A Rare Case of Organized Spinal Chronic Subdural Hematoma Associated with Unknown Fever

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Abstract

Non-traumatic spinal subdural hematoma is a rare condition that may present as a chronic subdural hematoma in rare instances and subsequently progress to an organized form. Here, we report the case of a 68-year-old woman with a history of atrial fibrillation who was receiving non-vitamin K antagonist oral anticoagulant therapy.

Case Description: The patient presented with sudden-onset left-sided hemiparesis and was transferred to a local hospital. There was no evidence of ischaemic stroke on brain magnetic resonance imaging (MRI); however, intravenous tissue plasminogen activator therapy was initiated 1 h 30 min after symptom onset. The patient's symptoms persisted despite the treatment. Subsequently, she developed persistent high-grade fever (>39°C) of unknown origin despite extensive investigations and empirical antibiotic therapy. Spinal MRI revealed an intradural lesion compressing the spinal cord at the C6–T2 level on day 22, which was a suspected spinal subdural abscess. Consequently, she was referred to our hospital, where imaging findings led to the diagnosis of a subdural hematoma. Conservative management was initially commenced; however, the persistent unexplained fever and absent neurological improvement necessitated surgical intervention on day 57. Intraoperatively, we observed thickened arachnoid membranes and organized yellowish hematomas in the subdural space, confirming the diagnosis of organized spinal chronic subdural hematoma. Postoperatively, the left-sided hemiparesis gradually improved. Therefore, she was transferred for rehabilitation 21 days post-surgery.

Conclusion: In this case report, we highlighted spinal subdural hematomas, which are rare and occasionally progress to an organized chronic form. Surgical treatment is crucial to reverse neurological deficits.

Key words: fever, hematoma, neurological deficits, organized hematoma, spinal chronic subdural hematoma

Introduction

Spinal hematomas can occur in the extradural, subdural, or subarachnoid spaces, with epidural hematomas being the most prevalent. Spinal subdural hematomas (SDHs) are rare and often associated with anticoagulation therapy, blood dyscrasias, lumbar puncture, trauma, spinal anaesthesia, or vascular malformations.[1] Clinically, spinal SDHs have manifestations indicative of spinal cord compression, including motor, sensory, and autonomic dysfunction.[2] Magnetic resonance imaging (MRI) is preferred over computed tomography (CT) for the diagnosis of spinal SDH.[3] MRI has a high sensitivity for classifying the bleed type and assessing the craniocaudal extension of the hematoma. The management of non-traumatic spontaneous spinal SDH has not been definitively established, which may be due to the rarity of this condition.[4]

Spinal SDH persisting for more than two weeks is a chronic spinal SDH (CSDH), an extremely rare condition.[1,5] Unlike intracranial CSDHs, which are relatively common, spinal CSDHs are uncommon and typically associated with a history of trauma, bleeding diatheses, spinal anaesthesia, or antithrombotic therapy. Nonetheless, spontaneous occurrences have been reported.[5]

There are no reports of spinal CSDH progressing to an organized state and being associated with fever of unknown origin despite documentation of such cases. In this study, we present a rare case of organized spinal CSDH accompanied by persistent fever that resolved following surgical removal of the organized hematoma.

Case Report

A 68-year-old woman presented with a sudden onset of paralysis in the left upper and lower extremities. She previously had atrial fibrillation, which was managed with apixaban, a non-vitamin K antagonist oral anticoagulant (NOAC). There was no

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history of major spinal column trauma before the onset of paresis. Her vital signs were within normal, and she was conscious, oriented, and free of cervical pain on admission. Neurological examination revealed partial paralysis, with Manual Muscle Test (MMT) scores of 3 for the left upper extremity and 2 for the left lower extremity. The initial head MRI showed no acute ischaemic changes on diffusion-weighted imaging (DWI). However, the patient's clinical presentation was consistent with acute-phase cerebral infarction despite the absence of hyperintense areas. Tissue plasminogen activator (tPA) was administered within 1 h of symptom onset; however, no improvement was observed. Subsequently, the patient developed a fever, prompting the initiation of cefepime on the second day of hospitalisation; however, the fever persisted. Blood cultures were obtained on day 5, and the antibiotic regimen was switched to meropenem. Full-body CT and cervical spine MRI were performed to investigate persistent fever and left-sided paralysis on day 22. There were no significant findings on CT, but cervical spine MRI revealed a subdural mass in the lower cervical and upper thoracic regions. The patient was referred to our department on day 34 for further evaluation and management following a clinical suspicion of spinal subdural abscess.

