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Pathogenesis of Spinal Subdural Hematoma Based on Histopathological Findings: A Case Report

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Abstract

Spinal subdural hematoma is a rare condition whereas intracranial chronic subdural hematoma is well-recognized and documented in clinical settings. Despite various theories that have been proposed, the exact pathogenesis of spinal subdural hematoma remains to be elucidated. Herein, we report a rare case of spinal subdural hematoma with a co-existing intracranial chronic subdural hematoma and deduce its etiology using histopathological findings. A 76-year-old Japanese man with slight hemiparesis due to intracranial chronic subdural hematoma underwent burr hole surgery with successful drainage of the hematoma. The patient could not walk unassisted after the surgery. Spinal magnetic resonance imaging was performed, revealing a subdural hematoma extending from the T11 to S2 levels. The patient subsequently underwent the evacuation of spinal subdural hematoma 12 days after initial surgery. Intraoperatively, we identified a dark liquefied hematoma with capsule formation. Histological examination of the resected capsule revealed loose fibrovascular tissues comprising capillaries, collagen fibers, a small number of macrophages, and hemosiderin deposits. These findings were very similar to the pathology of intracranial chronic subdural hematoma so the pathogenesis of spinal subdural hematoma in this case was considered identical to that of subacute or chronic subdural hematoma. The patient was transferred to a rehabilitation hospital, and 4 months after the lumbar surgery, no recurrence was observed in the magnetic resonance imaging examination. Subacute or chronic spinal subdural hematoma may result from various pathogeneses that clinicians should consider for correct diagnosis and appropriate management. Our case provides important insights into the pathogenesis of subacute or chronic spinal subdural hematoma.

Keywords: chronic subdural hematoma, histopathology, pathogenesis, spinal subdural hematoma

Introduction

Subdural hematoma (SDH) often occurs in the intracranial space, whereas its occurrence along the spinal column in the extracranial space is rare.[1,2] When presenting symptoms such as lower back pain, weakness in the lower extremities, or bowel and bladder dysfunction that persist for >2 weeks, it is classified as chronic SDH.[3] In the spine, the frequency of occurrence of chronic SDH is considerably lower than that of acute or subacute SDH.[4]

Previous studies have proposed various factors contributing to spinal SDH (sSDH), including a history of trauma, lumbar puncture, bleeding diathesis, use of antithrombotics, or vascular malformation, although instances of spontaneous occurrence have been noted.[5,6] In addition, various cases of concomitant intracranial chronic SDH (iCSDH) and sSDH have been reported. However, the exact etiology and pathogenesis of sSDH remain unknown. In addition, few reports have considered histopathological findings in investigating the pathogenesis of sSDH.

Herein, we present a case of concomitant iCSDH and sSDH and discuss the pathogenesis of the disease based

