Surgical Neurology International

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Case Report

Migration of traumatic intracranial subdural hematoma to lumbar spine causing radiculopathy

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Received: 24 January 13 Accepted: 06 May 13 Published: 19 June 13

This article may be cited as:
Li CH, Yew AY, Lu DC. Migration of traumatic intracranial subdural hematoma to lumbar spine causing radiculopathy. Surg Neurol Int 2013;4:81.
Available FREE in open access from: http://www.surgicalneurologyint.com/text.asp?2013/4/1/81/113647

Abstract

Background: There have been rare reports of intracranial subdural hematoma (SDH) that migrated into the spine. All previous cases have been surgically managed and in this case report, we describe the first case of conservatively managed spinal hematoma secondary to migratory intracranial SDH.

Case Description: A 26-year-old male presented with a left tentorial SDH after blunt trauma. He was conservatively managed and discharged home. He presented 8 days later with worsening lower back pain that was found to be secondary to a spinal SDH.

Conclusion: Spinal hematomas can be a serious sequelae of migrated intracranial hematomas. Tentorial and other caudally located intracranial hematomas may be more prone to this phenomenon.

Key Words: Radiculopathy, subdural hematoma, trauma

Access this article online

Website: www.surgicalneurologyint.com

DOI: 10.4103/2152-7806.113647

Quick Response Code:

INTRODUCTION

While the incidence of intracranial subdural hematoma (SDH) is estimated to constitute up to 11% of head trauma cases,[3,14,20] blunt trauma only rarely induces acute spinal SDHs. Spinal hematomas are more commonly secondary to iatrogenic etiologies (30%).[12] Cases have been reported of spinal SDH secondary to attempted lumbar punctures or as a rare complication of spinal anesthesia.[5,6,16,19] However, spinal SDH are very uncommon and in a retrospective review of 613 patients with spinal hematomas, only 25 (<5%) of the patients had a SDH compared with 455 patients (74.2%) with epidural hematomas.[12] A rare mechanism for formation of spinal SDHs is migration of an intracranial SDH.[2,15] Here, we present a case of radiculopathic pain secondary to a migrated intracranial SDH following blunt head trauma.

CASE REPORT

History and examination

Our patient is a 26-year-old male who presented with a left tentorial SDH following blunt trauma to the head. The patient was brought to our Emergency department alert and oriented to person, place, and date and had no focal neurologic deficits. Noncontrast computed tomography (CT) imaging of the head revealed a 4 mm left tentorial SDH and 2 mm of pneumocephalus [Figure 1]. Given the patient's nonfocal neurologic exam and the small size of hematoma, the SDH was managed conservatively, in accordance with accepted traumatic brain injury guidelines.[3] The patient was discharged on postadmission day 2 following an uncomplicated hospital course. At the time of discharge patient did not exhibit any lower extremity symptoms.

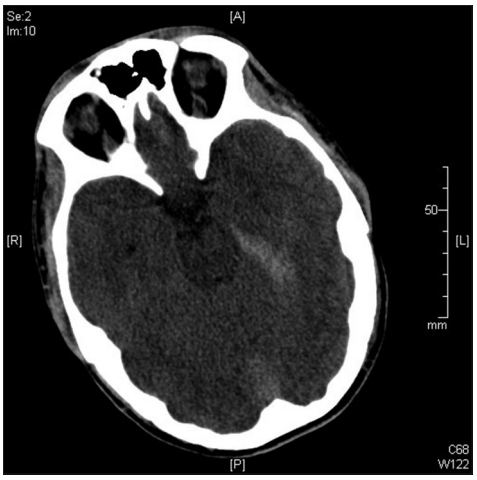

Figure 1: Noncontrast CT of the brain demonstrating a 4 mm left tentorial subdural hematoma

