

# Subarachnoid Hematoma of the Craniocervical Junction and Upper Cervical Spine After Traumatic Cerebral Contusion: Case Report

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## Abstract

Spinal subarachnoid hematoma (SSH) is a rare condition, more commonly occurring after lumbar puncture for diagnostic or anesthesiological procedures. It has also been observed after traumatic events, in patients under anticoagulation therapy or in case of arteriovenous malformation rupture. In a very small number of cases no causative agent can be identified and a diagnosis of spontaneous SSH is established. The lumbar and thoracic spine are the most frequently involved segments and only seven cases of cervical spine SSH have been described until now. Differential diagnosis between subdural and subarachnoid hematoma is complex because the common neuroradiological investigations, including a magnetic resonance imaging (MRI), are not enough sensitive to exactly define clot location. Actually, confirmation of the subarachnoid location of bleeding is obtained at surgery, which is necessary to resolve the fast and sometimes dramatic evolution of clinical symptoms. Nonetheless, there are occasional reports on successful conservative treatment of these lesions. We present a peculiar case of subarachnoid hematoma of the craniocervical junction, developing after the rupture of a right temporal lobe contusion within the adjacent arachnoidal spaces and the following clot migration along the right lateral aspect of the foramen magnum and the upper cervical spine, causing severe neurological impairment. After surgical removal of the hematoma, significant symptom improvement was observed.

Key words: subarachnoid, hematoma, craniocervical junction, laminectomy, spinal

## Introduction

Spinal hematomas may occur in epidural, subdural, or subarachnoid location. Spinal subarachnoid hematomas (SSHs) are very rare. They may occur after lumbar puncture,<sup>1,8,17,20,22,27</sup> anticoagulation therapy,<sup>3,6</sup> arteriovenous malformation rupture,<sup>15</sup> neoplasms,<sup>11</sup> and traumatic events.<sup>4,5,9,10,12,13,18,19,21,26</sup> Occasionally, no etiopathogenetic agent can be identified and a diagnosis of spontaneous SSH is established.<sup>2,9,14,23–25,29</sup> In 1923, Devic and Durand<sup>7</sup> were the first authors to describe an SSH, caused by an accidental fall. In 1928, Sillevs Smitt<sup>28</sup> defined the subarachnoid location of the clot accurately. A third case was reported in 1933 by Wilson and coworkers.<sup>29</sup> Since then several cases of SSH of different etiologies have been reported. The more accurate surgical description of this condition is attributed to 1966 Plotkin<sup>23</sup> series (three patients): “the dura was dark blue and non pulsatile. It

was incised longitudinally and a clot was then evident beneath the intact arachnoid: it extruded spontaneously as the arachnoid was opened.” The term SSH was introduced by Berney<sup>2)</sup> in 1967.

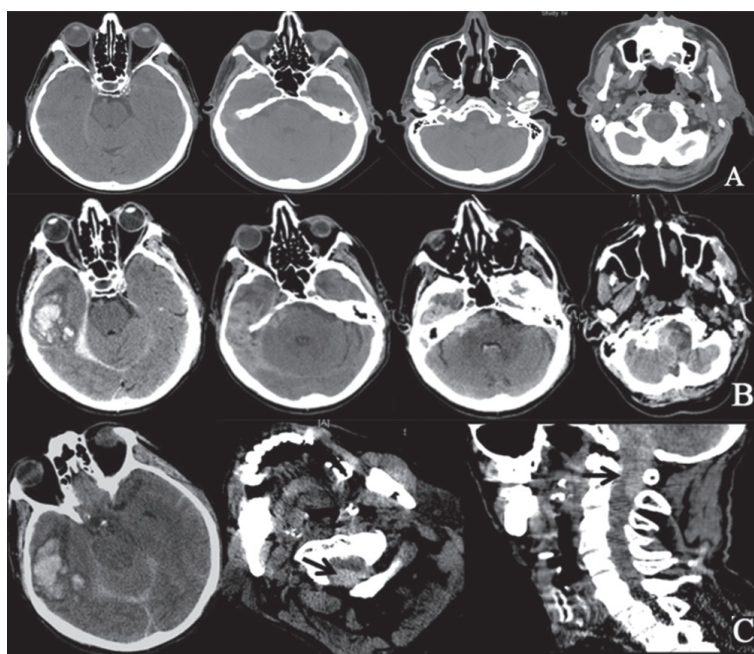
The differential diagnosis between spinal subdural and subarachnoid bleeding may be difficult to establish at common neuroradiological investigations<sup>10</sup> (including a magnetic resonance imaging [MRI]) and it is commonly made only at surgery. Diffusion of subarachnoid bleeding from the intracranial to the spinal compartment has been occasionally reported, involving the thoracic or lumbar vertebral segments.<sup>16,20</sup> In the majority of cases clinical evolution is fast and dramatic and surgical evacuation may lead to prompt symptom resolution.