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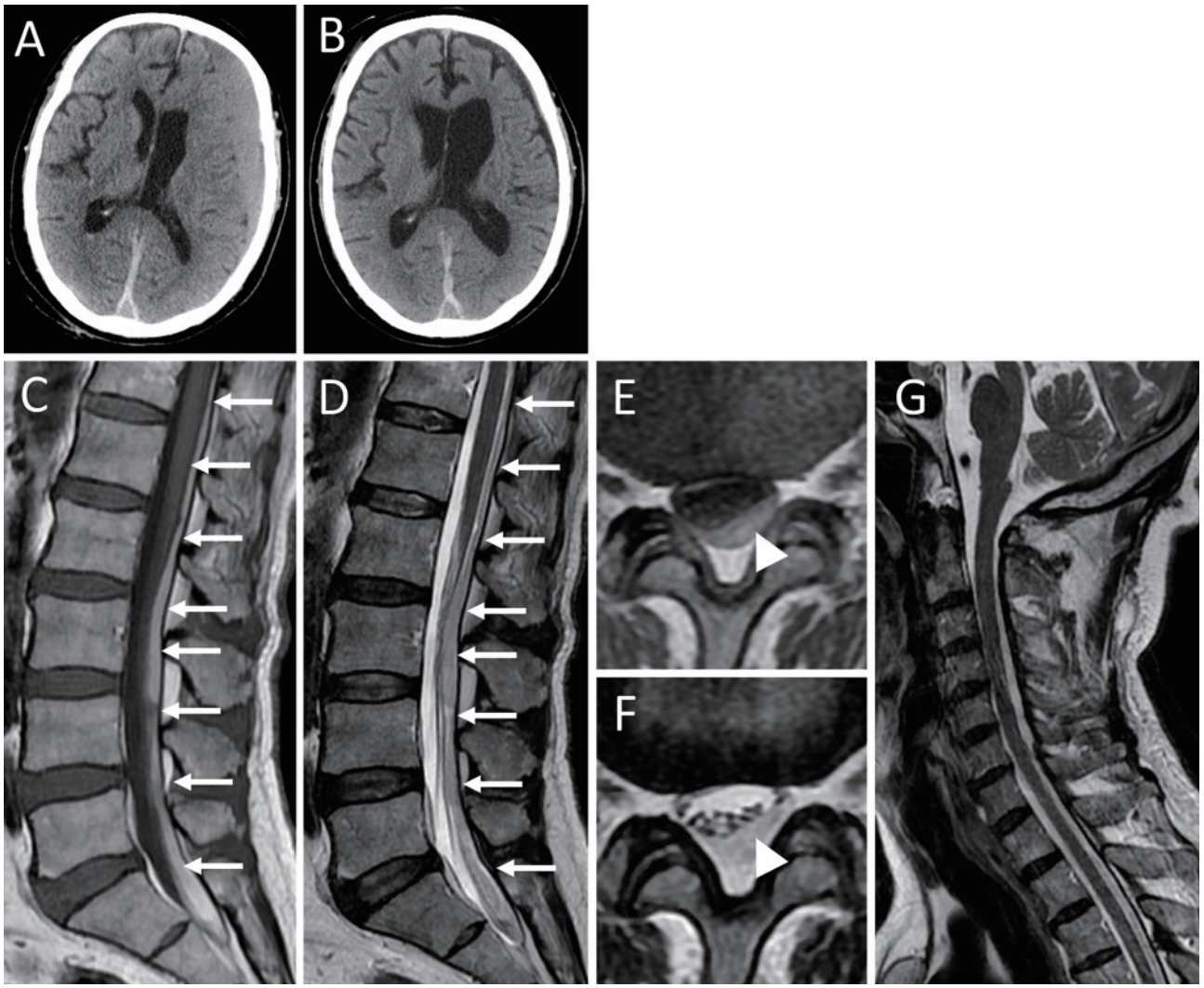

Fig. 1 CT images of iCSDH and preoperative MRI of the spine. (A) Preoperative CT image shows left iCSDH with moderate midline shift. (B) Postoperative CT image shows that iCSDH is well evacuated. Both T1 (C and E) and T2 (D and F) hyperintense subdural hematomas (arrowheads in E and F) extend from the T11 to S2 levels (arrows in C and D), compressing the cauda equina. (G) Cervical MRI displays no sign of hematoma.
CT: computed tomography; iCSDH: intracranial chronic subdural hematoma; MRI: magnetic resonance imaging

on the findings of histopathological examination.

Case Report

A 76-year-old man, who had suffered from head trauma 1 month ago, was transferred to our hospital after falling and sustaining head trauma again. He had a history of brain infarction a few decades prior to admission and had undergone burr hole surgery for a right iCSDH 4 years earlier. He was not on anticoagulant or antiplatelet medication. He presented with slight right hemiparesis (manual muscle test [MMT] 4/5). Head computed tomography (CT) revealed a left frontoparietal iCSDH with a mild midline shift (Fig. 1A). He underwent burr hole surgery upon admission to our hospital, resulting in successful drainage of the hematoma (Fig. 1B). However, he remained unable to stand or walk unassisted 1 week after surgery. The MMT score for his legs was approximately 3-4, and his patellar tendon reflex was attenuated. The Babinski reflex was negative. Spinal magnetic resonance imaging (MRI) revealed a SDH extending from the T11 to S2 levels (Fig. 1C-F), with no evidence of hematoma at the cervical level (Fig. 1G).

