



Delayed Posttraumatic Subacute Lumbar Subarachnoid Hematoma: Case Report and Review of the Literature

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Key words

- Lumbar spine
- Spinal hematoma
- Subarachnoid hematoma
- Trauma

Abbreviations and Acronyms

MRI: magnetic resonance imaging

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INTRODUCTION

Traumatic spinal subarachnoid hematoma, associated or not with a concurrent subdural hematoma, has rarely been described.¹⁻³ The evolution of such hematomas is heterogeneous, opposing acute collection few hours after spinal injury and chronic collection similarly to their cranial counterparts, occurring several weeks after minor trauma.^{2,4,5} They are revealed by symptoms related to spinal cord compression, radicular compression, or both^{6,7} that can be uni- or bilateral.^{1,8,9}

The management of such hematomas is not standardized.⁷ We herein report an unusual case of delayed posttraumatic purely subarachnoid subacute lumbar hematoma with a review of the literature.

CASE DESCRIPTION

A 20-year-old man was referred to the intensive care unit of a peripheral hospital after a very high-kinetic automobile accident. His medical history consisted of surgically managed hematoma of the right

■ **BACKGROUND:** Traumatic spinal subarachnoid hematoma, associated or not with a concurrent subdural hematoma, has rarely been described. The evolution of such hematomas is heterogeneous. This study aims at defining the most accurate management, which is currently not standardized.

■ **CASE DESCRIPTION:** A 20-year-old man, victim of a high-kinetic road accident 5 days before and with several nonneurologic nonsurgical vertebral fractures, experienced a sudden dorsolumbar pain radiating to his lower limbs. A rapidly progressive asymmetric paraparesis with loss of reflexes was noticed, associated with bilateral global hypoesthesia of the lower limbs and with acute urinary retention, whereas the anal tonicity was preserved (American Spinal Injury Association C). Magnetic resonance imaging scan revealed a conus medullaris compression at the level of the L1–L2 vertebrae by an intradural expansive mass. Immediate surgical decompression revealed a strictly subarachnoid hematoma. Venous bleeding was seen at the level of the conus medullaris and controlled. Pathologic examination of the clot excluded an underlying tumor or vascular abnormality. The complete coagulation profile was normal.

■ **CONCLUSION:** Six weeks after surgery, the neurologic examination revealed only a slight tactile hypoesthesia of the left thigh. With only 4 reported cases, purely subarachnoid spinal hematomas remain widely rarer than epidural hematomas. The reported case possesses a certain number of peculiarities: young age, pure subarachnoid location, lumbar location, occurrence after a car accident, subacute onset, and excellent neurologic recovery. In our opinion, a symptomatic subarachnoid spinal hematoma should be surgically evacuated at the early phase so neurologic recovery can be expected.

thigh after minor trauma a few years earlier. He was not taking any medication.

Initial computed tomography of the body showed evidence of thoracic trauma with costal fractures (rib fracture, K10 and K11) and bilateral lung contusions, pelvic fracture (left obturator ring), transforaminal fracture of the sacrum, and several nonneurologic vertebral fractures (Magerl A1 for T9, T10, and L1). None of these traumatic lesions required surgical treatment. In particular, for the vertebral fractures, orthopedic treatment was ordered (immobilization with thoracolumbar corset).

Five days after the accident, while the patient was at bed rest, he described sudden unbearable dorsolumbar pain

radiating to his lower limbs, which had started after a monobloc mobilization for nursing purposes. This pain was poorly relieved by morphine. A rapidly progressive asymmetric severe paraparesis with loss of reflexes was noticed (muscular strength: 2/5 for the right quadriceps, 1/5 for the left quadriceps, 0/5 for distal muscular groups), associated with a bilateral global hypoesthesia of the lower limbs and with acute urinary retention, whereas anal tonicity was preserved (American Spinal Injury Association C). Full spinal magnetic resonance imaging (MRI) revealed a conus medullaris compression at the level of the L1 and L2 vertebrae by an intradural expansive mass whose signal was isointense in



Figure 1. (A) Magnetic resonance image showing spinal cord (cauda equina) compression at the level of L1 and L2 vertebrae by a wide mass that seems to be located in the intradural space rather than the epidural space, appearing isointense in T1-weighted images. (B) Sagittal T2-weighted image showing a hyperintense aspect of the intradural mass. (C) Axial T2-weighted image showing an intradural hematoma widely extended in the spinal canal, pushing the conus medullaris on the right.

T1-weighted images and hyperintense in T2-weighted images (Figures 1A–C). A surgical evacuation was immediately planned without further exploration (notably at the vascular level) and started approximately 5 hours after the onset of the symptoms, taking into account the transfer from the peripheral hospital to our university hospital.

A thoracolumbar spinolaminectomy was performed. The dura mater appeared to be bluish and completely bulged. Under the dura mater, the arachnoid was

extremely stretched, and its opening allowed the evacuation of a strictly subarachnoid hematoma, organized between the cauda equina roots and pushing the conus medullaris to the right (Figures 2A–D). After evacuation of the hematoma, the subarachnoid space was rinsed with warm saline solution. Venous bleeding was identified and rapidly controlled with gelfoam at the level of the conus medullaris. At the end of the procedure, subarachnoid fluid pulsation was restored. The existence of an

underlying tumor or vascular malformation was excluded by pathologic examination of the hematoma. In addition, the peroperative aspect of the vessels was normal. Thus, we estimated that a medullar angiogram was not required.

The postoperative course was particularly favorable, with progressive sensitive and motor recovery. Six days after surgery, while the patient was discharged to a rehabilitation center, sensation in his lower limbs was almost normal, but asymmetrical motor deficit persisted (3/5 for the right lower limb and 2/5 for the left lower limb). Six weeks after surgery, his neurologic recovery was favorable, with normal motor and sphincter functions. The neurologic examination revealed only a slight tactile hypoesthesia of the left thigh. Postoperative MRI performed 6 weeks after the surgical procedure showed no signs of residual spinal cord compression or root compression (Figures 3A–C) or signs of spine instability despite the combination of stable fracture and posterior decompression. Insofar as the patient had a history of surgically managed hematoma of the right thigh after minor trauma a few years earlier, an extensive coagulation profile was studied (blood cell count, blood clotting, blood dyscrasias, intrinsic and extrinsic factors, Von Willebrand factor, antiphospholipid antibodies) but showed no significant anomaly.

DISCUSSION

Generally, the mechanisms that lead to the constitution of subdural and subarachnoid spinal hematomas can be classified into 3 categories^{5,8}: 1) Spontaneous bleeding secondary to vascular or intravascular causes: vascular malformation, hypervascularized tumor, anticoagulant or antiplatelet medications, dyscrasia, hemophilia, collagen vascular disorders^{3,6,9-15}; 2) major injury with sometimes subsequent vertebral fractures⁶ after a fall, spinal surgery,¹⁴ or a car accident⁴; or 3) moderate or minor injury, for instance lumbar puncture,^{6,14} spinal anesthesia contingently potentiated by associated intravascular causes,^{16,17} or acupuncture.¹⁸

To the best of our knowledge, only 4 cases of posttraumatic purely subarachnoid spinal hematomas^{1,14,15,19} (Table 1)

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