At the time of admission (day 34), the patient's vital signs included body temperature (38.4°C), blood pressure (104/75 mmHg), and pulse rate (95 bpm). Neurological examination revealed hemiparesis of the left upper and lower extremities with MMT scores of 3 for both, indicating a slight improvement. Furthermore, laboratory tests revealed the following: white blood cell count (6,200/μL), haemoglobin (9.7 g/dL), platelet count (259,000/μL), prothrombin time-international normalised ratio (1.78), activated partial thromboplastin time (36.9 s), and mildly increased C-reactive protein (3.00 mg/dL). The other biochemical parameters were within normal limits. Cervical and thoracic spine MRI revealed a subdural mass extending from C6 to T2, compressing the spinal cord. The mass exhibited hyperintense signals on T1-weighted imaging, hypointense signals on T2-weighted imaging (T2WI), partial enhancement on Gd T1-enhanced and no diffusion restriction on DWI (Figure 1). Our presumptive diagnosis was CSDH instead of a spinal subdural abscess. Therefore, we chose conservative treatment in anticipation of spontaneous resolution of the hematoma.

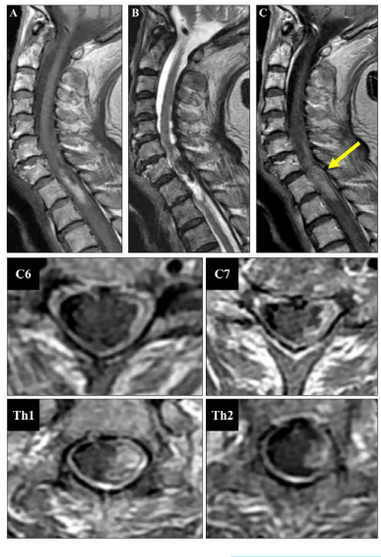

Figure 1: Sagittal magnetic resonance imaging (MRI) of the cervicothoracic spine show hyper- and hypointense masses in the subdural space on T1 (A) and T2 (B) images. Gd T1-enhanced MRI (C) showing partial enhancement (arrow). Axial MRI of the cervicothoracic spine at the C6 to Th2 levels shows a hyperintense mass in the left subdural space on the Gd-T1-weighted image.

However, the patient suffered intermittent fever during the follow-up period. Extensive investigations, including blood and urine cultures, influenza testing, and β-D-glucan assays, were negative. Repeat full-body CT identified no infectious sources except in the cervical mass region. Follow-up MRI showed a slight decrease in the subdural mass, with an intramedullary high-intensity area of the spinal cord on T2WI (Figure 2), indicating a persistent mass effect on the spinal cord. Consequently, we planned surgical intervention for the following reasons. First, owing to the mass effect, the patient's motor strength remained at an MMT score of 4 following rehabilitation. We did not expect a spontaneous decrease in the mass effect with an organized SDH. Second, the patient's fever persisted. If a subdural mass is an abscess, its evacuation can result in a reduced fever. Finally, the patient underwent surgical intervention on day 91.

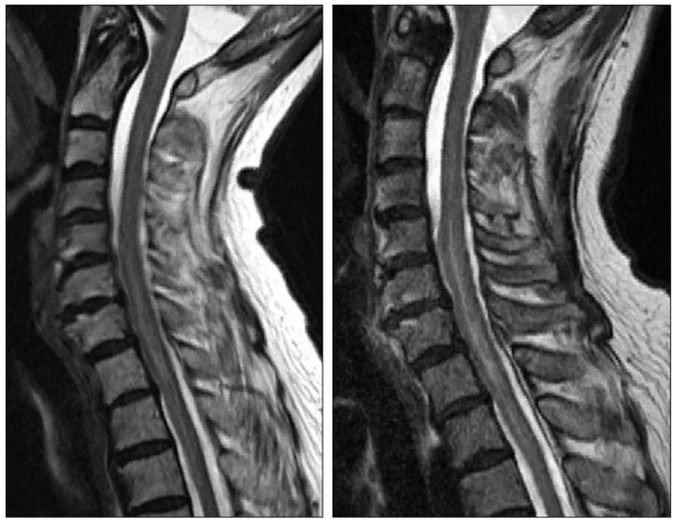

Figure 2: Sagittal magnetic resonance imaging of the cervicothoracic spine showing an increased intramedullary signal in the spinal cord (left: day 63, right: day 88).

The patient underwent C6–T2 laminectomy. The dura mater was intact intraoperatively, and a median dura incision revealed a white, cloudy arachnoid membrane. We observed a firm yellow tissue on the left side upon incision of the arachnoid membrane. Furthermore, the arachnoid membrane and adherent tissues were carefully separated from the spinal cord and nerve roots to achieve adequate decompression (Figure 3). There was no evidence of vascular malformation, tumours, or other pathological abnormalities. Histopathology showed the presence of fibrous connective tissue with hemosiderin deposits, which led to a diagnosis of organized CSDH. Tissue cultures were negative. Postoperative MRI confirmed subtotal hematoma removal. The postoperative course was uneventful.