To improve his neurological condition, he underwent an L2-4 hemilaminectomy for hematoma evacuation 12 days after initial surgery. When the dura mater was opened, a dark-brownish liquid hematoma covered by the membrane resembling that of an iCSDH was observed (Fig. 2A, B). Subsequently, the hematoma was washed and evacuated through saline irrigation using a drainage tube. Clear cere-

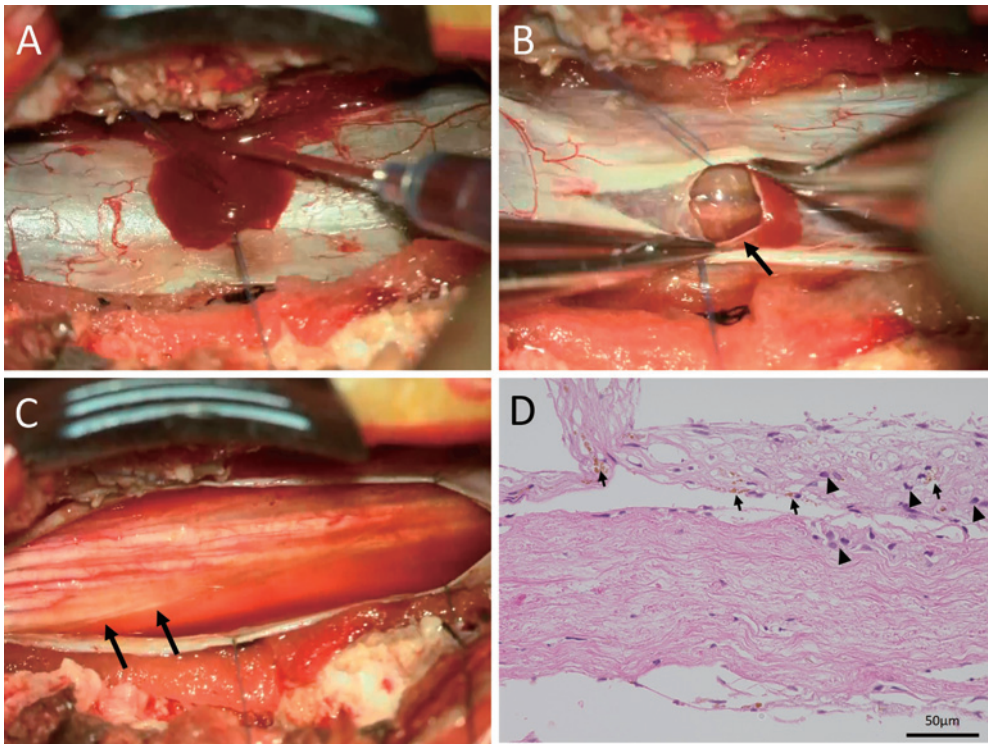

Fig. 2 Intraoperative photographs of spinal subdural hematoma and the histopathological images of the outer membrane of the spinal subdural hematoma. (A) Liquefied dark-colored hematoma is excreted after incision of the dura. (B) After wide opening of the dura, an outer membrane (arrow) of the hematoma is observed. (C) After rinsing the subdural hematoma, an intact arachnoid membrane (arrows) and clear cerebrospinal fluid are observed. (D) The membrane comprises fibroblasts, collagen fibers, capillaries, macrophages (arrowheads), and hemosiderin (arrows). The sections are stained by hematoxylin and eosin (original magnification, ×400).

brospinal fluid (CSF) was observed through the intact, normal arachnoid membrane, with no evidence of hemorrhage in the subarachnoid space (Fig. 2C). Histopathological examination revealed that the membrane of the hematoma contained fibroblasts, collagen fibers, capillaries, macrophages, and hemosiderin (Fig. 2D). Neither hemorrhage nor apparent cell infiltration was observed in the membrane.

