Missed Intracranial Subdural Hematoma in a Case of Spontaneous Subdural Spinal Hematoma: A Rare Case Report and Literature Review

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Learning Point of the Article:
The article highlights key radiological features to diagnose spinal SDH and the importance of imaging the brain in such cases.

Abstract

Introduction: Idiopathic spontaneous spinal subdural hematoma (ISSSDH) is uncommon in occurrence, and its association with concomitant intracranial subdural hematoma (ISDH) is very exceptional. Lack of recognition of ISDH in a patient with SSDH can lead to unanticipated events. We report a rare case of ISSSDH and unrecognized ISDH and treated surgically with a good outcome.

Case Report: A 71-year-old gentleman presented with features of spinal neurogenic claudication of 2 weeks duration and was diagnosed to have ISSSDH of the lumbar spine based on magnetic resonance imaging (MRI). In view of an impending cauda equina syndrome, he underwent an emergency decompression through a laminectomy, durotomy, and clot evacuation from L2 to L5. The next day, he developed sudden-onset hemiparesis and altered sensorium. The computed tomography (CT) scan of the brain demonstrated an ISDH, for which emergency burr hole evacuation was done. The patient improved rapidly after the surgery and regained his normal power, sensorium, and achieved comfortable ambulation within a week. Follow-up CT of the brain and MRI scan of the spine revealed adequate decompression. Since the CT features of ISDH were of acute on chronic nature, we presume that it had existed before the onset of spinal symptoms.

Conclusion: Through this very rare case, we highlight the importance of screening the brain pre-operatively in patients who present with subacute spontaneous spinal SDH.

Keywords: Sub-dural, hematoma, spinal, intracranial, spontaneous.

Introduction

Spinal subdural hematoma (SSDH) is very rare in occurrence and represents 4.1% of total spinal hematoma when compared with epidural (75%), and subarachnoid spinal hematoma (15.7%) [1]. Idiopathic Spontaneous SSDH (ISSSDH) is an infrequent phenomenon considering the absence of identifiable risk factors (trauma, spinal injection, anticoagulant, and coagulopathy) in these patients [2]. The coexistent ISSSDH with intracranial SDH (ISDH) is very exceptional, and only seven cases (Table 1) have been described [3, 4, 5, 6, 7, 8, 9]. We report the clinical and radiological features of a rare case of ISSSDH who developed ISDH a day after spine surgery and present a detailed review of the literature.

Case Report

A 71-year-old gentleman presented to us with complaints of bilateral lower limb radicular pain, more on the right side, with associated non-dermatomal patchy numbness in both lower limbs and minimal back pain. Symptoms were spontaneous in onset without a history of antecedent trauma, coagulopathy, or anticoagulant medication. It progressed over the 3 weeks, leading to incapacitating (visual analog scale [VAS] Score 9) pain in the lower limbs and difficulty in walking over the last 24 hours. He did not have other symptoms such as headache, nausea,
On clinical examination, the patient was fully conscious and oriented (Glasgow coma scale [GCS] 15/15). Gait was nonassessable due to severe radiculopathy, and straight leg raising test was restricted bilaterally. There was no local tenderness over the spine or paraspinal muscle spasm. He had mild weakness in the foot plantar and dorsiflexors (3/5 grade) with bilaterally diminished ankle jerk, without any sensory deficit and bladder-bowel involvement.

Radiological features

The patient was further evaluated with magnetic resonance imaging (MRI) lumbosacral spine with whole spine sagittal screening (Fig. 1), which was suggestive of subacute subdural hematoma from T12 to S2 level [10] (Fig. 1). Preservation of epidural fat without dural displacement or “Cap sign” (Fig. 1e) and incomplete “Inverted Mercedes Benz sign” (Fig. 1d) was typical of the subdural location of the hematoma [11]. Blood
investigations, including a complete coagulation profile, were within normal limits.