The patient's fever resolved postoperatively (Figure 4). She gradually recovered from the paraparesis and was able to ambulate using a cane. She was discharged to a rehabilitation facility on the 21st postoperative day (112 days after the onset of symptoms).

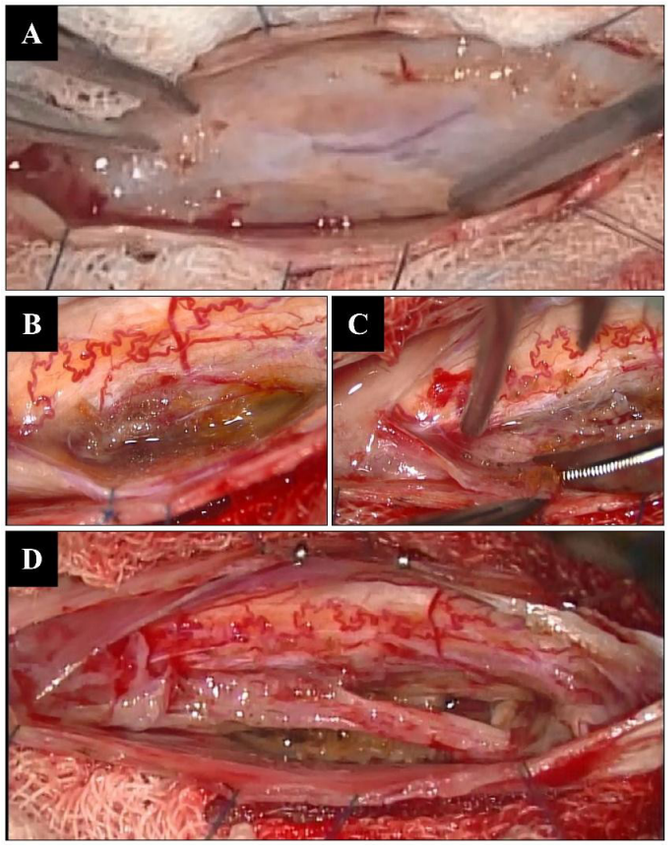

Figure 3: Laminectomy findings. A white-cloudy arachnoid membrane in the dura (A). A firm yellow tissue was observed in the arachnoid membrane on the left side (B), cutting the thick arachnoid membrane and the yellow tissue (C). After removing the arachnoid membrane and yellow tissue, the left nerve root was observed (D).

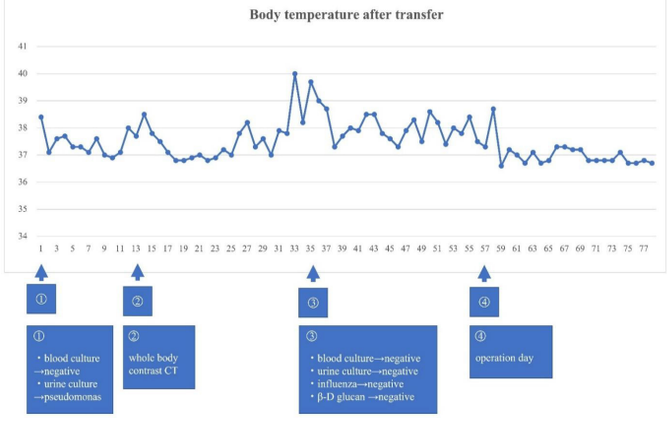

Figure 4: The patient's body temperature.

Discussion

In this study, we present a case of spinal CSDH that converted to an organized hematoma with persistent fever of unknown origin. This case involved a non-traumatic acute spinal SDH that was initially diagnosed as an acute cerebral infarction that was treated with tPA. Acute hemiparesis is a common early presentation of acute ischaemic stroke, and tPA administration within 4.5 h of stroke onset is a critical component of its treatment.[6] The initial diagnosis for a patient with hemiparesis without neck pain is difficult to associate with spinal disorders. Kim et al. reported that two of nine patients with cervical epidural hematoma who presented to the emergency department were misdiagnosed with acute stroke and received tPA. Cervical spine lesions require careful evaluation to avoid cerebral infarction misdiagnosis, particularly when considering tPA administration, as its use in cases of spinal epidural hematoma can lead to severe neurological deficits.[7] The tPA administration did not exacerbate the patient's symptoms; however, its fibrinolytic effect worsened the hematoma, thereby leading to a delay in hematoma regression. This subsequently progressed to an organized state in the chronic phase.

The pathophysiological mechanisms underlying spontaneous idiopathic spinal SDHs remain poorly understood. Domenicucci et al. reviewed 106 cases of non-traumatic spinal SDHs. They identified abnormal blood coagulation or use of anticoagulant therapy as the cause of bleeding in 54% of cases, medically induced lumbar puncture in 33%, and idiopathic causes in 14%.[8] Several cases of non-traumatic spinal SDH related to NOACs have been reported.[9-11] In this case, there was no history of trauma; however, the patient's use of NOACs may have been a contributing factor.