The patient was transferred to a rehabilitation hospital 9 days after lumbar surgery. Because of dementia, he lacked the willingness to rehabilitate, resulting in him being forced to use a wheelchair to move at discharge. Postoperative spinal MRI at 4 months showed no recurrence of SDH (Fig. 3A-D).

Discussion

In this study, we report a rare case of concomitant iCSDH and sSDH and the histopathological examination of its membrane. To the best of our knowledge, this is the first report to provide histopathological evidence of concomitant sSDH and iCSDH.

Intracranial SDH occurs spontaneously or is triggered predominantly by trauma,[7-9] followed by intracranial hypotension.[10] Intracerebral hemorrhage,[11,12] ruptured cerebral aneurysm,[13,14] and cerebral vascular malformations[15-18] are occasionally accompanied by SDH, when bleeding extends to the subdural space. Other less common causes include vasculopathy[19] and neoplasm.[20-22] One of the mechanisms of developing iCSDH is as follows: after the development of an acute SDH, the absorption of blood subsequently begins with the breakdown of erythrocytes and other cellular components. Next, collagen synthesis is induced, and fibroblasts spread over the inner surface of the dura to form a thick outer membrane.[23] Subsequently, a thinner inner membrane forms, leading to the complete encapsulation of the clot. In general, this process spans approximately 2 weeks.

The exact pathogenesis of sSDH remains unknown (Fig. 4), with various theories proposed. One theory suggests that disruption of bridging veins in the spine, caused by direct trauma or lumbar puncture, leads to hematoma formation, similar to intracranial hematomas. The spinal subdural space, known to be less vascularized,[24,25] may contribute to the lower frequency of sSDH compared to iCSDH. However, bridging veins exist not only in intracranial structures but also in the spine,[26] suggesting that disruption of these veins can also cause a sSDH. A previous