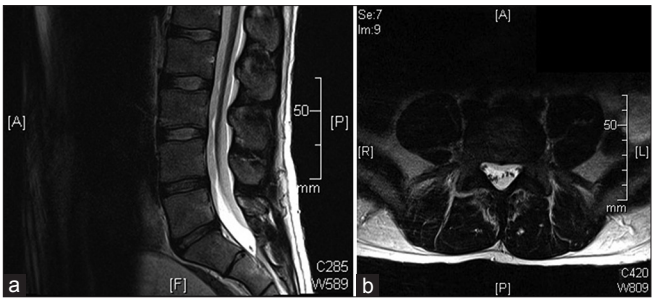

Figure 2: (a) Sagittal T2 weighted MRI of the lumbar spine demonstrating anterior and posterior subdural hematomas that extend from the thoracic levels down to the level of the L5-S1 disc space, (b) Axial T2 weighted MRI of the lumbar spine redemonstrating both anterior and posterior components. There is displacement of the cauda equina nerve roots secondary to mass effect

The patient returned on postinjury day 8 for follow up and discontinuation of his antiseizure prophylaxis. The patient noted that he was experiencing progressively worsening lower back pain that radiated into both lower extremities and had started one day after discharge. He also reported occasional ataxia and dizziness. He denied any changes in bowel or bladder function. The patient had 5/5 strength on confrontational motor testing in all lower extremity muscle groups.

The patient underwent contrast-enhanced magnetic resonance imaging (MRI) of the cervical, thoracic, and lumbar spine. Imaging of the cervical and thoracic spine revealed no abnormal enhancement and showed normal alignment. However, the MRI of the lumbar spine revealed a 6.84 mm SDH extending from the T4-T5 to the L5-S1 levels causing central displacement of the cauda equina. No cord deformity, compression, or edema was seen. There was a signal difference in the fluid of the thecal sac and moderate facet hypertrophy was observed at L4-L5 and L5-S1 [Figure 2]. Follow up MR angiography was negative for vascular malformations or spinal dural arteriovenous (AV) fistulas. Given that the patient had no focal neurologic deficits, the decision was made to manage the patient conservatively. At last follow up 3 months postinjury, the patient has had no further symptoms of radiculopathy and has no neurologic deficits on exam.

DISCUSSION

We have described a rare case of radiculopathy developing secondary to migration of an intracranial SDH to the lumbar spine. In addition to blunt trauma, spinal SDH has also been reported in cases of epidural injections,[16] in patients treated with anticoagulative agents[9,11] and in cases of AV malformations.[8] It has also known to occur spontaneously.[23] However, spinal SDH that resulted directly from the migration of a cranial SDH into the spinal subdural is exceedingly rare and has only been described rarely in the literature.

Bortolotti *et al.* were the first to suggest that spinal SDHs may develop secondary to migration of an intracranial SDH. They described a 23-year-old woman who initially had blunt head trauma with a left frontal convexity intracranial SDH that subsequently migrated to the tentorium with tentorial layering. The patient developed sciatica and back pain 4 days after her head injury and MRI of the lumbar spine performed 10 days post-injury revealed a lumbar SDH. Operative decompression was performed and the patient's symptoms resolved.

Shimada *et al.* reported a case of a 68-year-old male who presented with severe low back pain secondary to a spinal SDH 2 weeks following conservative management of an intracranial SDH from blunt head trauma.[21] The patient was taken for emergent surgical decompression and his symptoms resolved. A similar case[4] of 2-week onset of spinal SDH after intracranial SDH resolution was reported and treated with decompression. These authors recommend immediate surgical decompression for spinal SDH. In the most recently published case by Moscovici *et al.*, an 88-year-old male was diagnosed with spinal SDH 5 days after head trauma. Cauda equina syndrome subsequently developed and laminectomy was performed.[15]

The majority of spinal SDH secondary to migration of an intracranial SDH have required operative therapy. Ahn and Smith described the first case of a clivial SDH that presented simultaneously with cervical spinal SDH, which resolved with conservative treatment.[1] Hung *et al.* describes a 12-year-old boy who fell and struck his head and back.[10] The patient developed headache and left sided sciatica immediately postinjury. CT of the head demonstrated a left parietal convexity SDH that was managed conservatively. Given his persistent sciatica, spinal MRI was performed on postinjury day 3

and discovered a lumbar spinal SDH. This spinal SDH was managed nonoperatively as the patient exhibited rapid improvement in his radicular symptoms. Similarly, our patient was young and had a lumbar spinal SDH that was managed conservatively. However, it should be noted that in contrast to the previous two cases, which reported spinal pathology that presented via imaging or symptomatically immediately postinjury, our case presented in a delayed fashion. Our patient therefore represents the first example of delayed migratory spinal SDH that was successfully managed conservatively.

