Rapid Spontaneously Resolving Acute Subdural Hematoma

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Introduction: This study reports a rare patient of a rapid spontaneously resolving acute subdural hematoma. In addition, an analysis of potential clues for the phenomenon is presented with a review of the literature.

Patient Presentation: A 1-year-and-2-month-old boy fell from a height of approximately 2 m. The patient was in a superficial coma with a Glasgow Coma Scale of 8 when he was transferred to the authors’ hospital. Computed tomography revealed the presence of an acute subdural hematoma with a midline shift beyond 1 cm. His guardians refused invasive interventions and chose conservative treatment. Repeat imaging after 15 hours showed the evident resolution of the hematoma and midline reversion. Progressive magnetic resonance imaging demonstrated the complete resolution of the hematoma, without redistribution to a remote site.

Conclusions: Even though this phenomenon has a low incidence, the probability of a rapid spontaneously resolving acute subdural hematoma should be considered when patients present with the following characteristics: children or elderly individuals suffering from mild to moderate head trauma; stable or rapidly recovered consciousness; and simple acute subdural hematoma with a moderate thickness and a particularly low-density band in computed tomography scans.

Key Words: Acute subdural hematoma, rapid spontaneous resolution, traumatic brain injury

Acute subdural hematoma (ASDH) is associated with an overall mortality rate of 50% to 90%, the highest of all traumatic intracranial injuries.1 To avoid further deterioration associated with a secondary injury, current guidelines recommend a craniotomy in patients in whom the ASDH thickness is larger than 10 mm or the midline shift is beyond 5 mm.2 However, rapid spontaneous resolution of ASDH has been observed in a few patients, some of whom even required a craniotomy. An unexpected good recovery was reported for patients who were treated with conservative treatment. Here, the authors present a patient of rapid resolving ASDH with an analysis of the literature.

CLINICAL REPORT

A 1-year-and-2-month-old boy fell from a height of approximately 2 m and lost consciousness after he experienced head trauma. The patient was sent to a local hospital immediately. During the interval emergent computed tomography (CT), the patient vomited several times. The patient’s Glasgow Coma Scale (GCS) score was E2V2M4. The CT scan (Fig. 1A) revealed a right frontotemporal ASDH that was approximately 9 mm in size with a subarachnoid hemorrhage in the longitudinal fissure and midline shift beyond 10 mm. Two hours after the injury the patient was transferred to West China Hospital. He was in a superficial coma with the same GCS score that was documented in previous medical records. Both of the patient’s pupils were equal in size 2 mm but exhibited a blunted response to light. The second CT scan (Fig. 1B) showed a decrease in hematoma density and diffuse cerebral edema with obviously squeezed ventricles; the midline was still deviated compared with the first CT scan. Given his consciousness and imaging findings, intracranial pressure monitoring was preferred. Even though the laboratory features were not surgical contraindications, the boy’s guardians refused the placement of an intracranial pressure probe or a craniotomy. Conservative treatment was agreed upon in the neurointensive care unit. General monitoring aimed to maintain SaO2 at >95%, mean systolic blood pressure at >90 mm Hg, temperature at <37°C, and PaCO2 at 35 to 40 mm Hg. Owing to the lack of an accurate intracranial pressure assessment, mannitol was administered empirically at 0.5 g/kg every 6 hours. The third CT scan (Fig. 1C) revealed evident resolution of the hematoma and midline shift improvement, with only a subarachnoid hemorrhage present in the posterior longitudinal fissure 24 hours after the trauma. After he had received 2 days of conservative management,

FIGURE 1. Acute subdural hematoma evolution in sequential CT scans. (A) Initial CT scan revealed right frontotemporal acute subdural hematoma (arrow to show), with SAH in anterior longitudinal fissure and midline shift exceeding 10 mm. (B) A second CT scan showed the decrease of hematoma (arrow to show) with obvious diffusion cerebral edema. (C) A third CT scan was taken after 24 hours of the injury revealed an evident resolution of hematoma (arrow to show) and midline revert with only SAH left in posterior longitudinal fissure. CT, computed tomography; SAH, subarachnoid hemorrhage.

FIGURE 2. Magnetic resonance imaging on the fourth day of conservative management. Transverse (A), sagittal (B), and coronal (C) planes were performed which revealed acute subdural hematoma complete resolution without redistribution to upper cervical spinal cord.
TABLE 1. Summary Literatures of Acute Subdural Hematoma With Rapid Resolution