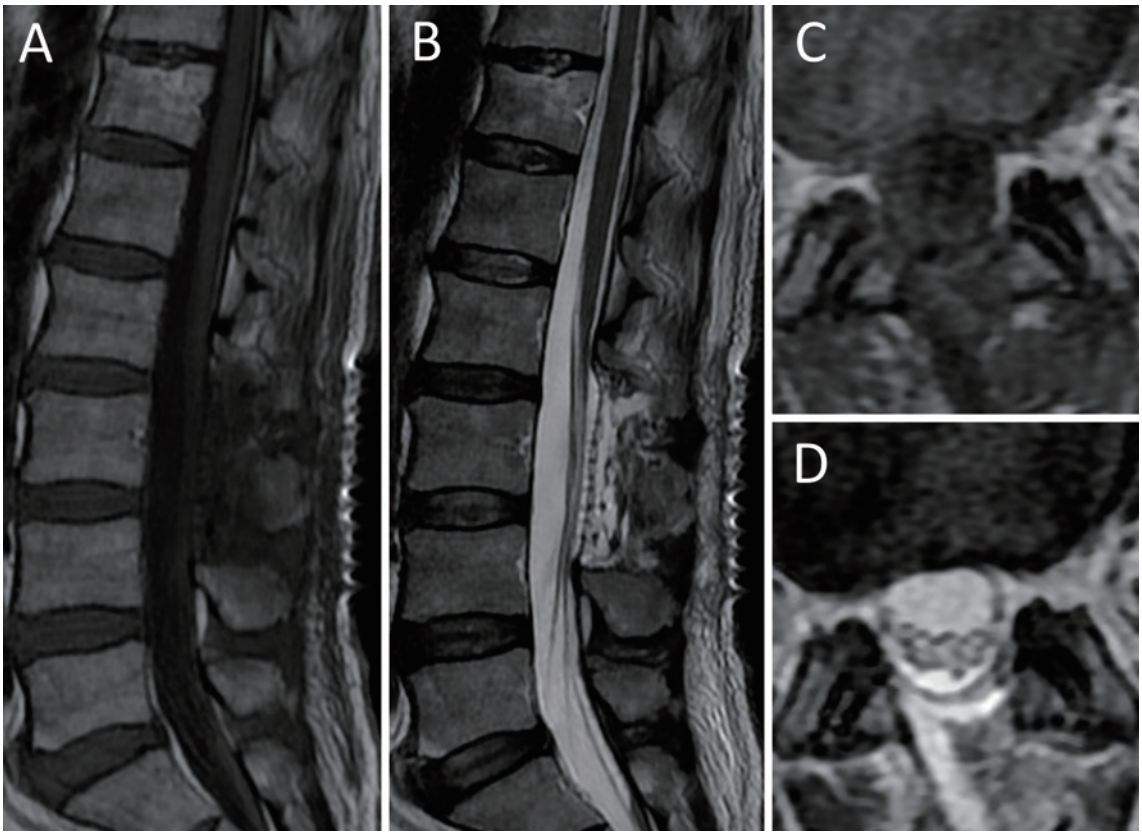

Fig. 3 Postoperative magnetic resonance imaging of sSDH. After 4 months, no recurrence of sSDH is observed (T1: A and C, T2: B and D).
sSDH: spinal subdural hematoma

prospective study reported a significant association between the direct impact on the lumbar area and sSDH in patients with iCSDH, suggesting double trauma to the head and lumbar regions as a major cause of concomitant intracranial and sSDH.[27] Another theory proposes that an iCSDH might migrate to the spinal area, supported by the less vascularized spinal subdural space and the subdural compartment between the cranium and spinal space proved to be connected.[28] Certain cases have shown hematomas in the posterior fossa or all through the spine,[29,30] with similar signal intensity on MRI, suggesting a common origin. However, our patient did not have a hematoma at the cervical level, as revealed through MRI. Furthermore, the previous prospective study found only 2 (1.2%) of 168 cases of iCSDH with concomitant sSDH on MRI.[27] A third theory suggests a different mechanism for the formation of sSDH from that of iCSDH.[31] In this theory, sSDH occurs due to a rapid increase in intravascular pressure of the intraspinal segments of lateral spinal vessels, caused by increased abdominal and thoracic pressure. This sudden increase in the intravascular pressure is not neutralized by a simultaneous increase in CSF pressure because of the shielding effect of the spinal column and its ligaments. Consequently, hemorrhage in the subdural space may occur. Such subdural accumulation of blood would be subject to the same set of forces that govern the formation of intracranial fluid collections. This theory is supported by a case report of sSDH co-existing with a ligament flavum hematoma, indicating a mechanism consistent with this theory.[32] Finally, some researchers have stated that the subarachnoid hemorrhage is thought to extend into the subdural space.[33,34] These potential pathogenesis may be more likely to be induced by the use of antithrombotics or because of bleeding diathesis.

During iCSDH formation, the outer and inner membranes that surround the hematoma form subsequently, as described previously.[35] According to histopathological examination, the outer membrane contains various components, such as neovasculature and fibrous tissue. Exudation from these neovasculatures contributes to hematoma enlargement. Previous studies have categorized histopathological features of the outer membrane into the following four subtypes: noninflammatory (I), inflammatory (II), hemorrhagic inflammatory (III), and scar inflammatory (IV).[36] The noninflammatory (I) type comprises immature fibroblasts, collagen fibers, sparse cellular infiltration, and neocapillaries. The inflammatory (II) type comprises a single sheet of immature connective tissue with marked cellu-