We present a case of severe neurological impairment sustained by the formation of an SSH occurring at the craniocervical junction, apparently secondary to blood migration from an intracerebral contusion. We discuss the potential mechanisms underlying symptom evolution, performing a detailed analysis of related literature.

## Case Report

A 71-year-old male was admitted to the Emergency Department of our Hospital after a car accident. At arrival, he was awake, able to interact with the observers, executing verbal commands at request. No motor or sensory deficits, neither cranial nerve dysfunction were evidenced. Laboratory examinations were in range, the patient was not taking antiaggregant or anticoagulant drugs. An initial computed tomography (CT) scan showed a small quote of subarachnoid hemorrhage within the middle temporal gyrus of the right temporal lobe (Fig. 1A). At 6 hours CT, a large, right temporal lobe contusion with minimal perilesional edema was observed (Fig. 1B). Blood surrounding the contused brain extended over the petrosal bone, descending inside the posterior cranial fossa, involving the cerebellopontine and cerebellomedullary cisterns, passing through the foramen magnum and reaching the right lateral aspect of the pons. A small amount of blood was found within the third and fourth ventricles also. Twenty-four hours after admission the patient started to complain of headache and nuchal pain, followed by the rapid development of a severe right-sided hemiparesis (F2), pharyngeal dysphagia, hoarseness, and pharyngo-laryngeal incoordination. A new CT scan showed an increase of the temporal contusion (Fig. 1C, on the left). Because of the incongruity of symptoms to lesion location, we decided to extend CT scan examination to the upper cervical spine. A large, intradural, right-sided blood clot occupied the upper spinal canal up to the third cervical

vertebra, displacing contralaterally the brainstem and the upper spinal cord (Fig. 1C, *black arrows*). Because of the severe neurological impairment the patient was taken to the operative room. A suboccipital craniectomy with major extension on the right side was performed, followed by C1–C2 laminectomy and C3 right hemilaminectomy. After bone removal, the dural layer was tense and, once microsurgically opened, the hematoma was immediately visible, enclosed by the arachnoidal layer. The cisterna magna was opened and adjunctive working space was gained by allowing free cerebrospinal fluid flow. The arachnoid layer was carefully opened and the hematoma progressively removed by suction and dissection, until full visualization of the underlying neurological structures. Nerves were carefully preserved during clot evacuation and completely freed. An expansive duraplasty was finally performed. The patient was transiently transferred to the ICU. Postoperative CT scan confirmed the evacuation of the whole spinal component of the hematoma and minimal residual clot within the cerebellopontine angle (Fig. 2A) Temporal contusion was unmodified. Two days after surgery a complete resolution of hemiparesis was observed. Dysphonia and dysphagia improved in the following 2 months but a complete recovery was never observed. A 3-month follow-up MRI showed a small ischemic lesion within the right dorsal aspect of the spinal cord in T2-weighted sequences (Fig. 2B, *black arrows*). CT scan performed at the same time showed full resorption of the temporal lobe contusion (Fig. 2B).