| Author             | Age | GCS | Initial Conc. | Change | Resolution Time | ASDH Thickness | ASDH Region | Low-Density Band | Shift of Midline | SAH | Contusion | Edema | Redistribution |
|--------------------|-----|-----|---------------|--------|-----------------|----------------|-------------|-----------------|-----------------|-----|-----------|-------|----------------|
| Polman et al       | 26 y| 15  | Deteriorated  |        | 6 h             | <15 mm         | F-T-P       | No              | >10 mm          | Yes | No        | Yes   | No             |
| Nagao et al        | 3 y | 9   | Improved      |        | 16 h            | <15 mm         | F-T-P       | No              | >10 mm          | No  | No        | No    | No             |
| Matsuyma et al     | 18 y| 9   | Improved      |        | 3 h             | 15 mm          | F-T         | Yes             | >10 mm          | No  | No        | No    | No             |
| Kato et al         | 70 y| 15  | Stable        |        | 12 h            | <15 mm         | F-T         | Yes             | No              | Yes | No        | No    | Yes            |
| Kato et al         | 41 y| 11  | Improved      |        | 12 h            | <15 mm         | F-T         | Yes             | >10 mm          | No  | No        | Yes   | No             |
| Berker et al       | 57 y| 7   | Improved      |        | 2 h             | <15 mm         | F-T         | No              | >10 mm          | No  | No        | No    | No             |
| Erol et al         | 1 y | 15  | Stable        |        | 36 h            | <15 mm         | F-T         | Yes             | No              | No  | No        | No    | No             |
| Huang et al        | 2 y | 14  | Stable        |        | 65 h            | <15 mm         | F-T         | Yes             | >10 mm          | No  | No        | No    | No             |
| Kapsalka et al     | 29 y| 8   | Stable        |        | 6 h             | >15 mm         | F-T         | Yes             | >10 mm          | Yes | No        | No    | No             |
| Cosar et al        | 8 y | 15  | Stable        |        | 9 h             | <15 mm         | F-P         | Yes             | No              | No  | No        | No    | No             |
| Wong et al         | 73 y| 8   | Improved      |        | 45 h            | >15 mm         | F-T-P       | Yes             | >10 mm          | No  | No        | No    | Yes            |
| Yadv et al         | 70 y| 6   | Improved      |        | 7 h             | <15 mm         | F-T         | Yes             | <10 mm          | Yes | No        | No    | No             |
| Shih et al         | 40 y| 6   | Improved      |        | 72 h            | <15 mm         | F-T-P       | Yes             | >10 mm          | No  | No        | No    | No             |
| Liu et al          | 2 y | 6   | Stable        |        | 0.5 h           | <15 mm         | F-T         | Yes             | >10 mm          | Yes | No        | No    | No             |
| Park et al         | 7 y | 5   | Stable        |        | 6 h             | <15 mm         | F-T-P       | Yes             | >10 mm          | No  | No        | Yes   | No             |
| Liu et al          | 48 y| 7   | Improved      |        | 24 h            | <15 mm         | F-T         | Yes             | <10 mm          | Yes | No        | No    | No             |
| Balik et al        | 66 y| 6   | Deteriorated  |        | 16 h            | <15 mm         | F-T         | Yes             | <10 mm          | Yes | No        | No    | Yes            |
| Present patient 1  | 1 y | 8   | Stable        |        | 15 h            | <15 mm         | F-T         | Yes             | >10 mm          | Yes | No        | Yes   | No             |

ASDH, acute subdural hematoma; F, frontal; GCS, Glasgow Coma Scale; P, parietal; SAH, subarachnoid hemorrhage; T, temporal.

the boy regained consciousness. The magnetic resonance images (Fig. 2A–C) showed that the ASDH was almost completely resolved on the fourth day after admission, without redistribution to the upper cervical spinal cord. The patient was finally discharged on the seventh day without neurological function impairment.

**DISCUSSION**

The following 2 possible explanations were proposed: the ASDH was diluted and washed out by cerebrospinal fluid (CSF) after the tearing of the arachnoid membrane and the hematoma was redistributed to the subdural space. Wong et al. measured the Hounsfield units of the arachnoid membrane that is compared to brain tissue and narrow compensatory space results in an arachnoid membrane slit under external force. For middle-age individuals, brain atrophy and a wide cavum of the subarachnoid space can cause an arachnoid membrane to be torn easily in children. In elderly individuals, brain atrophy and a wide cavum of the subarachnoid space can cause an arachnoid membrane slit under external force. For middle-age individuals, we speculate that cerebral edema leads to resolution via both mechanisms, but it may not be the prerequisite for hematoma rapid resolution. Kuroiwa et al. suggested that diffuse cerebral edema was necessary for the spontaneous resolution of a hematoma. We speculate that cerebral edema leads to resolution via both mechanisms, but it may not be the prerequisite for hematoma rapid resolution. Indeed, in most patients, the diffuse edema was not even observed (Table 1). We propose that the slice thickness of the arachnoid membrane that is comparatively difficult to tear with a mild injury. Wen et al. proposed that GCS was higher than 8 in patients who exhibited this phenomenon. However, in Table 1, 7 of 18 patients exhibited a GCS score of less than 8, and the minimum was 5. In the process of the clinical evaluation, the dynamic change in consciousness is a more heavily weighted factor to consider than the initial GCS score. According to a review of all patients (Table 1), most of those patients regained consciousness quickly. Although some of those patients did not regain consciousness quickly, their conditions were stable. In our case, the patient’s consciousness improvement was even slower than the time of resolution according to the CT scan. Thus, by comparing the initial GCS score after head trauma, we inferred that a rapid recovery or relative stabilization of consciousness is more persuasive in these patients.