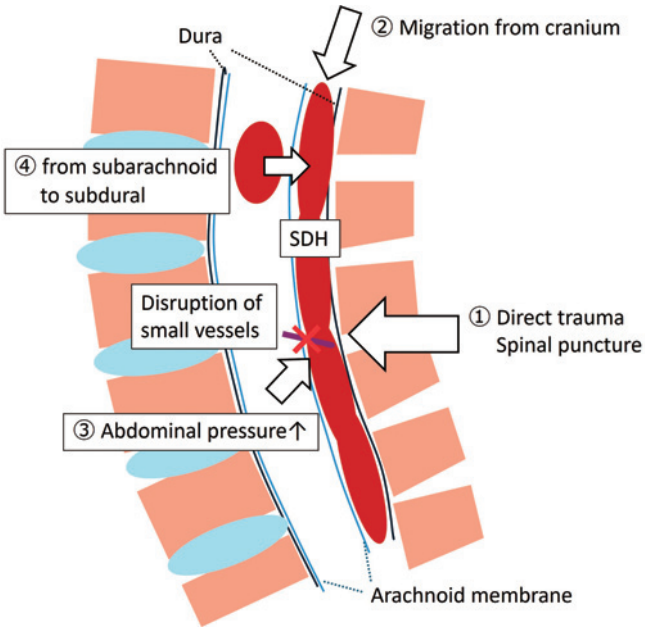

Fig. 4 Possible pathogenesis of sSDH. The first theory is direct trauma or spinal puncture to the lumbar area. This may cause disruption of small vessels such as bridging veins, resulting in sSDH. The second theory is that the intracranial chronic subdural hematoma may migrate to the spine. The third theory is an increase in abdominal or thoracic pressure. This may also cause disruption of small vessels. The fourth theory is that the hematoma extends from the subarachnoid space to the subdural space. Black lines in the spinal canal represent the dura, while blue lines represent the arachnoid membrane.
sSDH: spinal subdural hematoma

lar infiltration and vascularization. The hemorrhagic inflammatory (III) type consists of a few sheets of connective tissue with cellular infiltration and moderately sized capillaries. The scar inflammatory (IV) type comprises inflammatory cell infiltration, neovascularization, and hemorrhage. In addition, the outer membrane is thought to change from type I to IV in order;[37] thus, the histopathological appearance of the membrane is useful in estimating the duration of chronic SDH.[38]

In our patient, the histopathological features of the outer membrane of the sSDH were considered to be similar to those of type I iCSDH because of the slight cellular infiltration. Therefore, we suggest that in this case, the mechanism for sSDH formation may be trauma to the lumbar area, similar to that for iCSDH resulting from head trauma, rather than migration, although there is the possibility that the hematoma formed or expanded after the intracranial decompression. Moreover, based on the histopathology, this case was in the early phase of hematoma formation. In fact, the intensity of hematoma in MRI was considered to be in the subacute to chronic phase. Various studies have also reported that the outer membrane of the sSDH was observed during surgery,[4,39] similar to our case. On the other hand, some studies did not find an outer membrane,[40,41] which may indicate that not enough time had elapsed for capsular formation or that the hematoma migrated from the cranial space.

In conclusion, we demonstrated that sSDH may have various potential pathogeneses, which can be elucidated through histopathological examination as in our case. This finding provides important insights for clinicians to enable them to correctly diagnose and appropriately manage sSDH.

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Informed Consent

We obtained informed consent for publication from the patient.

Conflicts of Interest Disclosure

All authors have no conflict of interest.

References

1) de Beer MH, Eysink Smeets MM, Koppen H. Spontaneous spinal subdural hematoma. Neurologist. 2017;22(1):34-9. doi: 10.1097/NRL.0000000000000100
2) Benyaich Z, Laghmari M, Lmejjati M, et al. Acute lumbar spinal subdural hematoma inducing paraplegia after lumbar spinal manipulation: case report and literature review. World Neurosurg. 2019;128:182-5. doi: 10.1016/j.wneu.2019.05.002
3) Abla AA, Oh MY. Spinal chronic subdural hematoma. Neurosurg Clin N Am. 2000;11(3):465-71. doi: 10.1016/S1042-3680(18)30109-8
4) Tillich M, Kammerhuber F, Reittner P, et al. Chronic spinal subdural haematoma associated with intracranial subdural haematoma: CT and MRI. Neuroradiology. 1999;41(2):137-9. doi: 10.1007/s002340050719
5) Oichi Y, Toda H, Yamagishi K, et al. Multiple spinal chronic subdural hematomas associated with thoracic hematomyelia: a case report and literature review. World Neurosurg. 2019;131:95-103. doi: 10.1016/j.wneu.2019.07.209
6) Ichinose D, Tochigi S, Tanaka T, et al. Concomitant intracranial and lumbar chronic subdural hematoma treated by fluoroscopic guided lumbar puncture: a case report and literature review. Neurol Med Chir (Tokyo). 2018;58(4):178-84. doi: 10.2176/nmc.cr.2017-0177
7) Bullock MR, Chesnut R, Ghajar J, et al. Surgical management of acute subdural hematomas. Neurosurgery. 2006;58(3 suppl):S16-24; discussion Si-iv.
8) Besenski N. Traumatic injuries: imaging of head injuries. Eur Radiol. 2002;12(6):1237-52. doi: 10.1007/s00330-002-1355-9

