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Motor Recovery After Acute Hemiparesis Caused By Spontaneous Cervical Spinal Epidural Hematoma: A Case Report

Authors: Munkado Wilfried Meuga MD [1,2], Meleine Landry Konan MD, MS [1], Soress Dongo MD [3], Keableon Louis Derou MD [3], Marc Sidoine Gbazi MD [1], Djibi Marcel Okamon MD [1], Dominique Ndri Oka MD [1]

Affiliations:
1. Department of Neurosurgery CHU Yopougon, Abidjan Côte d'Ivoire
2. Department of Neurosurgery, Université Côte Azur University, CHU de Nice, France
3. Department of Neurosurgery CHU Bouaké, Bouaké - Côte d'Ivoire

Corresponding author: Meleine Landry Konan MD, MSc, E-mail address: landrix07@gmail.com, Phone: +2250707604150
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Abstract:

Spontaneous Spinal Epidural Hematoma (SSEH) is a rare condition characterized by the accumulation of blood within the epidural compartment of the cervical spinal column, leading to compression of neurological structures. Swift medical and surgical intervention is imperative due to the severe neurological consequences that may arise. In this context, we present the case of a 69-year-old woman who exhibited profound right hemiparesis, ultimately unveiling a posterior epidural compression. Given initial suspicions of a neoplastic process, the administration of 80 mg prednisolone was initiated as an emergency measure, remarkably resulting in rapid clinical amelioration. Subsequently, the planned surgical intervention for this lesion was deferred, and the patient's medical treatment persisted for 6 days. Strikingly, a full recovery of the motor deficit was discerned within 48 hours from the commencement of the medical regimen. This case underscores the successful employment of corticosteroids in the resolution of a SSEH, as evidenced by the complete recovery of motor function within a 48-hour window after the inception of medical intervention.

Key Words: Spontaneous Spinal Epidural Hematoma; corticosteroids; cervical medullar compression.

Introduction:

Spontaneous Cervical Spinal Epidural Hematoma (C-SSEH) is a condition where blood accumulates in the epidural compartment of the cervical spinal column. This is a rare condition with an estimated incidence of 0.1 in 100,000 per year [1]. Unlike traumatic spinal epidural hematoma, C-SSEH Hematoma is characterized by its sudden non-traumatic and rapidly worsening onset [2]. C-SSEH usually presents with a sudden onset of neck pain followed by symptoms and signs of rapidly evolving nerve root and spinal

cord compression [3]. Early diagnosis and prompt management correlate with good outcomes. Here, we present the case of a 69-year-old woman presenting with an idiopathic C-SSEH at the C4-C5 level of the cervical spinal segment revealed by a right hemiparesis and we then present a brief review of the literature.

Case presentation

A 69-year-old female was having difficulties driving her car. She was brought to the emergency department of the Nice CHU Côte d'Azur University, because of the sudden onset of a right cervico-brachial neuralgia with an associated weakness of the corresponding upper limb. She had no history of head and spinal trauma, smoking, drinking or any family history of vascular disease. She did not take any antiplatelet and anticoagulant agents. On physical examination, the patient was conscious, had no history of fever and her blood pressure was about 160/80 mmHg. Neurological examination revealed rapid worsening of the symptoms with right hemiparesis predominating in the upper limb. The patient had neurological level at C6 nerve root motor deficit graded 4/5 and C6 neuralgia and C7, C8, T1 motor strength was graded 0/5. On the right lower limb, the motor strength was graded 2/5 with sacral sparing. The rest of the examination was unremarkable.

Given the sudden onset, an ischemic vascular cerebral accident was initially suspected. However, a brain MRI ordered was normal. Hence, an additional Cervical spine MRI was ordered which revealed a posterior epidural lesion extending from C4 to C5, with a slightly hyperintense signal on T1-weighted images and hyperintense signal on T2-weighted images (Figure 1a-c). The MRI characteristics of the lesions were highly suspicious of an acute compressive posterior cervical epidural hematoma extending from C4 to C5.
As a differential diagnosis, a neoplastic vertebral tumour or a posterior epidural abscess was evoked. Given the neurological deficit, a trial of 80 mg of methylprednisolone was administered in an emergency. Blood tests including complete blood count, chemistry panel and coagulation profile were normal.