Moscovici *et al.*[15] and Bortolotti *et al.*[2] proposed that cerebrospinal fluid (CSF) influx into the subdural space can have a "dilutional effect" on the hematoma and can facilitate its migration into other parts of the spinal cord. They also hypothesized that hemorrhages can migrate into the cerebrospinal tract through the fourth ventricle. The authors noted that the hematoma may have resolved due to migration of the subdural blood into other parts of the spinal cord and subarachnoid tears may have allowed influx of CSF, causing the dilutional effect.

Hung *et al.* proposed that as a result of increased intracranial pressure, there is an increase in shearing forces between the dura and arachnoid layers in the spinal column.[10] This leads to an increased probability of rupture of the thin inner leaflet of the dura mater. Electron microscopy has shown that the cranial and spinal subdural cavities are connected, forming a passage for hematoma migration.[7,17,18] Wong *et al.*[22] described a 73-year-old experienced spontaneous resolution of an acute SDH that started in the tentorium and later spread into the posterior fossa. The authors made a similar observation in that redistribution of the hematoma from the tentorium hiatus into the posterior fossa and subsequently into the spinal canal can result in spontaneous resolution of the hematoma. The patient showed no symptoms of back pain and was successfully managed conservatively.

While no authors have systematically assessed the position of the intracranial SDH and its relationship to likelihood of subsequent migration, our review of the literature reveals that four of the six case reports[1,2,4,13,21,22] of migratory spinal SDH that reported the location of the intracranial SDH noted a tentorial or infratentorial position. One case reported a right frontal hematoma that could have migrated into the tentorial space.[4] Our patient similarly exhibited a tentorial SDH on initial presentation. We hypothesize that tentorial and infratentorial SDHs have a higher likelihood of migration to the spine due to its closer proximity.

While two authors[1,10] noted patients that had simultaneous discovery of their intracranial and spinal hematomas, the remaining majority of cases presented in a delayed fashion between 3 and 14 days postinjury. Our patient presented in this time window as well, with development of radicular symptoms in a delayed fashion 8 days postinjury. To our knowledge, our patient represented the first case report of spontaneous resolution of a delayed migratory spinal SDH. However, it should be emphasized that the majority of patients who presented with delayed spinal SDH secondary to intracranial went on to require operative management. This highlights the importance of prompt diagnosis of spinal symptoms in patients following intracranial injury.

CONCLUSION

Traumatic spinal SDH is a rare phenomenon and subsequent migration into the spinal canal has been described in only a handful of cases. We report a patient who developed radiculopathy and lower back pain following cranial SDH that was found to be the result of a migratory spinal SDH. Our case illustrates the variability of the phenomenon of a migratory hematoma and we hypothesize that migration may be related to the location of the initial intracranial hematoma.

ACKNOWLEDGEMENT

This research was made possible through the generosity of J. Yang & Family Foundation.

