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Concurrent Intracranial and Lumbar Chronic Subdural Hematoma Presenting with Severe Lumbar Radiculopathy: A Case Report

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Background: Concurrent intracranial and spinal chronic subdural hematomas are rare and may be difficult to diagnose. Intracranial CSDH usually presents with headache or neurological deficits, whereas spinal SDH may cause back pain and radiculopathy. **Case Presentation:** A 57-year-old man presented with 10 days of severe low back pain radiating to both lower limbs, associated with weakness and abnormal gait. Lumbar MRI showed a subdural hematoma extending from T12/L1 to S1/S2 with cauda equina crowding. Brain MRI revealed bilateral chronic intracranial subdural hematomas without significant mass effect or midline shift. The patient was managed conservatively, and his symptoms resolved completely within 2 weeks. **Conclusion:** Severe lumbar radiculopathy may be the initial presentation of concurrent intracranial and spinal CSDH. Brain and spine imaging should be considered in older patients with unexplained spinal symptoms, especially after minor trauma or anticoagulant use.

Key Words: Chronic Subdural Hematoma, Concurrent, Lumbar Radiculopathy, Spinal Subdural Hematoma

Introduction

Subdural hematoma (SDH) involves blood accumulation in the potential space between the dura and arachnoid mater.[1] Intracranial SDH is a common presentation, particularly in elderly individuals due to age-related brain atrophy stretching vulnerable bridging veins, even after minor or unremembered trauma.[1] In contrast, spinal subdural hematoma (SSDH) is a rare condition, accounting for only a small percentage of all spinal hematomas, as the spinal subdural space contains few major blood vessels.[2,3]

The simultaneous occurrence of both intracranial and spinal CSDH is an extreme clinical rarity, with a limited number of cases reported in the literature.[4,5] The etiology in these concurrent cases is often debated, but two primary mechanisms have been suggested: migration of the hematoma from the cranial to the spinal compartment via the anatomically continuous subdural space, or accidental simultaneous hemorrhage from separate traumatic events (double trauma).[5,7] Migration is often facilitated by gravity and changes in intracranial pressure.[5,8]

The challenge in diagnosing this condition lies in its variable clinical presentation. While intracranial CSDH typically manifests with non-specific symptoms like headache, confusion, or gait disturbances, spinal SDH presents with back pain and radicular symptoms.[9,10] The spinal symptoms can often be the prominent or initial complaint, potentially obscuring the underlying cranial pathology.[5] Given the potential for fatal outcomes if the intracranial component is missed, this case report aims to emphasize the importance of considering concurrent craniospinal SDH in patients presenting with severe, unexplained

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lumbar radiculopathy, especially in those with risk factors like advanced age, minor trauma history, or anticoagulant use.[4,11]

CASE PRESENTATION

A 57-year-old male presented to the neurosurgery department with complaints of headache and severe lower back pain.The patient reported a history of headache for several days, described as persistent and gradually progressive in nature. The headache was associated with nausea, without documented episodes of vomiting at presentation.

Simultaneously, the patient complained of severe lower back pain radiating to bilteral lower limbs associated with restricted walk and difficulty in change in position. The pain was severe enough to prompt neurosurgical consultation.There was no documented history of major trauma preceding the symptoms. No clear history of loss of consciousness, seizures, or focal neurological deficits was reported at the time of presentation. There was also no recorded history of bowel or bladder dysfunction, limb weakness, or sensory loss.

He has known medical history of type II Diabetes Mallitus under oral hypoglycemics but there was no any history of anticoagulant or antiplatelet use. On examination, the patient was conscious, alert, and well-oriented, with a Glasgow Coma Scale (GCS) score of 15. Pupillary examination revealed bilaterally equal pupils measuring approximately 3 mm in diameter, which were reactive to light.

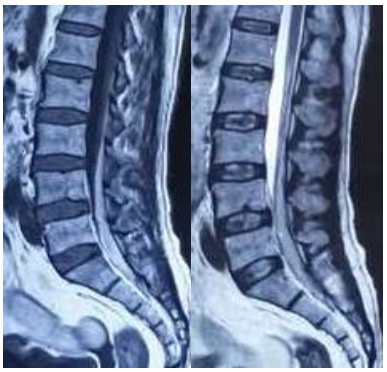

Figure 1: MRI sagittal T1 and T2 showed mixed intensity over L3-S1

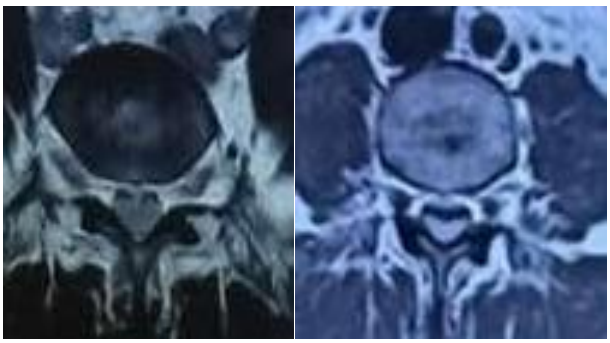

Figure 2: MRI Axial T1 and T2 showed mixed hyperintensity inside the thecal sac suggestive of different ages of blood