**Surgical treatment**

In view of the incapacitating pain and weakness in the distal muscles, the patient underwent emergency L1-L5 decompression and hematoma evacuation (Fig. 2). After hematoma evacuation, the dura was closed with 5-0 Prolene. There was no cerebrospinal fluid (CSF) leak, and the dural discoloration reversed to white at the end of the procedure (Fig. 2c). Post-operatively, the patient had stable vital and normal higher mental function without a sensory-motor deficit.

On the next day of surgery (18 hours later), the patient rapidly became drowsy and developed incoherent speech, nausea, and vomiting. On examination, he was partially conscious and drowsy and developed incoherent speech, nausea, and vomiting. He had dilated right pupil with normal reaction to light in both the pupils and left hemiparesis. All the symptoms developed and worsened rapidly over 2 h. An emergency computed tomography (CT) brain (Fig. 3) showed acute on chronic right fronto-parietal ISDH with a midline shift of 8–9 mm to the left side. The patient underwent emergency right frontal and parietal burr hole decompression and subdural hematoma evacuation. The patient was ventilated electively for a day and on weaning showed improved mentation (GCS 15/15). His neurology improved (4/5 Grade) gradually over the next 2 days, and he started to walk with support from the 6th post-operative day. Follow-up MRI (Fig. 4a), and CT scan (Fig. 4b) showed significant resolution of ISDH and SSDH, with reversal of midline shift, and a restituted CSF flow in the spinal canal. The patient was discharged on the 10th day after surgery and had complete resolution of symptoms and normal ambulatory capacity.

**Discussion**

Spinal hematoma, irrespective of its location, can be a catastrophe if not diagnosed and treated expeditiously. SSDH refers to a collection of blood between the layers of dura and arachnoid. It is commonly described after a traumatic event like spine injury, or spinal anesthesia (traumatic SSDH) or can develop after non-traumatic situations such as coagulopathy, anticoagulant medications, intra-spinal tumours, and arteriovenous (AV) malformations (spontaneous SSDH) [1, 10]. In the absence of these initiating factors, it is described as idiopathic SSSDH [2]. Although there are several case reports on SSSDH, the combination of idiopathic SSDH with intra-cranial SDH is very rare (Table 1).

SSDH usually presents between the 4th and 6th decade without sex predilection [1]. Spontaneous SSDH has been shown to involve multiple segments and areas of the spine from almost the entire spine to a single segment. However, it most commonly involves the thoracolumbar region, followed by the cervical spine [12]. However, in patients with concomitant SSDH and ISDH, it has been mostly observed in the lumbar-sacral region, as in our case [13].

Clinically, SSDH can present with myriad symptoms and signs. It commonly presents with back pain, unilateral or bilateral radiculopathy, with varying degrees of a sensory-motor deficit. The neurological deficit may vary from completely normal

