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# Spontaneous Spinal Epidural Hematoma as a Potentially Important Stroke Mimic

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**ABSTRACT:** Hemiparesis develops in response to a wide range of neurological disorders, such as stroke, neoplasms and several inflammatory processes. Occasionally, it may also occur due to a lesion located in the high cervical spinal cord. In this concise review, we describe the features of spontaneous spinal epidural hematoma, which should be included in the large list of stroke mimics. Various concerns regarding the diagnostic and therapeutic conundrums relating to the condition are also discussed.

**KEYWORDS:** hemiparesis, ischemic stroke mimic, spontaneous spinal epidural hematoma

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## Introduction

Hemiparesis develops in response to a wide range of neurological disorders. The presence of concomitant cranial nerve signs or facial weakness generally prompts a search for cerebral etiologies such as stroke, neoplasms or inflammatory processes, while it may occasionally be due to a lesion located in the high cervical spinal cord.[1] In this concise review, we describe the features of spontaneous spinal epidural hematoma (SSEH), which is a rare cause of spinal cord compression and a neurological emergency requiring prompt diagnosis and management to prevent morbidity and mortality.[2,3] Most SSEH patients present with either paraplegia or tetraplegia; however, there are numerous descriptions about acute hemiparesis as an initial manifestation of SSEH, which may lead physicians to include an acute ischemic cerebrovascular event as a diagnostic consideration,[4] thus leading to the concept that SSEH should be included in the large list of stroke mimics. In the following sections, we also discuss various concerns regarding the diagnostic and therapeutic conundrums associated with this disease.

## Epidemiology and Pathophysiology

SSEH represents a rare spinal emergency, with a frequency accounting for less than 1% of spinal epidural space-occupying lesions.[2,3] Jackson first described SSEH in a 14-year-old female in 1869,[5] and the first surgically-treated case was reported by Bain in 1897.[6] Although the introduction of neuroradiological investigations and progress in neurosurgery may lead to a sharp increase in the number of diagnoses of spinal bleeding,[7] only approximately 530 cases were included in the largest recent literature search for SSEH.[8] Holtas et al have reported their experience with 13 patients with SSEH during a nine-year period in a population of 1.49 million total patients, giving an incidence, on the basis of their population, of approximately 0.1 patient per 100,000 patients per year.[9] Most of the patients present in their 60s or 70s, but all age groups from six months[10] to late 80s have been affected, with a slight predominance of the male sex.[2,7]

Any level of the spinal canal may be involved; the location of the hematoma appears to have a bimodal distribution,

with peaks at C6 to C7 and T12,[2,8] while the level distribution has been shown to depend strongly on age. The localization of SSEH at the lower dorsal and lumbosacral segments under the age of 40 appears to be an exception.[2] The hematoma remains limited to a small number of segments (three to four).[2,11] The disease-related mortality rate ranged from 6 to 8%, and highly correlated with cervical or cervicothoracic hematomas, especially in patients with cardiovascular disease and those undergoing anticoagulant management.[2,11,12] A recent study also revealed that a long hematoma may predict a worse outcome.[13]

Although the precise pathogenesis of the disease remains to be delineated, hemorrhagic disorders due to anticoagulants, thrombolytics and anti-platelet agents, platelet dysfunction, pregnancy, vascular malformation, neoplasms and systemic diseases, such as hypertension and rheumatoid arthritis, have been considered as predisposing factors, while many cases without any known underlying cause also have been demonstrated.[2,12–14] Approximately 3% of patients are hypertensive; however, a relationship between hypertension and the development of a hematoma is considered to be marginal.[2] Nevertheless, the clinical significance of these previous findings should be evaluated carefully in terms of the fact that there is still no established definition of SSEH. A spontaneous hematoma is most often defined as a condition occurring in the absence of any traumatic event or iatrogenic procedure,[15] and thus, some of the predisposing factors described above cannot be excluded. Other authors argue that a hematoma can only be labelled "spontaneous" when it is of idiopathic origin.[16]

Regardless of the etiological background, the actual origin of spinal bleeding has been the subject of some discussion. Despite the presence of literature in support of both venous and arterial origin,[17,18] the posterior epidural venous network is believed to be the most likely source of the hematoma, due to the predominance of posterolateral hematomas, the segmental distribution of SSEH and the anatomical characteristics of the internal vertebral venous plexus, in addition to the fact that the spinal epidural veins have no valves and are thus prone to damage by changes in abdominal or thoracic pressure.[2,12,15] In a recent case series analysis, more than half of the patients with SSEH were reported to have experienced a subjective straining-associated event during the initial attack, lending even more credibility to the venous etiology theory.[14] On the other hand, Beatty and Winston made a radical argument for an arterial source of hemorrhage, at least in the cervical region, and they focused on the fact that the intrathecal pressure is higher than the venous pressure at the cervical level, which would preclude bleeding in the intrathecal space from veins.[18]

