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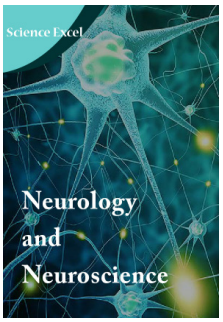
Review Article



Mild-Type Infantile Acute Subdural Hematoma Followed by Subdural Hematohygroma: Report of A Case Suggesting Common Etiology Causing Acute Subdural Hematoma and Hematohygroma

Nobuhiko Aoki

[1]Department of Neurosurgery, Bethlehem Garden Hospital, Tokyo, Japan
[2]Department of Neurosurgery, Tokyo Metropolitan Tama Medical Center, Tokyo, Japan

*Correspondence
Nobuhiko Aoki
Department of Neurosurgery, Bethlehem Garden Hospital, 3-14-72 Umesono, Kiyose, Tokyo 204-0024, Japan
Tel: (+81) 42-401-2525
Fax: (+81) 42-495-2878

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Abstract

Background: *Infantile acute subdural hematoma (IASDH) and subdural hematohygroma (SDHy) have been seemingly closely related disorders, to date, however, there have been poorly elucidated concerning the etiological relationship. The present case report aimed to approach possible etiology commonly developing both disorders.*

Case description: *A one month-old girl suffered fever associated with poor milk feeding. Because these symptoms failed to subside and the diagnosis was undefinitive, she was transiently hospitalized for sepsis workup. Head CT revealed focal enlargement of subarachnoid space (SAS), and MRI 9 days later showed benign enlargement of subarachnoid space.*

Two months later, however, she was noted to have generalized convulsive seizure, prompting to refer to a nearby hospital. At the emergency room, although she was alert and asymptomatic, head CT revealed acute subdural hematoma (ASDH). Ophthalmological examination failed to reveal retinal hemorrhage. MRI 9 days later disclosed disappearance of ASDH and newly developed bilateral SDHy. Her clinical course was uneventful, and follow-up CT at the age of 4 months showed enlargement of subarachnoid spaces on both frontotemporal regions and disappearance of SDHy.

Discussion and conclusion: *In the present case, clinical course and serial neuroimaging findings, particularly CT and MRI at the age of one month undergone as routine sepsis workup without neurological events, are invaluable to elucidate the pathological similarity between IASDH and SDHy.*

Second CT taken after seizure showed thin film-like ASDH associated with enlargement of SAS, typical features seen in mild-type IASDH. On the other hand, second MRI 9 days after CT revealed disappearance of ASDH, and disclosed bilateral SDHy associated with benign enlargement of subarachnoid space. This phenomenon indicates early disappearance of mild-type IASDH together with progressive enlargement of SAS probably leading to development of SDHy.

Keywords

Infantile acute subdural hematoma, Subdural hematohygroma, Benign enlargement of subarachnoid space, Large sylvian fissure, Arachnoid rupture

Introduction

Infantile acute subdural hematoma (IASDH) and subdural hematohygroma (SDHy) have been positioned as closely related disorders, to date, however, the etiological relationship is poorly elucidated. The immaturity of the cerebrospinal fluid (CSF) dynamics in infancy has been reported as possible causative pathology, proceeding to accumulation of CSF in subarachnoid space (SAS) [1,2].

This phenomenon is mostly observed in infants between the age of 6 to 10 months in IASDH after mild head impact [3-5]. On the other hand, SDHy has been revealed in early infancy within a few months after birth, and most of them are non-traumatic in origin and associated with favorable outcome [6,7]. The present case report aimed to approach possible pathoetiology commonly causing both disorders (i.e.,IASDH and SDHy).