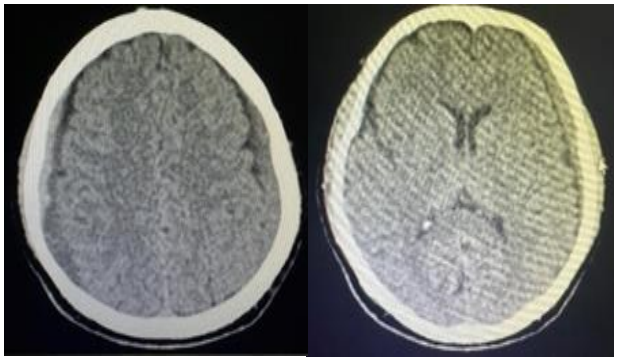

Figure 3: CT Head axial showing bilateral acute on chronic SDH

Motor examination demonstrated normal muscle strength (5/5) in all four limbs, with no evidence of focal neurological deficits but his straight leg test(SLRT) was severely compromised to 30 degrees bilateral. The patient's vital parameters were within normal limits, with a blood pressure of 120/90 mmHg, heart rate of 88 beats per minute, respiratory rate of 20 cycles per minute, and a body temperature of 98°F. Oxygen saturation was recorded at 94% on room air. His regualar blood tests with complete blood count and coagulation profile including PT/INR showed no abnormality.

Initially the patient was evaluated with magnetic resonance imaging(MRI) of the lumbo-sacral(LS) spine to rule out spinal pathology.His MRI of LS spine revealed mixed intensity lesion over L3-S2, significantly compressing the cauda equina, which was hyper intense in T1 weighted image and mixed to iso intense on T2 weighted image(Figure 1 and

2) suggestive of blood of different ages in subdural space.

A computer Tomography(CT) scan of the head was obtained as he had persistent headache despite of appropriated analgesic use. His CT head revealed bilateral acute on chronic subdural hematoma with minimal mass effect without midline shift (Figure3).

Management and Outcome

As the patient showed no significant signs of increased intracranial pressure, a conservative treatment approach was adopted, including intravenous dexamethasone, analgesics, and PPIs. Following significant clinical improvement, he was discharged after 6 days of hospital stay. At his two-week follow-up, repeat CT imaging of head demonstrated complete resolution of the subdural hematoma. Furthermore, the patient was asymptomatic for low back pain, exhibited no neurological deficits, and had a normal bilateral SLR of 90 degrees.

Discussion

The rapid and complete resolution of the patient's spinal symptoms following treatment of the intracranial lesion strongly supports the hypothesis that the spinal subdural hematoma (SSDH) resulted from caudal migration of blood from the cranial subdural space, rather than representing an independent primary spinal event.[3] This phenomenon is well-documented in the literature and is anatomically plausible due to the continuity of the craniospinal subdural space, which allows redistribution of blood under the influence of gravity, cerebrospinal fluid dynamics, and intracranial pressure changes.[6,7]

Several reports have demonstrated that intracranial chronic subdural hematoma (CSDH) may extend into the spinal compartment, particularly when pressure gradients favor downward movement.[3,10] In the present case, the absence of persistent or progressive spinal pathology after cranial intervention further reinforces the concept of a secondary spinal manifestation rather than a primary lesion. This aligns with prior observations that spinal hematomas often resolve spontaneously once intracranial pathology is adequately managed.[9]

A notable diagnostic challenge in this case was the initial predominance of severe lumbar symptoms, which masked the underlying intracranial pathology. Such presentations highlight an important clinical pitfall—over-reliance on the most prominent symptom may delay recognition of potentially life-threatening intracranial disease.[4] The later emergence of features suggestive of raised intracranial pressure (ICP), including disorientation, underscores the need for a broad and integrative diagnostic approach, particularly in elderly patients.

The so-called "double-trauma" hypothesis, wherein independent cranial and spinal hemorrhages occur due to separate traumatic events, appears unlikely in this scenario due to the absence of a clear recent history of trauma. However, the possibility of minor or forgotten trauma in the preceding weeks cannot be entirely excluded, especially in older individuals.[3] Nevertheless, the temporal sequence of symptom evolution and the resolution pattern strongly favor a unified pathophysiological mechanism originating intracranially.

Management strategies for concurrent cranial and spinal subdural hematomas remain guided by the severity of neurological impairment. Conservative treatment is generally appropriate for patients with mild or non-progressive spinal symptoms, as spontaneous resolution is frequently observed.[10] In contrast, surgical decompression of the spinal hematoma may be required in cases with significant or progressive neurological deficits. Importantly, when both cranial and spinal lesions coexist, priority should be given to treating the intracranial hematoma first, as failure to address elevated intracranial pressure may increase the risk of brain herniation.[11] Furthermore, spinal decompression prior to cranial stabilization may

exacerbate pressure gradients and worsen neurological outcomes.

This case also highlights the "great imitator" nature of CSDH, which can present with a wide spectrum of non-specific symptoms, often mimicking other neurological or musculoskeletal conditions.[8,9] Clinicians particularly those evaluating spinal complaints should maintain a high index of suspicion in elderly patients presenting with acute or severe back pain accompanied by subtle cognitive changes, headache, or a history of anticoagulant use or minor trauma.

In such contexts, early and comprehensive neuroimaging is essential. A low threshold for obtaining both cranial and spinal MRI can facilitate timely diagnosis and prevent delays that may result in significant morbidity or mortality. This case reinforces the importance of considering craniospinal continuity in subdural hematomas and adopting a holistic diagnostic strategy when clinical features appear discordant.

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