An emergent decompressive laminectomy was initially indicated; however, a subsequent neurological examination within eight hours of admission revealed clinical improvement. Consequently, we opted to continue the medical treatment, ensuring close clinical and radiological monitoring within our department. Clinical monitoring demonstrated a complete recovery of the motor deficit, and neuroimaging conducted 48 hours after corticosteroid administration showed the total resolution of the posterior epidural hematoma. (Figure 1d-f). We chose to administer methylprednisolone intravenously at a dose of 80 mg per day for a total of six days after which the patient was discharged. Medical and neuroimaging follow-up conducted three months post-hospitalization showed a complete motor recovery and complete resorption of the posterior epidural collection (Figure 1g-i).

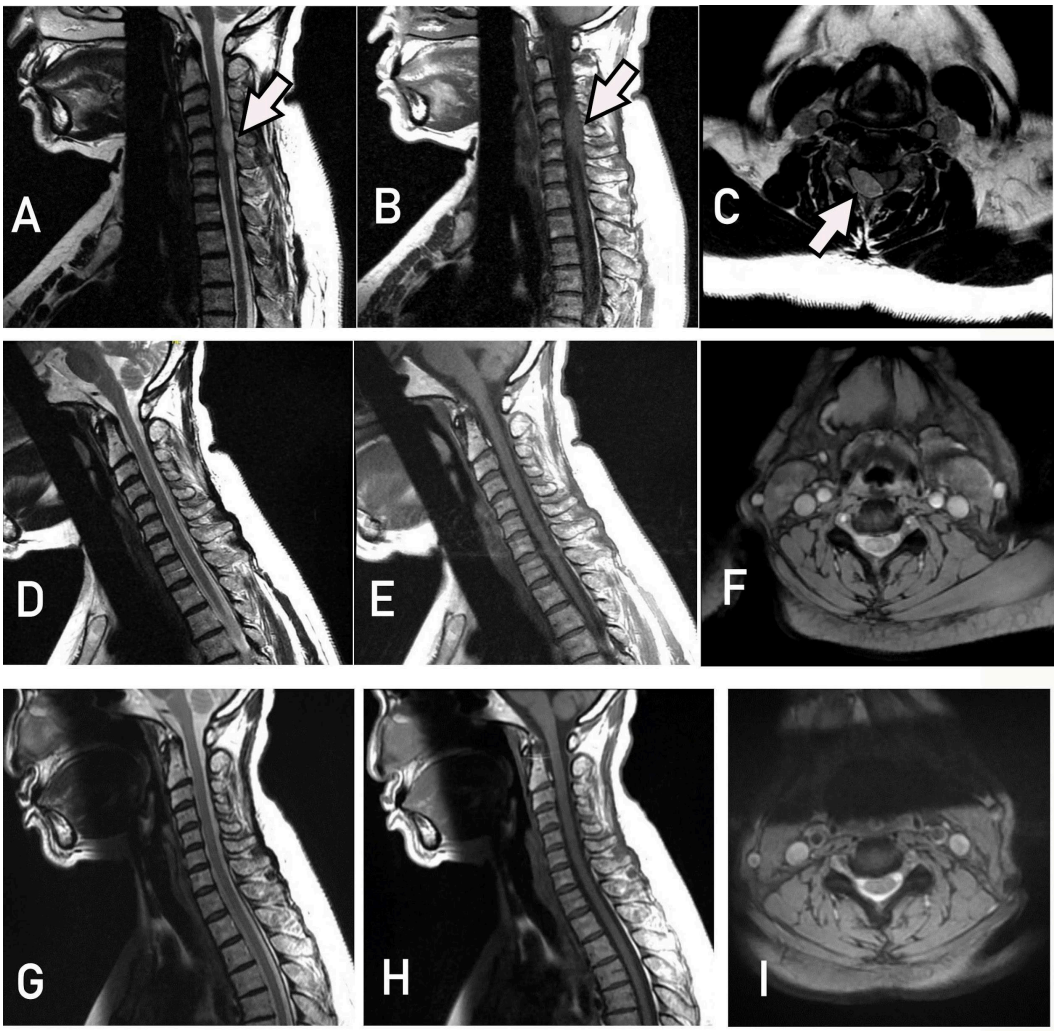

Figure 1: Initial and follow-up cervical MRI. A-C: Initial cervical MRI showing the compressive mass at C4-C5 (white arrow) level isointense in Sagittal T2-weighted imaging (A), slightly hyperintense in Sagittal T1-weighted (B), and Axial T2-weighted (C) characteristic of a hematoma. D-F: Follow up on Day 2 after administration of Glucocorticoid showing complete disappearance of the posterior epidural hematoma. A slight thickening of the posterior epidural compartment at the C4-C5 level is noticed. G-I: Follow-up MRI at 3 months showing the disappearance of the posterior epidural hematoma and the hyperintense signal seen in the cervical spinal cord.