### Table 1: Summary of reported cases of ISSSDH with concomitant ISDH

| Author               | Year  | Age/sex | Comorbidities | Symptoms at presentation | Clinical Course | Neurology | Treatment of Intracranial SDH | Treatment of SSDH | Recovery |
|----------------------|-------|---------|---------------|--------------------------|----------------|-----------|-------------------------------|-------------------|----------|
| Morihige et al.      | 2007  | 54 year/f | Headache, lumbago | Simultaneously diagnosed to have SSDH and brain SDH. | Suboccipital fossa to S2 | Normal | Burr hole drainage | Lumbar puncture | Good     |
| Lee et al.           | 2007  | 60 year/f | Hypertension  | Initially diagnosed to have chronic cranial SDH with back pain and lower limb pain on 3rd post-operative day. | L4-S1 | Normal | Burr Hole Cranietomy | Conservative initially but underwent lambection and hematoma evacuation after 13 days of cranial surgery. | Good     |
| Yang et al.          | 2009  | 35 year/f | No           | Simultaneously diagnosed to have SSDH and brain SDH. | L3-S1 | Paraparesis | Burr hole and hematoma evacuation | Lumbarectomy and hematoma evacuation | Good     |
| Nagashima et al.     | 2010  | 66 year/f | Rheumatoid, arthritis, hypertension | Initially diagnosed to have SSDH, two days after admission patient developed disorientation, behaviour problem, and diagnosed with intracranial SDH | L1-S1 | Both Psaas 3/5, Quadriceps 4/4, Hamstring 4/5, altered sensation below L1 | Burr hole hematoma evacuation | SSDH evacuation after trial of conservative management | Good     |
| Jibu et al.          | 2012  | 73 year/f | No           | Initially diagnosed to have SSDH with brain SDH as an incidental finding on brain screening | L3-S2 | Normal | Cranietomy with hematoma evacuation after one month of conservative management | Conservative | Good     |
| Lin et al.           | 2014  | 70 year/m | Hypertension, metastatic non-small cell lung cancer | Initially diagnosed to have SSDH, consequently done CT showed Brain SDH. | L5-S1 | Normal | Subdural drain followed by craniotomy after one month, for acute on chronic SDH | Conservative | Good     |
| Cui et al.           | 2015  | 45 year/f | No           | Initially diagnosed with SSDH followed by intracranial SDH two days after spine surgery. | L4-S3 | Lower limb paresis, sensory disturbance in the saddle region | Burr hole hematoma evacuation | Conservative | Surgery  |
| Present case         | 2020  | 71 year/f | No           | Initially diagnosed to have SSDH followed by intracranial SDH on the next day of spine surgery. | T12-S1 | Bilateral L4, L5, S1-3/5 | Burr hole and hematoma evacuation | L1-L5 lumbarectomy and hematoma evacuation | Good     |

ISSSDH: Idiopathic spontaneous spinal subdural hematoma; ISDH: Intracranial subdural hematoma
neurology to complete paraplegia [1, 12, 14]. In patients of ISSDH with ISDH, symptoms at presentation can vary greatly. Patients reported by Cui et al. [3], Jibu et al. [4], and Nagashima et al. [8] had initial symptoms related to the spine only, followed by the subsequent appearance of symptoms of ISDH. However, in the case reported by Lee et al. [5], the patient had no symptoms related to the spine at presentation but developed low back pain with lower limb radiculopathy on the third post-operative day of cranial surgery. Neurological deficit was reported in cases of Cui et al. [3], Nagashima et al. [8], and Yang et al. [9] Our patient had symptoms associated with the spine only at presentation and developed headache, nausea, incoherent speech, and hemiparesis on the next day of spine surgery.

The radiological diagnosis of SSDH is the most crucial step in the management of this pathology. MRI is the modality of choice for diagnosis, not only to differentiate subdural from an epidural hematoma but also best depicts the extent, location of hematoma in relation to cord or cauda equina, and can reveal underlying pathology. SSDH and epidural spinal hematoma can be differentiated based on MRI findings listed in Table 2 [1, 10, 11, 15, 16]. MRI is also important to diagnose the age of hematoma because the signal intensity of hematoma on MRI changes with time. It depends on the state of haemoglobin within hematoma and integrity of red blood cells [10, 15] (Table 3).

The patho-mechanism of concomitant spinal and intracranial SDH is yet to be clearly defined. Few theories have been postulated, which include bleeding caused by increased shearing force between spinal subdural and subarachnoid spaces caused by raised intracranial pressure [17] or redistribution and migration of ISDH to the dependent lumbar or sacral spine [18]. Cui et al. [3] and Morishinge et al. [7] queried the redistribution hypothesis as chronic ISDH has an outer and inner membrane, and the mechanism of movement of hematoma out of these membranes is questionable. Cui et al. [19] noted that rebleeding or increased intracranial pressure might rupture the membrane, leading to redistribution of hematoma to the spine.