The evaluation of images from CT scans that were performed during different phases of our patient clearly demonstrated the rapid transition of hematoma density from a high density to a mixed density. This finding indicated that CSF dilution probably occurred during this phenomenon. Some authors agreed that the potential sign in early CT of a rapid resolution was the presence of a low-density band. In our patient’s second CT scan (Fig. 1B), it was observed that remarkable cerebral edema could squeeze, in some way, the dilated hematoma. Kuroiwa et al. suggested that diffuse cerebral edema was necessary for the spontaneous resolution of a hematoma. We speculate that cerebral edema leads to resolution via both mechanisms, but it may not be the prerequisite for hematoma rapid resolution. Indeed, in most patients, the diffuse edema was not even observed (Table 1). We propose that the slice thickness of the arachnoid membrane (<1.5 cm) would be suitable in the consideration of rapid resolution. Lee et al. reported a patient with a hematoma thickness that was larger than 2.5 cm, yet it finally converted to a chronic subdural hematoma. The presence of simple ASDH without contusion appears to be associated with rapid resolution. We suspect that this effect is due to the mass effect of the contusion, which impeded the flow of CSF in the limited space.

**CONCLUSION**

This patient has been reported because of clear evidence of CSF dilution and a flush effect that led to resolution of the hematoma, and the finding that a distinctly swollen brain probably accelerated the process. There is no need for the deliberate evaluation of the phenomenon as a coincidence during treatment. We suggest that ASDH rapid resolution should be considered when patients present...
with the following characteristics: children or elders suffering from mild to moderate head trauma; stable or rapidly recovered consciousness; and simple ASDH with moderate thickness and a particularly low-density band in CT scan images.

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Open Reduction in Pediatric Condylar Fracture
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Abstract: Facial fractures in children are rare. Lack of pneumatization, fat pockets, mixed dentition, contribute to the elasticity and bone stability. When mandibular fractures occur in children, most often involve the condyle by indirect trauma. Such fractures are the center of discussion on the form of treatment if this should be performed conservatively, or held the reduction and fixation of the fracture with surgical exposure of the fragments. In condylar fractures in children, in most patients, the proposed treatment is closed reduction. Treatment with open reduction and fixation has its specific indications. In this case, the authors report a patient cycling accident victim, with cut-contrusion injury in ment with limited mouth opening and left condylar fracture with medial rotation. The treatment was the reduction and fixation of fragments by open process.

Key Words: Condylar fracture, pediatric fracture, facial trauma

Facial trauma affecting children displays important features regarding the incidence, diagnosis, and treatment, which leads the child facial trauma to be object of special attention to the psychological and physiological conditions, own age. Pediatric fractures are unusual compared with fractures in adults. Most of the time children are in protected environments under the supervision of parents and therefore less exposed to major trauma. Lack of jaw pneumatization, the considerable number of pockets of fat, bone developing, and mixed dentition contribute to the elasticity and bone stability, requiring more force to produce a facial fracture and reflecting its low incidence of facial skeletal. The index of facial fractures in pediatric patients according to studies is around 15%, occurring mostly in the age group over 5 years. Mandibular fractures in children represent 32.7% of all facial fractures, followed by nasal fractures (30.2%) and midface and zygomatic bone fractures (28.6%). Mandible fractures are rare in children under 5 years.1,2 The condyle fracture, in most patients, incomplete type “green stick,” generally aged less than 6 years of age, where the bone is more flexible.3 The fractures of the mandibular condyle are in about 11% to 16% of facial fractures and 30% to 40% of all mandibular fractures. Most are not caused by direct trauma, but following indirect forces transmitted to the condyle by a blow elsewhere.4 Boys are more commonly affected than girls in all age groups; this has been attributed to the most dangerous physical activity experienced by boys.5,6 The fractures of the mandibular condyle in children can lead to serious problems such as malocclusion, temporomandibular dysfunction, TMJ ankylosis, interference in mandibular growth.5,6 From the 1Postgraduate Program in Dental Sciences; and 2School of Dentistry, Federal University of Alfenas, Minas Gerais, Brazil. Received September 30, 2016. Accepted for publication December 8, 2016. Address correspondence and reprint requests to Ronaldo Célio Mariano, PhD, Rua Gabriel Monteiro da Silva, 700 Centro, Alfenas, MG CEP:37.130-000, Brazil; E-mail: ronaldocmariano@gmail.com The authors report no conflicts of interest. Copyright © 2017 by Mutaz B. Habal, MD ISSN: 1049-2275 DOI: 10.1097/SCS.000000000003538

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