## Clinical Presentation and Misleading Symptoms: Relevance to a Potentially Important Stroke Mimic

The onset of SSEH may be associated with neck or back pain radiating to the corresponding dermatome, which may sometimes be vague and ignored until the subsequent cord compression and neurological deficits arise.[13] Most patients present with paraplegia or tetraplegia,[5,11,19] while hemiparesis is considered to be a rare feature of SSEH.[11,19] Due to the rarity of the disease, no prospective series from single departments or study groups are available to date, and the international medical literature, including incidental cases from one's own department, is the only source of information for evaluating this topic.[8]

More than two decades ago, Anderson et al reported that the association with hemiparesis is low, occurring in only 6 of more than 250 reported cases.[20] However, modern radiological imaging modalities may overcome the shortfalls of clinical examinations in the management of acute spinal cord injuries, and may lead to new findings. Following the first report published on the use of magnetic resonance imaging (MRI) in the diagnosis of acute SSEH in 1987,[21] the mean incidence of such cases increased from 2.2 to 6.4 new cases per year.[22] Therefore, the precise incidence of hemiparesis among the overall patients with SSEH must be evaluated carefully. Indeed, anecdotal information regarding cases with hemiparesis as an initial presentation is still being accumulated.[4,14,19,23–33] The majority of hematomas in such cases were located within the cervical region, while four patients with hematomas that ranged from the cervical to thoracic region were also reported (Table 1). The presence of a spinal epidural hematoma was promptly diagnosed in more than half of these cases during the observation period.

Persistent neck or back pain and fluctuating neurological symptoms should result in a high index of suspicion of the illness. However, it is noteworthy that there were at least twelve patients who were suspected to have an ischemic stroke during the initial assessment,[4,23,25,27,29–33] and thus, there were four patients whose neurological manifestations were falsely attributed to ischemic cerebrovascular events who were consequently subjected to anti-coagulation with heparin,[25,27] thrombolytic treatment with a recombinant tissue plasminogen activator[29] or treatment with an anti-platelet agent as an adjunct to warfarin,[23] prior to the final diagnosis that their neurological deficits were etiologically linked to the cervical spinal epidural hematoma. In these cases, the presence of neck pain with or without shoulder pain might have been inadvertently dismissed during the initial physical assessment. Alternatively, or in addition, the occurrence of concurrent dysarthria,[29] which has been demonstrated to be a potential sign for discriminating stroke mimics from actual ischemic stroke in emergency settings,[34,35] might also have played a role in the false attribution of the illness to a cerebrovascular event.[1] The main cause of dysarthria in the patient with hemipareic SSEH described by Son et al.[29] is unclear. However, they concluded that dysarthria is a rather subjective symptom of patients with stroke, and they noted that removing the patient's dentures during the acute phase of the disease might have resulted in the slurring of speech.[29] Finally, the hemorrhagic risks associated with anticoagulants may not be equivalent to those of a

**Table 1.** The recently reported cases of SSEH with hemiparesis as an initial presentation.

| AUTHOR (REF NO.) | AGE (YEARS) | SEX (M/F) | INITIAL PRESENTATIONS | TREATMENT FOR ISCHEMIC STROKE | LOCATION OF HEMATOMA | TREATMENT FOR SSEH | OUTCOME |
|---|---|---|---|---|---|---|---|
| Schmidley JW et al[4] | 96 | f | neck pain, shoulder pain, left hemiparesis | (–) | C5–C7 | surgery | incomplete recovery |
| | 81 | f | neck pain, shoulder pain, right hemiparesis | (–) | C3–C6 | surgery | incomplete recovery |
| Lobits B et al[14] | 85 | f | neck pain, shoulder pain, right hemiparesis | (–) | C5–C6 | surgery | incomplete recovery |
| Marinella MA et al[19] | 60 | f | neck pain, left hemiparesis | (–) | C2–C6 | conservative | complete recovery |
| Sakamoto N et al[23] | 75 | f | neck pain, right hemiparesis | anti-platelet treatment | C3–C4 | surgery | complete recovery |
| Lin IY[24] | 83 | f | neck pain, right hemiparesis | (–) | C1–C7 | surgery | died |
| Hsieh CF et al[25] | 65 | m | neck pain, right hemiparesis | anti-coagulation | C3–C5 | surgery | incomplete recovery |
| Ishikawa E et al[26] | 83 | m | neck pain, left hemiparesis | (–) | C2–C6 | surgery | incomplete recovery |
| Wang CC et al[27] | 69 | m | neck pain, shoulder pain, right hemiparesis | anti-coagulation | C4–C5 | surgery | complete recovery |
| Nakanishi K et al[28] | 73 | f | neck pain, right hemiparesis | (–) | C3–C5 | surgery | incomplete recovery |
| | 62 | m | neck pain, right hemiparesis | (–) | C6–T1 | surgery | complete recovery |
| | 60 | f | neck pain, left hemiparesis | (–) | C2–C4 | conservative | complete recovery |
| Son S et al[29] | 63 | m | neck pain, shoulder pain, left hemiparesis | thrombolytic treatment | C4–T2 | surgery | incomplete recovery |
| Shima H et al[30] | 84 | f | neck pain, right hemiparesis | (–) | C2–C3 | conservative | complete recovery |
| Matsumoto H at al[31] | 71 | f | neck pain, right hemiparesis | (–) | C2–T4 | conservative | complete recovery |
| | 54 | f | neck pain, right hemiparesis | (–) | C3–T2 | conservative | complete recovery |
| Liou KC et al[32] | 60 | f | neck pain, right hemiparesis | (–) | C2–C6 | surgery | incomplete recovery |
| | 58 | f | neck pain, right hemiparesis | (–) | C2–C5 | conservative | complete recovery |
| Lemmens R et al[33] | 66 | f | shoulder pain, right hemiparesis | (–) | C2–C7 | conservative | complete recovery |