In our case, considering the presence of acute on chronic ISDH and small non-compressing midthoracic SSDH, a possible mechanism could be the redistribution of ISDH. The acute exacerbation of the chronic ISDH can be explained by a sudden change in intracranial pressure caused by the loss of a substantial amount of CSF during spine surgery [19]. Since the possibility of ISDH tracking to the spinal sub-dural region exists in patients with idiopathic SSDH, we suggest a CT scan of the brain in all patients with SSDH to diagnose or rule out ISDH. This would help the surgeons to counsel the patients and plan the treatment accordingly.

In idiopathic SSDH, even though treatment is limited to the hematoma management, as there is no underlying etiology to address, still controversy exists over preferred treatment. Those with coexistent ISDH have been managed differently in different studies, and it varies from simultaneous surgical evacuation to conservative management of SSDH/ISDH (Table 1). Lee et al. [5] considered conservative management initially but surgery on 13th post-operative day due to increased back pain, numbness, and radiculopathy in the lower limb, whereas Lin et al. [6] considered subdural drain for ISDH and conservative management for SSDH, but the patient developed gait instability after one month and underwent craniotomy for acute on chronic SDH. Our case considering severe, intractable radicular pain with bilateral foot muscle weakness, leading difficulty to walk; we decided to perform decompression with hematoma evacuation. The patient’s immediate post-operative period was uneventful until he developed incoherent speech and hemiparesis due to acute on chronic ISDH. As the patient developed an acute neurological deficit, he underwent emergency burr hole with hematoma evacuation.

Table 2: MRI features differentiating SSDH and epidural spinal hematoma

| Location | Epidural spinal hematoma | Subdural spinal hematoma |
|----------|--------------------------|--------------------------|
| Sagittal Image | Well-demarcated biconvex lesion with tapered superior and inferior edges | Concave delination confined to the shape of the spinal canal |
| Axial Image | Present in epidural space in contact with the osseous structure | Present in subdural space and separated from the osseous structure |
| | Obliteration of epidural fat with dural displacement | Separated from epidural fat (cap sign) without dural displacement |
| | Biconvex in shape. | At cord level |
| | | Crescentic/biconvex |

Table 3: MRI appearance of spinal subdural hematoma

| Age     | Hemoglobin           | T1W   | T2W   |
|---------|----------------------|-------|-------|
| Hyperacute | <24 h Oxymoglobin | Isointense | Hyperintense |
| Acute       | 1-3 Days Deoxyhemoglobin | Hypointense | Hypointense |
| Early subacute | 3-7 Days Intracellular methemoglobin | Very hyperintense | Hypointense |
| Late subacute | 7-14 Days Extracellular methemoglobin | Hyperintense | Hyperintense |
| Chronic   | >14 Days Hemosiderin | Hypointense | Hypointense |

MRI: Magnetic resonance imaging; SSDH: Spinal subdural hematoma; ISDH: Intracranial subdural hematoma; T1W: T1-weighted image, T2W: T2-weighted image. | www.jocr.co.in

Conclusion

The presentation of a rare case of SSSDH in the background of a possible pre-existing ISDH is clearly explained in this case report. Based on this report, we highlight the importance of the surgeon being aware of this possibility. Whenever a patient is diagnosed to have SSSDH without any predisposing factor or antecedent event, the patient should be considered for a pre-operative CT scan.
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Hajare SS, Pushpa BT, Kanna RM, Shetty AP, Babu R, Rajasekaran S. Missed Intracranial Subdural Hematoma in a Case of Spontaneous Subdural Spinal Hematoma: A Rare Case Report and Literature Review. Journal of Orthopaedic Case Reports 2021 April;11(4): 75-79.

Conflict of Interest: Nil
Source of Support: Nil
Consent: The authors confirm that informed consent was obtained from the patient for publication of this case report

How to Cite this Article
Hajare SS, Pushpa BT, Kanna RM, Shetty AP, Babu R, Rajasekaran S. Missed Intracranial Subdural Hematoma in a Case of Spontaneous Subdural Spinal Hematoma: A Rare Case Report and Literature Review. Journal of Orthopaedic Case Reports 2021 April;11(4): 75-79.

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