9) Morris NA, Merkler AE, Parker WE, et al. Adverse outcomes after initial non-surgical management of subdural hematoma: a population-based study. Neurocrit Care. 2016;24(2):226-32. doi: 10.1007/s12028-015-0178-x
10) Beck J, Gralla J, Fung C, et al. Spinal cerebrospinal fluid leak as the cause of chronic subdural hematomas in nongeriatric patients. J Neurosurg. 2014;121(6):1380-7. doi: 10.3171/2014.6.JNS14550
11) Patel PV, FitzMaurice E, Nandigam RN, et al. Association of subdural hematoma with increased mortality in lobar intracerebral hemorrhage. Arch Neurol. 2009;66(1):79-84. doi: 10.1001/archneur.66.1.79
12) Vega RA, Valadka AB. Natural history of acute subdural hematoma. Neurosurg Clin N Am. 2017;28(2):247-55. doi: 10.1016/j.nec.2016.11.007
13) Barton E, Tudor J. Subdural haematoma in association with intracranial aneurysm. Neuroradiology. 1982;23(3):157-60. doi: 10.1007/BF00347560
14) Koerbel A, Ernemann U, Freudenstein D. Acute subdural haematoma without subarachnoid haemorrhage caused by rupture of an internal carotid artery bifurcation aneurysm: case report and review of literature. Br J Radiol. 2005;78(931):646-50. doi: 10.1259/bjr/60601877
15) Rengachary SS, Szymanski DC. Subdural hematomas of arterial origin. Neurosurgery. 1981;8(2):166-72. doi: 10.1227/00006123-198102000-00005
16) Li G, Zhang Y, Zhao J, et al. Isolated subdural hematoma secondary to Dural arteriovenous fistula: a case report and literature review. BMC Neurol. 2019;19(1):43. doi: 10.1186/s12883-019-1272-z
17) Schmitt AJ, Mitha AP, Germain R, et al. Subdural hematoma from a cavernous malformation. World Neurosurg. 2014;82(3-4):535.e1-3. doi: 10.1016/j.wneu.2013.01.018
18) Kieu HD, Le TD, Hoang TM. Acute spontaneous subdural hematoma secondary to ruptured arteriovenous malformation: a rare entity. Ann Med Surg (Lond). 2021;68:102613. doi: 10.1016/j.amsu.2021.102613
19) Alves OL, Gomes O. Cocaine-related acute subdural hematoma: an emergent cause of cerebrovascular accident. Acta Neurochir (Wien). 2000;142(7):819-21. doi: 10.1007/s007010070098
20) Laigle-Donadey F, Taillibert S, Mokhtari K, et al. Dural metastases. J Neurooncol. 2005;75(1):57-61. doi: 10.1007/s11060-004-8098-1
21) Nayak L, Abrey LE, Iwamoto FM. Intracranial dural metastases. Cancer. 2009;115(9):1947-53. doi: 10.1002/cncr.24203
22) Boukas A, Sunderland GJ, Ross N. Prostate dural metastasis presenting as chronic subdural hematoma. A case report and review of the literature. Surg Neurol Int. 2015;6:30. doi: 10.4103/2152-7806.151713
23) Sajanti J, Majamaa K. High concentrations of procollagen propeptides in chronic subdural haematoma and effusion. J Neurol Neurosurg Psychiatry. 2003;74(4):522-4. doi: 10.1136/jnnp.74.4.522
24) Brandt RA. Chronic spinal subdural haematoma. Surg Neurol. 1980;13(2):121-3.
25) Khosla VK, Kak VK, Mathuriya SN. Chronic spinal subdural hematomas. Report of two cases. J Neurosurg. 1985;63(4):636-9. doi: 10.3171/jns.1985.63.4.0636