**Fig. 1** A: Admission computed tomography (CT) scan showing minimal subarachnoid bleeding within the right middle temporal gyrus. B: At 6 hours CT development of a large right temporal contusion. Blood surrounding the contusion descends over the petrosal bone inside the cerebellopontine and cerebellomedullary cisterns, reaching the right lateral aspect of pons. C: Twenty-four hours CT, after worsening of symptoms. A volume increase of temporal contusion is observed (on the left). The same CT extended to the cervical spine. A large intradural clot is seen displacing the upper spinal cord to the left side (*black arrows*).

## Discussion

SSHs are rare events, which have been more frequently reported following diagnostic or therapeutic rachicentesis,<sup>1,8,17,20,22,27</sup> in patients under anticoagulation or antiaggregation treatment,<sup>3,6</sup> or occurring spontaneously.<sup>2,9,14,23–25,29</sup> A traumatic etiology is extremely rare. In 1968, Bouzarth and Gutterman<sup>4</sup>) reported two cases of SSH after an accidental fall. In the first patient, a T1–T2 hematoma was surgically removed, with full neurological recovery from paraparesis at admission. The second patient presented with flaccid paraplegia and a laminectomy from T9 to C7 was performed, with the evacuation of a large SSH associated with an intraspinal C7 contusion, with no symptom improvement. Two years later Dabbert<sup>6</sup>) described a T7–L1 SSH following chiropractic manipulation. Patient's paraparesis at admission rapidly evolved to paraplegia, and completely resolved after laminectomy and hematoma removal. In 1980, Russell<sup>26</sup>) and coworkers operated on a C1–C6 SSH in association with brachial plexus avulsion, with incomplete neurological improvement. In 1985, Lesoin<sup>19</sup>) reported an SSH of the cauda equina caused by an accidental fall from a ladder. The resulting neurological symptoms promptly resolved after surgery. In 1987, Mori and colleagues<sup>21</sup>) described the occurrence of a Brown-Séquard syndrome following a hyperextension cervical injury. CT scan examination disclosed a C1–C3 left-sided SSH, which was fully removed at surgery, with complete neurological recovery at 6-month follow-up. In 1992, Katoh<sup>13</sup>) reported the case of a 76-year-old woman who had fallen down by accident 1 month before, admitted to the hospital with lumbar

pain radiating into her right thigh, monoplegia of the right leg, and urinary incontinence. Myelography and metrizamide CT demonstrated a filling defect mimicking intradural extramedullary tumor at the level of L1 and L2. An MRI revealed a subacute hematoma compressing the conus medullaris and the cauda equina. At surgery, an old hematoma was found within the subjacent subarachnoid space, which compressed the conus and cauda equina from right to left. Total hematoma removal was associated with full neurological recovery. In 1997, Gupta<sup>10</sup>) reported a T11–T12 hematoma occurring in a 6-year-old boy hit by a truck. At surgery the patient was paraplegic and there was no improvement after hematoma evacuation. Domenicucci et al.<sup>9</sup>) reported the only case of successful conservative treatment of a cervical SSH secondary to a domestic fall in a patient admitted asymptomatic.

In 2008, Chen<sup>5</sup>) and coworkers described a case of presumed migration of a traumatic SSH from the intracranial to the spinal compartment. After a fall from a motor vehicle, this 41-year-old female started complaining of left thigh numbness and heavy sensation, subsequently evolving in diffuse numbness of both legs and lower back pain. Admission CT scan evidenced traumatic subarachnoid bleeding within the left sylvian cistern. An MRI of the lumbar spine showed a fusiform lesion located at the lower end of the lumbar thecal sac. At surgery an old blood clot was removed after opening the arachnoidal layer and clinical symptoms fully resolved. The author supposed that blood migration could have occurred from the intracranial compartment to the end of the spinal canal. Nonetheless, considering the traumatic event, the