Organized CSDH is defined as a CSDH with a thick membrane and multiple septations, forming an encapsulated area with solid consistency.[12] Unlike typical CSDH, it may develop over a longer time course, which generally develops over at least three weeks following head trauma. However, its exact pathogenesis and mechanisms remain unclear.[13-15] Shrestha et al. suggested that an ongoing chronic inflammatory process contributes to the formation of an organized CSDH membrane comprising neovascularisation, fibroblasts, and granulation tissue.[16]

Organized spinal CSDH is an extremely rare condition, and only four of such cases, characterised by thick fibrotic tissue within the dura rather than a liquid-like component, have been reported to date.[5,17,18] Siddiqi et al. described a case of a 76-year-old woman who underwent surgical treatment for CSDH with syringomyelia of the lower thoracic spine following a 14-year history of progressive myelopathy. Intraoperative findings revealed a yellowish-brown, thickened membrane composed of hypocellular, loose fibrovascular tissue containing capillaries and spindle cells. Histopathology analysis confirmed the presence of hypocellular fibrovascular tissue, capillaries, spindle cells, collagen, and iron deposits.[17] Subsequently, Virender et al. described two cases of spinal CSDH in male patients aged 40–45 years. Both patients presented with progressive paralysis symptoms and hematomas localised within the spinal dura mater.

Surgical intervention through laminectomy revealed thickened arachnoid membranes, consistent with arachnoiditis, alongside organized hematomas.[18] The precise mechanisms underlying hematoma organization were not identified in these reports; however, the intraoperative findings consistently demonstrated thickened and inflamed arachnoid membranes, suggesting an inflammatory response, with haemorrhage as a potential contributing factor.[17,18] We observed similar intraoperative findings in this present case, indicating that inflammatory processes following haematopoiesis are significant in the organization of spinal CSDHs.

The most unique feature of this present case was persistent fever without an infectious origin. No study has described the direct relationship between acute or chronic spinal SDHs and fever without infectious complications. Sporadic reports have documented haemorrhage within the spinal dura mater to be associated with fever caused by infections such as dengue fever and hantavirus.[19,20] This background is associated with dengue fever infection, involving thrombocytopenia, impaired platelet functions in aggregation and release, blood coagulation disturbances, immune complex formation, and the production of cytokines that damage small blood vessels.[19,21,22] However, in this present case, there was no history of travel, and these infections were not reported in Japan, making an infectious aetiology unlikely.

Non-infectious fever associated with spinal SDH has rarely been documented. Zeynep et al. reported the case of a 64-year-old man who developed spinal SDH and subarachnoid haemorrhage following a lumbar puncture. The patient exhibited persistent fever without evidence of infectious complications, which resolved spontaneously.[23] The authors did not discuss the cause of the fever. Patients with subarachnoid haemorrhage due to ruptured intracranial aneurysm often develop non-infectious fever within three days of onset.[24] In the present case, the patient probably had acute spinal SDH with associated subarachnoid haemorrhage. The hematoma persisted for an extended period. These clinical causes can contribute to non-infectious fever.

The association between spinal cord injury (SCI) and fever has been well documented.[25-28] Fever in post-traumatic SCI is often attributed to thermoregulatory abnormalities caused by autonomic dysfunction and occurs in 38.5–71.7% of cases.[25] Common causes of fever in patients with SCI include infections, deep vein thrombosis, pulmonary embolism, and drug-induced fever, with urinary tract infections being the most frequent aetiology.[22] Furthermore, a fever of unknown origin, categorised as a neurogenic fever, can occur. The incidence of fever of unknown origin ranges from 2.6–27.8%, with mean and median incidences of 8.0% and 4.7%.[25,26] Patients with paraplegia after SCI will likely develop fever due to impaired thermoregulation.[26] However, the precise mechanisms underlying post-SCI fever have not been fully elucidated.[27] Neurogenic fever is more common in cervical and thoracic cord injuries and is more frequently observed in complete than in incomplete injuries.[25] According to Colachis and Otis, the duration of fever in patients with SCI during acute care is significantly longer (4.9 days) than during rehabilitation (1.9 days).[28] No studies have demonstrated a correlation between SCI and the persistence of fever. In the present case, SCI caused by a hematoma might have affected the neurogenic temperature control system, leading to prolonged fever.

Conclusion

Non-traumatic acute spinal SDHs are rare entities that can progress to chronic and organized states. Organized spinal CSDHs can present with prolonged fever and neurological symptoms. Therefore, surgical intervention can be an effective treatment strategy when symptoms of spinal cord compression, including fever and neurological deficits, occur following hematoma organization.

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