Acute clival and spinal subdural hematoma with spontaneous resolution: clinical and radiographic correlation in support of a proposed pathophysiological mechanism

Case report

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Infratentorial and spinal subdural hematomas (SDHs) from traumatic injury in the pediatric population occur with such rarity that they can present the clinician with a challenge in diagnosis and management. When such hematomas are correctly identified, clinicians must decide whether to evacuate the lesion or manage it expectantly. The authors discuss the case of a 4-year-old child who presented with a clival and spinal SDH after a fall from a fourth-story window. The clinical and radiographic findings support a possible mechanism of evolution of these lesions. There is little evidence to guide management of clival and spinal SDHs. This case supports the evaluation for a spinal SDH when a clival hematoma is diagnosed. In the setting of a good neurological examination, expectant management can be an appropriate method of treatment. Additionally, this case lends insight into the pathophysiology of spinal SDHs. Unlike its intracranial counterpart, the spinal subdural space lacks bridging veins. The mechanism of formation of spinal SDHs after trauma has been heretofore relatively unclear. The images in this case support the hypothesis that redistribution of the clival SDH to dependent areas in the spinal subdural space is a significant mechanism in the evolution of these lesions.

KEY WORDS • pediatric trauma • spinal subdural hematoma • clival subdural hematoma • pediatric neurosurgery

Infratentorial and spinal SDHs from traumatic injury in the pediatric population occur with such rarity that they can present the clinician with a challenge in diagnosis and management. Among infratentorial locations, the subdural space between the clivus and the brainstem is an unusual location for hematoma formation. The clival SDH has been associated with traumatic injury, hemophilia, and a fatal outcome. There have also been isolated reports of the appearance of both intracranial and spinal SDHs after traumatic injury in the absence of blood dyscrasia. Treatment options for spinal SDHs vary from urgent operative intervention to watchful management. The mechanism of spinal SDH formation after trauma is unclear. Unlike the intracranial subdural space, the spinal counterpart lacks bridging veins. Presentation with both an intracranial and spinal SDH leads one to question if there is a common source of hemorrhage.

We present the case of a 4-year-old boy who fell from a fourth-story window. Initial imaging revealed a clival SDH that was in continuity with a spinal SDH on the ventral surface of the cervical and upper thoracic spinal cord. Follow-up imaging later revealed spontaneous resolution of the clival component, with a diminished hematoma volume in the spinal subdural space. The series of images suggests that there was redistribution of the clival SDH to more dependent areas in the spinal subdural space.

Case Report

This 4-year-old boy fell from a fourth-story window of an apartment building and landed in a bush. His initial neurological examination at the scene revealed that he was opening his eyes to voice, moving all extremities, and moaning in distress. He was placed on a backboard in a hard collar and transported to the nearest trauma center. There, he continued to demonstrate movement in all four extremities spontaneously; however, his left arm and leg appeared to move less freely than his right. There were brisk reflexes throughout and beats of clonus in the right foot. His pupils were equal and reactive. There was no deficit in extraocular or facial movements.
On arrival at the hospital, he was intubated, sedated, and paralyzed for airway protection. Laboratory analysis revealed a normal platelet count and coagulation profile. Subsequent screening for blood dyscrasia was negative. The initial head CT scan revealed an SDH lying along the clivus, with posterior displacement of the brainstem (Fig. 1). This SDH extended along the tentorium cerebelli. There were no other apparent intracranial abnormalities.

Further MR imaging confirmed the presence of a clival SDH with slight posterior displacement of the brainstem (Fig. 2). The hematoma extended around the cerebellum to the retrocerebellar and supracerebellar space along the tentorium cerebelli. Imaging of the cervical and upper thoracic spine revealed an acute spinal SDH displaying a heterogeneous pattern mostly ventral to the spinal cord, with a small dorsal component in the upper cervical spine (Fig. 3). The spinal SDH was in continuity with the intracranial clival SDH. After this imaging session, approximately 6 hours after the injury, the patient was transferred to our institution. On arrival at our institution, the patient’s neurological examination consisted of eye opening to voice with brisk, spontaneous movement in all extremities, with four-fifths strength in the left arm and leg. Repeated MR imaging 12 hours after the initial one demonstrated complete resolution of the clival component of the SDH, with elements remaining only in the lower cervical and upper thoracic canal ventral to the spinal cord (Fig. 4). Additionally, a significant reduction in the hematoma surrounding the cerebellum had taken place.

The patient also sustained a right distal femur fracture, for which the right lower extremity was splinted. On hospital Day 2, he was extubated. A decrease in hematocrit from 28.7 to 23.5% prompted a CT scan of the abdomen and pelvis, which showed bilateral pleural effusions and peritoneal fluid. There was no obvious intraabdominal organ injury. The hematocrit remained stable without operative intervention. The patient’s left-sided weakness improved during the course of his admission to the point at which his motor strength was equal in both upper extremities after 48 hours. He was discharged from the hospital 1 week after admission in stable condition.

