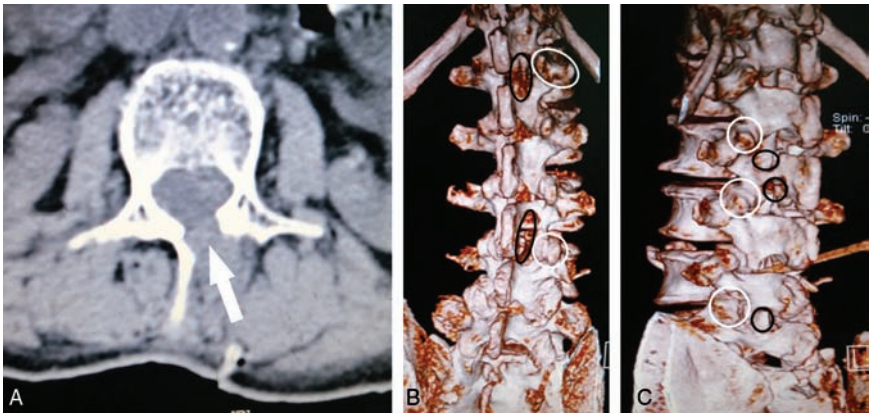

Figure 5. CT scan (A) and reconstructed 3-dimensional CT scan (B and C) of this patient 9 days postoperative. The surgical channel of operation (white arrow and circle) and the zygapophysis joint integrity (black circle).

patient did not feel obvious wound pain 3 days after surgery. Additionally, postoperative MRI showed complete removal of the SSEH, the thecal sac and spinal cord expansion and recovery (Figs. 4A–C). We report that through this surgical approach, surgery channels were small and the zygapophysis maintained integrity in postoperative CT scan and reconstructed 3-dimensional CT scan (Fig. 5A–C). During the 3-month follow-up, spinal instability was found and the walking capability of the patient was observed to be gradually improving.

4. Conclusion

The minimally invasive surgery used to decompress the spinal canal and evacuate the hematoma via paraspinal approach through tubular retractors may be an effective alternative method to treat SSEH. This method has the advantages of causing minimal damage to the spinal bony structure, only a minor trauma to the paraspinal muscles, and a relatively short postoperative pain recovery time.

Acknowledgments

We would like to thank Clarity Manuscript Consultant LLC (Indianapolis) for their language-editing service.

References

[1] Holtas S, Heiling M, Lonntoft M. Spontaneous spinal epidural hematoma: findings at MR imaging and clinical correlation. Radiology 1996;199:409–13.
[2] Shin JJ, Kuh SU, Cho YE. Surgical management of spontaneous spinal epidural hematoma. Eur Spine J 2006;15:998–1004.
[3] Alexiadou-Rudolf C, Ernestus RL, Nanassis K, et al. Acute nontraumatic spinal epidural hematomas. An important differential diagnosis in spinal emergencies. Spine 1998;23:1810–3.
[4] Liao CC, Hsieh PC, Lin TK, et al. Surgical treatment of spontaneous spinal epidural hematoma: a 5-year experience. J Neurosurg Spine 2009;11:480–6.
[5] Buyukkaya R, Aydin O, Hakyemez B, et al. Rapid spontaneous recovery after development of a spinal epidural hematoma: a case report. Am J Emerg Med 2014;32:291e291-291.e293.
[6] Ciancio RAM, Drain O, Rillardon L, et al. Acute spontaneous spinal epidural hematoma: an important differential diagnosis in patients under clopidogrel therapy. Spine J 2008;8:544–7.
[7] Thiele RH, Hage ZA, Surdell DL, et al. Spontaneous spinal epidural hematoma of unknown etiology: case report and literature review. Neurocrit Care 2008;9:242–6.
[8] Park J, Lee JB, Park JY, et al. Spinal cord infarction after decompressive laminectomy for spontaneous spinal epidural hematoma–case report. Neurol Med Chir (Tokyo) 2007;47:325–7.
[9] Gopalkrishnan CV, Dhakoji A, Nair S. Spontaneous cervical epidural hematoma of idiopathic etiology: case report and review of literature. J Spinal Cord Med 2012;35:113–7.
[10] Miyagi Y, Miyazono M, Kamikaseda K. Spinal epidural vascular malformation presenting in association with a spontaneously resolved acute epidural hematoma. J Neurosurg 1998;88:909–11.
[11] Lawton MT, Porter RW, Heiserman JE, et al. Surgical management of spinal epidural hematoma-relationship between surgical timing and neurological outcome. J Neurosurg 1995;83:1–7.
[12] Patel H, Boaz JC, Phillips JP, et al. Spontaneous spinal epidural hematoma in children. Pediatr Neurol 1998;19:302–7.
[13] Beatty RM, Winston KR. Spontaneous cervical epidural hematoma. A consideration of etiology. J Neurosurg 1984;61:143–8.
[14] Groen RJM, Ponssen H. The spontaneous spinal epidural hematoma. a study of the etiology. J Neurologic Sci 1990;98:121–38.
[15] Elgafy H, Bransford RJ, Chapman JR. Epidural hematoma associated with occult fracture in ankylosing spondylitis patient—a case report and review of the literature. J Spinal Disord Tech 2011;24:469–73.
[16] Kreppel D, Antoniadis G, Seeling W. Spinal hematoma-a literature survey with meta-analysis of 613 patients. Neurosurg Rev 2003;26:1–49.
[17] Matsumura A, Namikawa T, Hashimoto R, et al. Clinical management for spontaneous spinal epidural hematoma: diagnosis and treatment. Spine J 2008;8:534–7.
[18] Liao CC, Lee ST, Hsu WC, et al. Experience in the surgical management of spontaneous spinal epidural hematoma. J Neurosurg 2004;100: 38–45.
[19] Woon CYL, Peng BCW, Chen JLT. Spontaneous spinal epidural haematomas and the prognostic implications of interval to surgical decompression- a report of two cases. J Orthopaed Surg 2009;17:216–9.
[20] Foo D, Rossier AB. Preoperative neurological status in predicting surgical outcome of spinal epidural hematomas. Surg Neurol 1981;15: 389–401.
[21] Hentschel SJ, Woolfenden AR, Fairholm DJ. Resolution of spontaneous spinal epidural hematoma without surgery-report of two cases. Spine 2001;26:525–7.
[22] Iida Y, Kataoka O, Sho T, et al. Postoperative lumbar spinal instability occurring or progressing secondary to laminectomy. Spine 1990;15:1186–9.
[23] Watanabe K, Matsumoto M, Ikegami T, et al. Reduced postoperative wound pain after lumbar spinous process-splitting laminectomy for lumbar canal stenosis: a randomized controlled study. J Neurosurg Spine 2011;14:51–8.
[24] Yang JC, Kim SG, Kim TW, et al. Analysis of factors contributing to postoperative spinal instability after lumbar decompression for spinal stenosis. Korean J Spine 2013;10:149–54.
[25] Jong HS, Gun P, Chang IJ, et al. Radiological analysis of symptomatic complications after bilateral laminotomy for lumbar spinal stenosis. Korean J Spine 2012;9:18–23.
[26] Seung JH, Young BK, Yang SK, et al. Postoperative changes in paraspinal muscle volume: comparison between paramedian interfascial and midline approaches for lumbar fusion. J Korean Med Sci 2007;22: 646–51.
[27] Alimi M, Hofstetter CP, Pyo SY, et al. Minimally invasive laminectomy for lumbar spinal stenosis in patients with and without preoperative spondylolisthesis-clinical outcome and reoperation rates. J Neurosurg Spine 2015;22:339–52.