thrombolytic agent and antiplatelet agent; however, a previous case presentation demonstrating the expansion of an already developing cervical spinal epidural hematoma by the addition of heparin, based on a presumptive diagnosis of a cardiac ischemic event,[36] suggests that the clinical picture of patients with SSEH mimicking an ischemic stroke might be modulated by the agents described above.

## Management of SSEH

The mainstay of treatment for SSEH has been surgical evacuation, combined with prompt decompressive laminectomy. There have been several studies that have provided valuable information regarding the outcome following the surgical management of SSEH.[11–13,37,38] Based on these findings, the degree of preoperative neurological deficit and the

interval between the onset and surgery have been considered to be the critical factors determining the qualitative postoperative recovery.[12] Incomplete deficits, compared to complete deficits, should be associated with better outcomes. Indeed, the one-year complete recovery rate has been demonstrated to be approximately 89% for patients with incomplete deficits, but only 37.5% for those with complete deficits,[11] and similar trends have been demonstrated numerous times in the literature.[12,38] In addition, it is necessary to focus on the timing of surgery. In patients with a complete deficit, it has been shown in an analysis of 35 patients with SSEH that a good neurological recovery can be achieved after prompt surgery when the time interval from initial ictus to surgery is less than 48 hours, and when the duration of the complete neurological deficits is less than 12 hours.[11] Another study has demonstrated that a significantly better outcome was obtained if surgical decompression was carried out within 36 hours in patients with complete sensorimotor loss, and within 48 hours in those with incomplete deficits.[12] In a series of 14 patients reported by Shin et al, the patients who were surgically managed within 12 hours after the onset of symptoms scored significantly higher (84%) on the recovery scale of the Japanese Orthopedic Association[39] than those who underwent an operation from 12 to 24 hours after the initial attack (63.6%) and those who underwent surgery more than 24 hours after onset (46.7%).[38] Similar trends were also noted by Lawton et al.[37] The postoperative mortality rate of SSEH can thus be expected to range from around 3% to 6%.[11,37]

On the other hand, conservative management may still be an important treatment option in some subsets of SSEH patients with mild, rapid and spontaneously recovering neurological deficits.[8,40] Groen et al recently demonstrated that 84% of patients with SSEH treated non-surgically recovered completely.[8] However, this finding should be evaluated carefully in terms of the severity of the neurological deficits manifested by the subjects in their study. Indeed, as they stated, the subjects managed conservatively had less severe signs and symptoms and were more likely to be diagnosed based on an imaging modality alone, compared to the surgically-treated cases reported in the literature.[8] Moreover, a concurrent high surgical risk, such as bleeding diathesis, may also be a determinant in the decision for conservative management.[8] It is not surprising that some patients with minimal neurological signs may respond successfully to conservative management; however, the presence of cases in which an initial recovery was followed by deterioration requiring surgery should be kept in mind, and thus, the close observation of such patients should be carried out in a hospital with a neurosurgical specialty team.[40]