REFERENCES

1. Ahn ES, Smith ER. Acute clival and spinal subdural hematoma with spontaneous resolution: Clinical and radiographic correlation in support of a proposed pathophysiological mechanism. Case report. J Neurosurg 2005;103 (2 Suppl):175-9.
2. Bortolotti C, Wang H, Fraser K, Lanzino G. Subacute spinal subdural hematoma after spontaneous resolution of cranial subdural hematoma: Causal relationship or coincidence? Case report. J Neurosurg 2004;100 (4 Suppl Spine):372-4.
3. Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, *et al.* Surgical management of acute subdural hematomas. Neurosurgery 2006;58 (3 Suppl):S16-24.
4. Chen HJ, Liang CL, Lu K, Liliang PC, Tsai YD. Cauda equina syndrome caused by delayed traumatic spinal subdural haematoma. Injury 2001;32:505-7.
5. DeAngelis J. Hazards of subdural and epidural anesthesia during anticoagulant therapy: A case report and review. Anesth Analg 1972;51:676-9.
6. Dickman CA, Shedd SA, Spetzler RF, Shetter AG, Sonntag VK. Spinal epidural hematoma associated with epidural anesthesia: Complications of systemic heparinization in patients receiving peripheral vascular thrombolytic therapy. Anesthesiology 1990;72:947-50.
7. Haines DE, Harkey HL, al-Mefty O. The "subdural" space: A new look at an outdated concept. Neurosurgery 1993;32:111-20.
8. Han PP, Theodore N, Porter RW, Detwiler PW, Lawton MT, Spetzler RF. Subdural hematoma from a Type I spinal arteriovenous malformation. Case report. J Neurosurg 1999;90 (2 Suppl):255-7.
9. Hausmann, Kirsch E, Radu E, Mindermann TH, Gratzl O. Coagulopathy induced spinal intradural extramedullary haematoma: Report of three cases and review of the literature. Acta Neurochir (Wien) 2001;143:135-40.
10. Hung KS, Lui CC, Wang CH, Wang CJ, Howng SL. Traumatic spinal subdural hematoma with spontaneous resolution. Spine 2002;27:E534-8.
11. Jagatsinh Y. Cauda equina syndrome: A rare complication in intensive care. Indian J Orthop 2009;43:309-11.
12. Kreppel D, Antoniadis G, Seeling W. Spinal hematoma: A literature survey with meta-analysis of 613 patients. Neurosurg Rev 2003;26:1-49.
13. Leber KA, Pendl G, Kogler S, Kammerhuber F, Ebner F. Simultaneous spinal

and intracranial chronic subdural hematoma. Case illustration. J Neurosurg 1997;87:644.
14. Massaro F, Lanotte M, Faccani G, Triolo C. One hundred and twenty-seven cases of acute subdural haematoma operated on. Correlation between CT scan findings and outcome. Acta Neurochir (Wien) 1996;138:185-91.
15. Moscovici S, Paldor I, Ramirez de-Noriega F, Itshayek E, Shoshan Y, Spektor S, *et al*. Do cranial subdural hematomas migrate to the lumbar spine? J Clin Neurosci 2011;18:563-5.
16. Moussallem CD, El-Yahchouchi CA, Charbel AC, Nohra G. Late spinal subdural haematoma after spinal anaesthesia for total hip replacement. J Bone Joint Surg Br 2009;91:1531-2.
17. Nicholas D, Weller R. The fine anatomy of the human spinal meninges. A light and scanning electron microscopy study. J Neurosurg 1988;69:276-82.
18. Reina MA, De Leon Casasola O, Lopez A, De Andres JA, Mora M, Fernandez A. The origin of the spinal subdural space: Ultrastructure findings. Anesth Analg 2002;94:991-5.
19. Seow K, Drummond KJ. Subdural spinal haematoma after spinal anaesthesia in a patient taking aspirin. J Clin Neurosci 2011;18:1713-5.
20. Servadei F, Nasi MT, Giuliani G, Cremonini AM, Cenni P, Zappi D, *et al*. CT prognostic factors in acute subdural haematomas: The value of the 'worst' CT scan. Br J Neurosurg 2000;14:110-6.
21. Shimada Y, Sato K, Abe E, Miyakoshi N, Tsutsumi Y. Spinal subdural hematoma. Skeletal Radiol 1996;25:477-80.
22. Wong ST, Yuen MK, Fok KF, Yuen SC, Yam KY, Fong D. Redistribution of hematoma to spinal subdural space as a mechanism for the rapid spontaneous resolution of posttraumatic intracranial acute subdural hematoma: Case report. Surg Neurol 2009;71:99-102.
23. Yang NR, Kim SJ, Cho YJ, Cho do S. Spontaneous resolution of nontraumatic acute spinal subdural hematoma. J Korean Neurosurg Soc 2011;50:268-70.