Discussion

Spontaneous cervical spinal epidural hematoma is a relatively rare pathology characterized by blood accumulating into the epidural space and compressing the spinal cord. The first case of spontaneous spinal epidural hematoma (SSEH) was reported by Jackson et al in 1869[3]. In the literature, there is still a great controversy behind the definition of SSEH. Many authors include in this pathology: hematomas secondary to coagulopathy, vascular malformations and hemorrhagic tumors, while others define SSEH as hematoma of idiopathic origin [3, 4].

According to Liu Zhan et al., approximately 40 to 61% of cases without any underlying cause reported could match the criteria for idiopathic SSEH, the main aetiology of secondary SSEH being coagulopathies, accounting for 20% to 30% of all SSEH [5]. The incidence of SSEH was estimated approximatively to 0,1 per 100,000 people, however, this has increased nowadays with the development and availability of MRI [3, 6].

SSEH can occur in people of any age, but more frequently in the elderly between 40 to 80 years old such as our case where

the patient was 69 years old.[4, 6, 7] Any level of the spinal canal may be involved, nevertheless, the location of the hematoma appears to have a bimodal distribution, with peaks at C6 to C7 and T12 not in line with our case which was in C4 to C5.[3, 5, 7] The hematoma generally remains limited to few segments, two to four. The disease-related mortality rate ranged from 6 to 8% and was highly correlated with cervical or cervicothoracic hematomas, especially in patients with cardiovascular disease and those undergoing anticoagulant management.[8, 9] Risk factors of secondary SSEH include hemorrhagic disorders due to anticoagulants, thrombolytics and antiplatelet agents, platelet dysfunction, pregnancy, vascular malformation, neoplasms and systemic diseases, such as hypertension and rheumatoid arthritis.[4, 7, 8, 10] We did not find any of those risk factors in our patient. The cause of SSEH is uncertain, despite the presence of literature in support of both venous and arterial origin.[8] However, the most widely accepted physio-pathological hypothesis is venous bleeding from internal vertebral venous plexus, which are epidural valveless veins not protected against abdominal or thoracic pressure variations.[4, 6, 11, 12] The hematoma in this case has the same location as reported in the prior literature, that is usually posterior to the spinal cord according to one large literature survey.[12]

Clinical presentation and differential diagnosis

The typical initial symptom of SSEH is generally acute pain in the neck or back, including radiative pain on occasion, followed by neurological deficits of varying severity. There can be a delay between the onset of neck or back pain and the development of neurological impairment. This delay has been reported to range from a few minutes to several days or months.[6] However, the clinical manifestation in some patients may be atypical or absent, both of which may contribute to delays in diagnosis and treatment.[6, 7, 10] Our patient presented with neck pain radiating to the corresponding upper limb, with secondary progression to a right hemiparesis without sensitive dysfunction. Patients with hemiplegia secondary to SSEH may be misdiagnosed with an acute ischemic cerebrovascular event and treated with anticoagulation or antiplatelet leading to worsening of the epidural hematoma and corollary neurological impairment.[8] Therefore, persistent neck or back pain and fluctuating neurological symptoms should result in a high index of suspicion of the illness. MRI is currently the goal standard for the diagnosis of SSEH.[13] It serves for identification of the hematoma, differential diagnosis with other compressive lesions such as tumors, arteriovenous malformation or epidural abscess and identification of spinal cord edema related to it compression, which is a sign of poor clinical outcome.[13, 14] In the initial phase, an acute haematoma has an iso signal in T1 weighted scans and hypersignal in T2 weighted scans. In the following days, the haematoma signal intensity can alternate in both sequences, but it typically increases in T1 weighted scans during days 3 to 5.[13, 14] Afterwards, in chronic haematoma, both T1 and T2 signal decrease due to hemosiderin formation. Loss of signal in the T2* gradient sequence helps to differentiate haematoma from other expansive lesions; this phenomenon is caused by the presence of deoxyhemoglobin and hemosiderin in acute and chronic haematoma, respectively.[13-15] In our case, we also suspected a neoplastic vertebral tumour and an epidural abscess. However, the MRI findings help rule out an epidural abscess which appears contrast-enhanced on T1 gadolinium MRI. Similarly, neoplastic vertebral tumours were readily excluded given the absence of pathologic vertebral on MRI.