## Perspectives

In the ordinary clinical setting, acute stroke patients almost never contact a neurologist initially, since most communities do not have an academic medical center with a systemic neurological assessment program.[41] It is known, however, that neurological complications contribute to morbidity and mortality in a wide range of patients,[42–44] and an accurate recognition of the condition is required to ensure prompt transfer to an appropriate treatment unit. Moreover, there is a limited time window for the administration of a thrombolytic agent, which is the only available medical treatment for acute ischemic stroke that has been proven to be effective.[45] In this regard, not only emergency medical services and emergency room departments, but also primary care physicians, should be familiar with the wide range of neurological problems that can develop in order to assure the optimal management of patients. We feel that an early and accurate diagnosis, as well as awareness of SSEH, remains a challenge for physicians, despite the accumulation of studies disclosing the nature of the disease. The current concise review emphasizes the pitfalls of evaluating patients with acute hemiparesis due to SSEH. The imaging analyses routinely applied for patients with acute cerebral ischemia do not rule out spinal bleeding, while the higher a cervical SSEH extends, the more likely it is to be seen on non-contrast computed tomographic scans.[4] The sudden onset of neck and/or back pain and progression of hemiparesis to paraplegia or tetraplegia during the observation period are clues leading to a timely diagnosis of cervical spinal lesions.[4,19,24,27,30–32] Although there may be limited access to MRI,[46] negative brain MRI findings, which strongly suggest the absence of ischemic cerebrovascular events,[47] may be an alternative trail turning the attention towards other regions, including the cervical spine. In addition, the presence of concurrent Broun-Séquard syndrome, characterized by the ipsilateral loss of proprioceptive sensitivity with the contralateral loss of pain and temperature sensitivity, can help to promptly diagnose the disease,[4,48–51] although this syndrome is not necessarily associated with hemiparesis as an initial neurological presentation.[52]

Stroke mimics may account for 20 to 25% of suspected stroke presentations,[53] and there are various conditions (Table 2) that have been demonstrated to act as stroke mimics in previous studies.[54–57] There are some discrepancies in the distribution of diagnoses, depending on the context, while seizure seems to be one of the major common denominators, being present in approximately 15 to 20% of the stroke mimic cases.[54–57] Some studies included the presence of cervical or spinal lesions in the conditions that mimicked stroke;[54,56] however, the lack of information regarding the disease spectrum precludes us from determining the actual frequency of SSEH among subjects with a condition mimicking stroke. Nevertheless, we believe that SSEH, especially in the cervical region, should also be included as such a clinical entity,[4] thereby leading to a high index of suspicion, prompt recognition and immediate intervention, which is essential to reduce or prevent major morbidity of the disease.[36] Several studies have demonstrated the safety of thrombolytic treatment in some subjects with stroke mimics,[58] although there has been

**Table 2.** The previously reported conditions that mimicked stroke.

| |
|---|
| Seizure |
| Systemic infection/sepsis |
| Brain tumor |
| Toxic/metabolic |
| Vestibular dysfunction |
| Syncope |
| Subdural hematoma |
| Subarachnoid hemorrhage |
| Intracranial hemorrhage |
| Transient global amnesia |
| Dementia |
| Conversion disorder |
| Migraine |
| Peripheral neuropathy |

only a single report regarding a patient with SSEH mimicking a stroke being administered a thrombolytic agent as an initial treatment, and this was followed by urgent laminectomy without any bleeding complications.[29] Considering the narrow window for thrombolytic treatment in cases of ischemic stroke and the rarity of SSEH, one may argue that our proposal is not necessarily justified. On the other hand, the association between the administration of a thrombolytic agent and the secondary development of spinal epidural hematoma has been demonstrated anecdotally.[59,60] Moreover, the inappropriate administration of agents used for restoring cerebral blood flow may preclude prompt surgery for SSEH, due to the patient's modified hemostatic nature, although the information currently available may not necessarily support this concept.[29] Thus, we believe that it is necessary to take a proactive approach by adding SSEH to the list of differential diagnoses of ischemic stroke before fatal outcomes accumulate.[36] Finally, it should be kept in mind that we are always facing, as do most physicians at various times, diagnostic and therapeutic dilemmas, and carefully weighing all of the options and potential outcomes on a case-by-case basis is therefore essential.

## Author Contributions

TA and TY drafted the manuscript and made equal contributions to the literature survey. SS, YA, and DN provided a detailed review of the contents and structure of the manuscript, resulting in significant changes to the original document. All authors have read and approved the final manuscript.

**DISCLOSURES AND ETHICS**

As a requirement of publication the authors have provided signed confirmation of their compliance with ethical and legal obligations including but not limited to compliance with ICMJE authorship and competing interests guidelines, that the article is neither under consideration for publication nor published elsewhere, of their compliance with legal and ethical guidelines concerning human and animal research participants (if applicable), and that permission has been obtained for reproduction of any copyrighted material. This article was subject to blind, independent, expert peer review. The reviewers reported no competing interests.

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