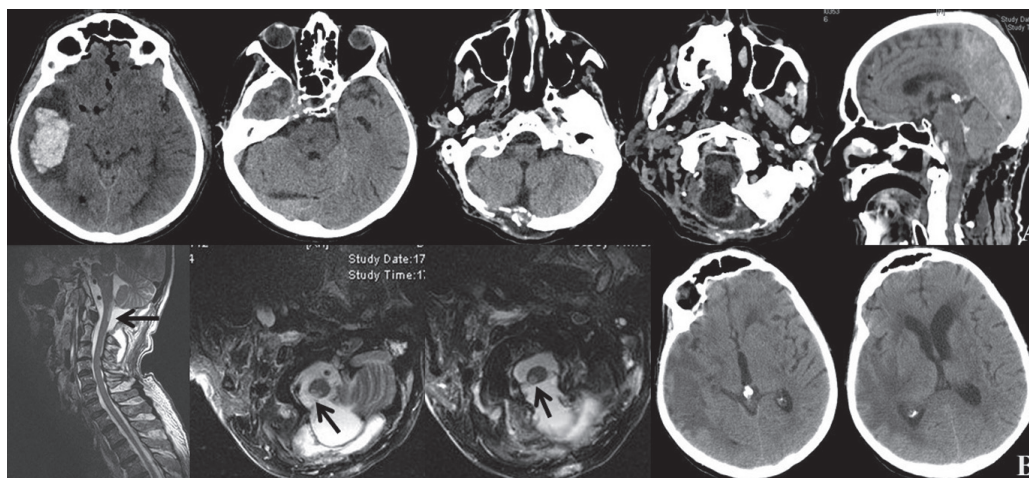


Fig. 2 A: Postoperative computed tomography (CT) showing no evolution of temporal contusion and the characteristics of bone opening (suboccipital craniectomy, C1–C2 laminectomy, C3 right hemilaminectomy). B: Three months postoperative magnetic resonance imaging (MRI) showing a small ischemic lesion within the right dorsal aspect of the spinal cord (*black arrows*). CT scan confirms full resorption of temporal contusion.



local development of SSH could not be excluded.

Our case presents some similarities to the one presented by Chen because of the traumatic etiology and the distant site appearance of the SSH. Nonetheless, the leading difference is that in our case the mechanism of hematoma formation seems well explained by imaging studies and fully accords to the timing of symptom progression. Presumably, the physiological evolution of the initial temporal lobe contusion led to blood spreading within the adjacent subarachnoid spaces. The organization of the arachnoid cisterns in this area is rather complex. The cisterns of the posterior cranial fossa include the interpeduncular, prepontine, cerebellopontine, premedullary, cerebellomedullary, quadrigeminal, superior cerebellar cisterns, and the cisterna magna. The anterior spinal and posterior spinal cisterns communicate through the foramen magnum with the posterior fossa cisterns. The glossopharyngeal, vagus, and spinal accessory nerves arise in the cerebellomedullary cistern, whereas the hypoglossal nerve passes through the premedullary cistern. The interpeduncular cistern communicates with the crural and ambient cisterns, which are situated in the tentorial area between the temporal lobe and the midbrain. Relying on this peculiar anatomy, we believe that blood extravasated from the temporal lobe contusion oozed from the cisterna ambiens to the cerebellopontine angle and cerebellomedullary cistern and finally to the posterior spinal cistern, as documented by neuroradiological examinations. In this restricted space, the large volume of the clot caused an underpressure plugging of the arachnoidal layer, causing the rapid evolution of symptoms. Even though prompt hematoma evacuation was performed, the direct mass effect and the immediate contact of blood with the IX–XIIth cranial nerves and the dorso-lateral surface of the brainstem and upper spinal cord did not allow a full neurological recovery, as lately documented at follow-up MRI by the development of a spinal cord contusion. Nonetheless, neurological improvement after surgery was remarkable and the partial recovery from dysphonia and dysphagia granted an autonomous lifestyle. To our knowledge, this is the only case ever reported of such an unusual evolution of a brain contusion and it seems to be the only case where a clear correlation between a severe head trauma and the development of an SSH could be documented.

### Conflicts of Interest Disclosure

None of the authors has any financial or personal relationship with people or organizations that could inappropriately influence their work. No actual or potential conflicts of interest exist with regard to the above submitted manuscript on behalf of any of the authors. All authors who are members of The Japan Neurosur-

gical Society (JNS) have registered online Self-reported COI Disclosure Statement Forms through the website for JNS members.

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