Case Report

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Coexistence of Rapidly Resolving Acute Subdural Hematoma and Delayed Traumatic Intracerebral Hemorrhage

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Key Words
Delayed traumatic intracerebral hemorrhage · Subdural hematoma

Abstract
Rapid resolution of acute subdural hematoma is rare. Delayed traumatic intracerebral hematomas following medical or surgical treatment of increased intracranial pressure have also been reported. Coexistence of a quickly resolving acute subdural hematoma and a delayed traumatic intracerebral hemorrhage has not been reported before. A 13-month-old boy was admitted to our emergency department after a car accident. On CT, a thin acute subdural hematoma on the right frontotemporal region and a small epidural hematoma on the left frontal region could be seen. On 24-hour follow-up CT, the right subdural hematoma was found to be less dense but larger than it had been before. At 36 h after hospitalization, CT showed that the right acute subdural hematoma had completely disappeared; however, a delayed traumatic intracerebral hematoma on the left occipital region was identified. We think that the mechanism involved in the development of a delayed intracerebral hematoma in our case was similar to the one causing delayed traumatic intracerebral hematoma after treatment for increased intracranial pressure.

Introduction

Acute subdural hematoma in the pediatric age develops almost always due to a minor head trauma following a motor vehicle accident [1]. It correlates with the severity of the trauma and develops after laceration of the bridging veins between the cortex and the dural sinuses (due to the fact that the sinuses stay fixed but the brain adapts to acceleration) [2]. Spontaneous resolution of these hematomas usually takes place within weeks or months; rapid resolution is quite rare [3–5]. We present a case of acute subdural hematoma in the right frontotemporal region that rapidly resolved within 36 h and coexisted with a delayed traumatic intracerebral hematoma in the left occipital lobe.

Case Report

A 13-month-old boy was admitted to our emergency department after a car accident. The initial examination was unremarkable except for a skin laceration extending from the frontal hairline to the lateral orbital rim. The anterior fontanel was open and the neurological and other systemic examination findings were normal. The boy had a previous history of iron deficiency anemia, and laboratory examination revealed the hematocrite level to be 28% and hemoglobin at 8.9 mg/dl. On plain cranial roentgenograms, a linear fracture line was identified emerging from the frontal region and extending to the left temporal region. CT scan showed a thin acute subdural hema-

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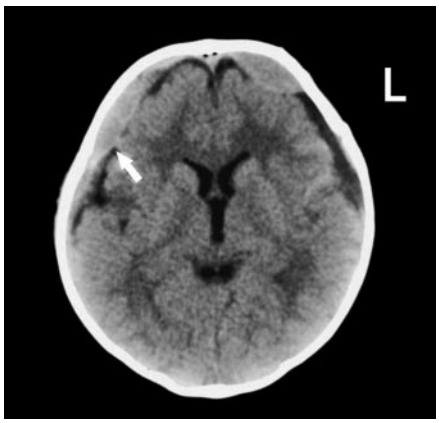

Fig. 1. On the initial CT immediately after the trauma, an isodense small epidural hematoma on the left and an isodense acute subdural hematoma with no mass effect (white arrow) on the right side are delineated.

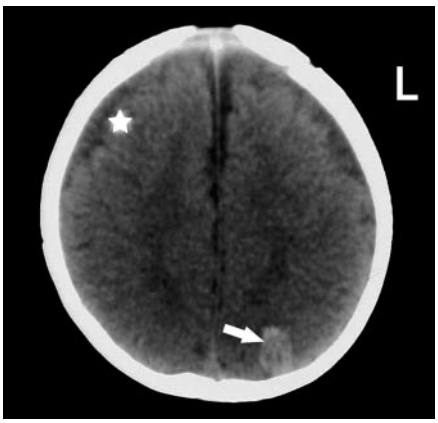

Fig. 3. Thirty-six hours after hospitalization the acute subdural hematoma (white star) has disappeared but the delayed traumatic intracerebral hematoma in the left occipital region can be seen (white arrow).

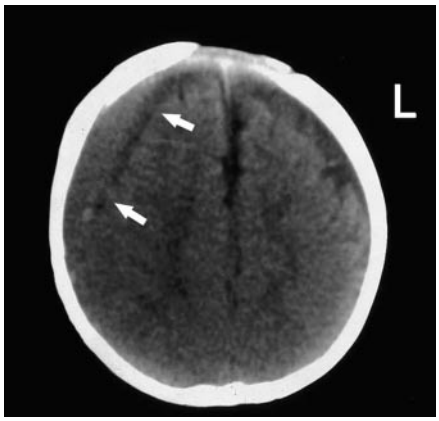

Fig. 2. Twenty-four hours after hospitalization, CT shows a decrease in density but an increase in volume (double white arrow) of the acute subdural hematoma on the right side.