Discussion

The presence of an infratentorial or spinal SDH in a pediatric trauma patient can present a diagnostic and management dilemma. The relative rarity with which SDHs occur in these locations can result in delayed or missed diagnoses. In addition, when they are correctly identified, the clinician may be unsure of how to proceed—whether to evacuate the lesion because of concern for delayed mass effect or to manage the lesion expectantly. This confusion...
can be compounded by the likely scenario in which the patient arrives at the hospital intubated and sedated, without a reliable neurological examination. The pathophysiology of coincidental intracranial and spinal SDHs is unclear; however, the pattern of hemorrhage in this case supports the hypothesis that blood can redistribute from the intracranial to the more dependent spinal subdural space.

Myers, et al.,13 reported a similar case of a clival SDH in a hemophiliac patient after minor trauma with a fatal outcome. An autopsy revealed extension of the subdural hemorrhage into the upper cervical spinal canal. Although this case differs notably from ours in regard to the bleeding disorder present in the former, the two cases are similar in that they suggest that discovery of a clival SDH warrants suspicion of a spinal component and MR imaging of this region.

Treatment of these lesions should be based on a neurological examination of the patient without pharmacological paralytic and sedative agents. Proposed treatments for spinal SDHs include both operative1–3,5,8,9,12,16–18 and nonoperative4,6,7,11 approaches as well as lumbar puncture.10 This report demonstrates successful nonoperative treatment of a patient with a near-normal neurological examination presenting with a large clival SDH with spinal extension. Had the neurological examination instead demonstrated progressive worsening, with weakness or persistent spasticity, this patient would have been considered for an operation. A decompressive laminectomy with dural opening and evacuation of the hematoma would have been performed, which is the procedure that is described in other reports.

This case is one of six reported in the literature that demonstrates the occurrence of both intracranial and spinal SDHs after traumatic injury in the absence of blood dyscrasia.4,6,10,16,18 Of these cases, this report is the first to demonstrate a clival SDH as the intracranial component. Much is unknown about the pathophysiology and natural history underlying the concurrence of these hemorrhages; however, the analysis of patterns of injury in these cases may elucidate more about the mechanism of hematoma formation. This case suggests that intracranial subdural blood can redistribute into the spinal subdural space.

The cause of isolated spinal SDHs is unclear. The most common precipitating factors are blood dyscrasia, anticoagulant therapy, trauma, and lumbar puncture.15 Unlike the intracranial subdural space, the spinal subdural space lacks bridging veins. Rader14 originally proposed that a sudden increase in thoracic and abdominal pressures elevates intravascular pressure in the spinal subdural space, which can then cause rupture of a spinal vessel. The occurrence of both intracranial and spinal SDHs, however, deserves specific considerations regarding the relevant mechanism. Hung, et al.,4 in their report of a boy with a left hemispheric and lumbar spinal SDH, propose that excessive intracranial pressure can increase shearing force between the spinal subdural and subarachnoid spaces, the result of which is tearing and bleeding of the inner dural layer of the spine. On the other hand, Bortolotti, et al.,1 hypothesize that intracranial subdural blood can progressively migrate to more dependent regions in the spine. They also suggest that small arachnoid tears can allow CSF to dilute the intracranial subdural blood, which may result in its spontaneous resolution. In our case, the heterogeneous pattern of the acute spinal SDH on the initial MR image may represent different densities of blood with various amounts of CSF dilution (Fig. 3).

The pattern of hemorrhage and progression of imaging in our case support the redistribution hypothesis proposed by Bortolotti, et al.1 First, the continuity of the clival and cervical spinal SDHs (Fig. 3) confirms the continuity be-

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**Fig. 3.** Sagittal T1-weighted (A) and T1-weighted (B) MR images of the cervical spine revealing acute blood with a heterogeneous pattern in the ventral spinal subdural space. On T1-weighted sagittal (B) and axial (C) images, the hematoma is hypointense. The spinal SDH is in continuity with the clival SDH.
tween the intracranial and spinal subdural spaces that is necessary for this redistribution to occur. Second, on delayed imaging, the resolution of the clival component with a remnant in the spinal subdural space (Fig. 4) suggests further redistribution of blood to more dependent subdural spaces, perhaps with CSF dilution from arachnoid tears.

Conclusions

The diagnosis of an infratentorial or spinal SDH can be a challenge in the setting of the pediatric traumatic injury, especially in the case of a pharmacologically sedated and paralyzed patient in whom a reliable neurological examination was not performed. If imaging of the head reveals a clival SDH, a spinal SDH should be suspected, given the concurrence of these findings in this case and others. A spinal MR imaging study therefore is appropriate to fully evaluate the lesion. The imaging studies in this case suggest a mechanism whereby the initial intracranial hemorrhage migrates to dependent areas in the spinal subdural space, with probable CSF dilution of the blood from concomitant arachnoid tears.

This case has demonstrated that expectant management of a clival and spinal SDH can be a reasonable method of treatment in the setting of a good neurological examination. With expectant management, we have documented a rapid and complete recovery of this patient with radiographic resolution of the hematoma on follow-up imaging.

References


Fig. 4. Sagittal T1-weighted (A) and T2-weighted (B) MR images obtained 12 hours after the initial MR imaging study (Fig. 3), demonstrating resolution of the clival, retrocerebellar, and tentorial components of the SDH. In the lower cervical and upper thoracic spinal subdural space, there are signals that represent remaining hematoma; however, the amount of signal is significantly less than in the initial study (Fig. 3).

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