Principles of conservative treatment, selection criteria and role of glucocorticoids

Treatment options reported in the literature consist of either conservative or surgical management depending on the severity and dynamics of the neurological

symptoms [11, 13] . The mainstay of treatment for SSEH has been early surgical decompression via a prompt decompressive laminectomy or hemilaminectomy combined with hematoma evacuation [6, 8, 9, 11, 13, 16, 17]. The rationale for surgical intervention is to promptly relieve the compression on the spinal cord and prevent further neurological deterioration. Several studies have demonstrated that early surgical decompression, preferably within 24-48 hours of symptom onset, is associated with better neurological outcomes while delayed surgical intervention beyond this time window may result in poorer prognosis and increased risk of permanent neurological deficits [18]. However, not all patients qualify for a prompt surgical decompression and these patients are treated conservatively. Patients with severe neurological deficits after SSEH may receive conservative treatment because of serious coagulopathy or anticipated surgical risks. Other reasons for not opting for surgery include patient refusal. Groen et al in a systematic review of 64 cases reported that 36% of these patients suffered from coagulopathy due to either haemophilia or intake of anticoagulants [19]. Hence, the choice for conservative treatment of SSEH isn't always due to a mild clinical course. Several reports indicate that conservative methods have led to the spontaneous resolution of SSEH [11, 19].

The decision for conservative treatment should be made cautiously, as there is a potential risk of neurological deterioration if the hematoma expands or fails to resolve spontaneously. Close monitoring and frequent neurological assessments are essential for patients managed nonoperatively, with a low threshold for surgical intervention if any worsening occurs. The key factors favouring non-operative management include (1) minimal or mild neurological deficits at presentation, (2) non-progressive or stable neurological status over time, (3) minimal or moderate degree of spinal cord compression on imaging studies, and (4) the presence of serious coagulation disorders [20]. Conservative SSEH management is based on the elimination of risk factors, minimizing the risk of further bleeding and administration of glucocorticoid with close observation in a neurosurgical unit and repeated Magnetic Resonance imaging studies. Practically, SSEH patients should undergo comprehensive screening for coagulopathies, which may stem from either inherited disorders like hemophilia, or the consumption of anticoagulant medications prescribed for cardiovascular conditions. In such scenarios, prompt initiation of replacement therapy, comprising the administration of clotting factors or anticoagulant reversal agents, is warranted. While Crabbe has proposed temporary cervical immobilization for cervical or cervicothoracic hematomas, the clinical benefit of this approach remains unsubstantiated by empirical studies [21]. Furthermore, the utilization of glucocorticoids is predicated on their well-established potent effect on platelet adhesion[22]. Glucocorticoids inhibit tissue plasminogen activator (t-PA) by increasing the level of plasminogen activator inhibitor and increasing the level of clotting factors VII, VIII, XI and fibrinogen, promoting a procoagulant state [23].

Outcomes of conservative treatment

The neurological outcome is related to the preoperative status of the neurological deficit and the operative time interval. Zhang et al. also reviewed 17 cases of spontaneously resolved SSEH patients reported in the English-language literature, with radiological imaging proving the complete disappearance of the epidural hematoma and concluded that spontaneously resolved SSEH patients experienced neurologic improvement in the first 24 h, hence if neurologic improvement is not observed within the first 24 h in a patient with severe neurologic deficit, or if neurologic deterioration occurs and neurologic improvement stops at an unacceptable level, surgical intervention should be undertaken as soon as possible.[7] In our case, motor improvement occurred 8 hours following the onset of neurological

deterioration, suggesting conservative management could be a relevant management option. In his review, Groen et al reported a complete recovery rate of 84% acutely following conservative treatment. Another study reported that 73% of conservatively managed patients made a full recovery, compared to 48% improvement in the surgically managed group [24]. A more robust study is required to further compare the outcomes of surgical versus conservative treatment.

Conclusion

SSEH is a rare case of emergency, needing a high index of suspicion for prompt clinical diagnosis and MR imaging performed. Screening for risk factors should be an integral part of the management. While early decompressive surgery is strongly supported, a conservative approach remains viable in cases with moderate neurological deficits if neurological improvement is observed within the first 8 hours. However, decompressive laminectomy should be promptly available if the patient's neurological status worsens or fails to improve shortly after presentation.

Author contributions

The authors were granted complete access to the data, actively participated in the study, endorsed the final version for publication, and assumed accountability for its precision and integrity.

Disclosure statement

The authors report no conflict of interest.

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