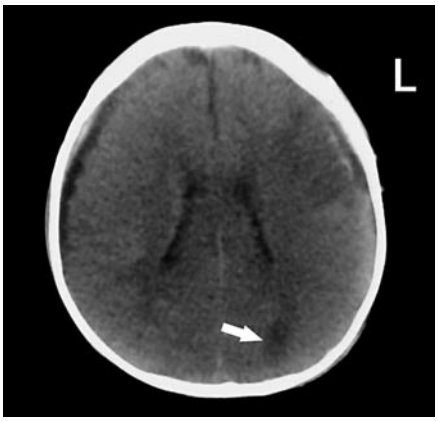

Fig. 4. On 1-month follow-up CT, the delayed traumatic intracerebral hematoma has been completely resorbed and an infarct lesion can be seen (white arrow).

toma on the right frontotemporal and a small epidural hematoma on the left frontal region (fig. 1). The patient was hospitalized and on 24-hour follow-up CT, the right subdural hematoma was found to be less dense but larger than it had been before. Compression of the brain parenchyma due to the hematoma mass effect and development of a cerebral edema, particularly in the right parietooccipital region, were observed as well (fig. 2).

Antiedema therapy (mannitol, furocemide, intermittent oxygen) was begun to reduce the size of the hematoma. At 36 h after hospitalization, a partial epileptic seizure involving only the right upper extremity was observed and CT was repeated. The right acute subdural hematoma was found to have completely disappeared; however, the presence of a delayed traumatic intracerebral hematoma on the left occipital region was noted (fig. 3).

Diphenylhydantoin was given for the epileptic seizures. No deterioration in the neurological status or recurrences in the seizures were observed, antiepileptic therapy was regulated and the patient discharged. At 1-month follow-up the left occipital hematoma had disappeared (fig. 4).

Discussion

The mechanism in the rapid resolution of acute subdural hematomas is not well known. However, there is consensus about the two basic stages of the rapid resolution process [3, 6]: (1) hematomas are diluted by the cerebrospinal fluid (CSF) that egresses into the hematoma space as a result of the torn arachnoid membrane, and (2) acute cerebral edema contributes to an increase in intracranial pressure, thus promoting its dilution and facilitating the liquidization of the hematoma which eventually interferes with CSF circulation and the venous compartment. However, it has been reported that any intracerebral hematoma or contusion which is likely to block the foramina and obstruct CSF circulation should not occur at the same time [3]. On 24-hour follow-up CT, a decrease in density and an increase in the size of the subdural hematoma was identified. This image suggested that the arachnoid tear had caused CSF to egress into the hematoma space and dilute the hematoma, thereby decreasing the density but increasing the volume. Besides, as visible on the patient's initial CT, acute subdural hematomas, although rarely reported, can be isodense in some patients with low hematocrite levels because of preexisting anemia [7]. The dilution of the hematoma by CSF also facilitated hematoma flow and absorption, i.e. the rapid spontaneous resolution.

Delayed traumatic intracerebral hematomas are frequently encountered after countercoup mechanisms [8]. Diagnosis is usually performed with serial CTs once deterioration in neurological status and/or increase in intracranial pressure have become apparent [9]. In our case, the diagnosis was made by CT performed after the development of an epileptic seizure and a hematoma was found on the countercoup occipital lobe. The most significant point in the development of these hematomas is the physical damage to the vessels and to their supportive neural structures after trauma [8]. Necrosis quickly affects these vessels. When treating the increased intracranial pressure medically or surgically, perfusion pressure is restored and the necrotic vessels rupture, thus causing delayed intracerebral hematomas [10, 11].

In our case, a mechanism similar to the one reported in the development of delayed cerebral hematoma following treatment for increased intracranial pressure was thought to take place when the delayed intracerebral hematoma developed after the spontaneous rapid resolution of the acute subdural hematoma. Delayed traumatic intracerebral hematomas develop within the first 72 h, particularly the first 48 h. We believe that the disappearance of the blocking effect (due to the rapid resolution of the acute subdural hematoma) aided in the development of the delayed traumatic intracerebral hematoma. The fact that the delayed hematoma developed on the countercoup side of the trauma once the acute subdural hematoma located in the trauma-affected region had resolved supports our hypothesis.

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