26) Komiyama M. Spinal radiculomedullary vein and bridging vein. Interv Neuroradiol. 2020;26(6):703-5. doi: 10.1177/15910199209628282
27) Kokubo R, Kim K, Mishina M, et al. Prospective assessment of concomitant lumbar and chronic subdural hematoma: is migration from the intracranial space involved in their manifestation? J Neurosurg Spine. 2014;20(2):157-63. doi: 10.3171/2013.10.SPINE13346
28) Stone RS, Jones OW. Encephalography: a review of 113 cases, and a Report of postmortem Studies on the Injection of Air. Radiology. 1933;21(5):411-9. doi: 10.1148/21.5.411
29) Jain V, Singh J, Sharma R. Spontaneous concomitant cranial and spinal subdural haematomas with spontaneous resolution. Singapore Med J. 2008;49(2):e53-8.
30) Lecouvet FE, Annet L, Duprez TP, et al. Uncommon magnetic resonance imaging observation of lumbar subdural hematoma with cranial origin. J Comput Assist Tomogr. 2003;27(4):530-3. doi: 10.1097/00004728-200307000-00013
31) Rader JP. Chronic subdural hematoma of the spinal cord: report of a case. N Engl J Med. 1955;253(9):374-6. doi: 10.1056/NEJM195509012530906
32) Shimada Y, Kasukawa Y, Miyakoshi N, et al. Chronic subdural hematoma coexisting with ligamentum flavum hematoma in the lumbar spine: a case report. Tohoku J Exp Med. 2006;210(1):83-9. doi: 10.1620/tjem.210.83
33) Banach S. [Spontaneous subdural hematoma of the spinal cord]. Neurol Neurochir Pol. 1970;4(2):243-6.
34) Smith RA. Spinal subdural hematoma, neurilemmoma, and acute transverse myelopathy. Surg Neurol. 1985;23(4):367-70. doi: 10.1016/0090-3019(85)90208-3
35) Sahyouni R, Goshtasbi K, Mahmoodi A, et al. Chronic subdural hematoma: a perspective on subdural membranes and dementia. World Neurosurg. 2017;108:954-8. doi: 10.1016/j.wneu.2017.09.063
36) Nagahori T, Nishijima M, Takaku A. [Histological study of the outer membrane of chronic subdural hematoma: possible mechanism for expansion of hematoma cavity]. No Shinkei Geka. 1993;21(8):697-701.
37) Sato S, Suzuki J. Ultrastructural observations of the capsule of chronic subdural hematoma in various clinical stages. J Neurosurg. 1975;43(5):569-78. doi: 10.3171/jns.1975.43.5.0569
38) Bokka S, Trivedi A. Histopathological study of the outer membrane of the dura mater in chronic sub dural hematoma: its clinical and radiological correlation. Asian J Neurosurg. 2016;11(1):34-8. doi: 10.4103/1793-5482.154979
39) Leber KA, Pendl G, Kogler S, et al. Simultaneous spinal and intracranial chronic subdural hematoma. Case illustration. J Neurosurg. 1997;87(4):644. doi: 10.3171/jns.1997.87.4.0644
40) Kanamaru H, Kanamaru K, Araki T, et al. Simultaneous spinal and intracranial chronic subdural hematoma cured by craniotomy and laminectomy: a video case report. Case Rep Neurol. 2016;8(1):72-7. doi: 10.1159/000445709
41) Matsumoto H, Matsumoto S, Yoshida Y. Concomitant intracranial chronic subdural hematoma and spinal subdural hematoma: a case report and literature review. World Neurosurg. 2016;90:706.e1-9. doi: 10.1016/j.wneu.2016.03.020

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