Case presentation

A one month-old girl with uncomplicated delivery followed by smooth postpartum clinical course suffered fever associated with poor milk feeding. Because these symptoms failed to subside and the diagnosis was undetermined, she was transiently hospitalized for sepsis work-up. Head CT revealed focal enlargement of SAS, and bilateral large sylvian fissure (LSF) (Figure 1). MRI taken 9 days later showed benign enlargement of subarachnoid space (BESS) and bilateral LSF (Figure 2). No cerebral parenchymal abnormality was noted. Although her symptoms resolved spontaneously, two months later at the age of 3 months she suffered a generalized convulsive seizure, prompting to refer to a nearby hospital. At the emergency room where she was alert and asymptomatic, head CT revealed acute subdural hematoma (ASDH), and enlargement

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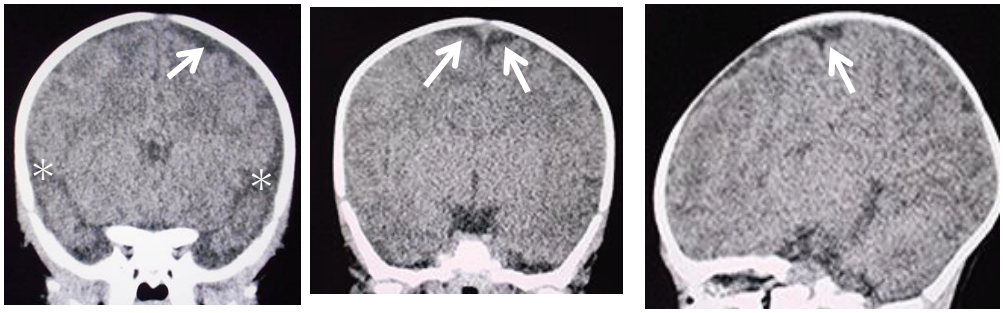

Figure 1. CT taken for sepsis workup at the age of one month

Left (coronal view): Enlargement of subarachnoid space on the left frontal region (arrow) and large sylvian fissure on both sides (asterisks)
Center (coronal view): Bilateral enlargement of subarachnoid space at the parasigittal regions (arrows)
Right (sigittal view): Enlargement of subarachnoid space at parietal region

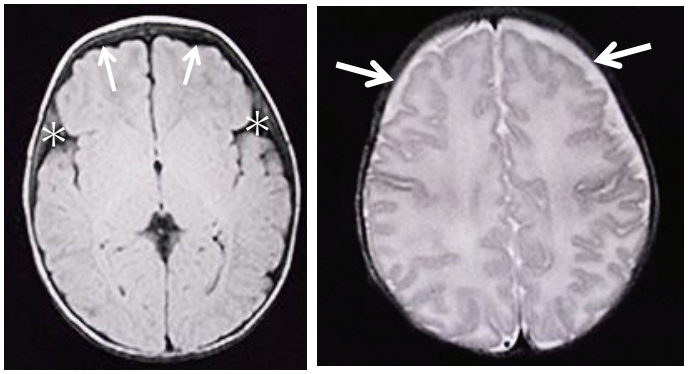

Figure 2. MRI 9 days after CT

Left fluid-attenuated inversion recovery: Bilateral enlargement of subarachnoid spaces on frontal regions (arrows) Large sylvian fissure on both sides (asterisks)
Right (T2WI): Bilateral enlargement of subarachnoid spaces on frontal regions (arrows)

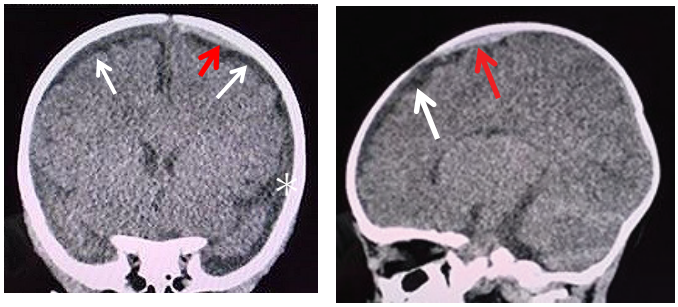

Figure 3. CT at the age of 3 months, presenting with a seizure

Left coronal view): Thin film-like acute subdural hematoma (red arrow). Enlargement of subarachnoid space on both sides (white arrows) Large sylvian fissure on the left side (asterisk)
Right (sigittal view): Thin film-like acute subdural hematoma at the parietal region (red arrow). Enlargement of subarachnoid space at the frontal region (white arrow)

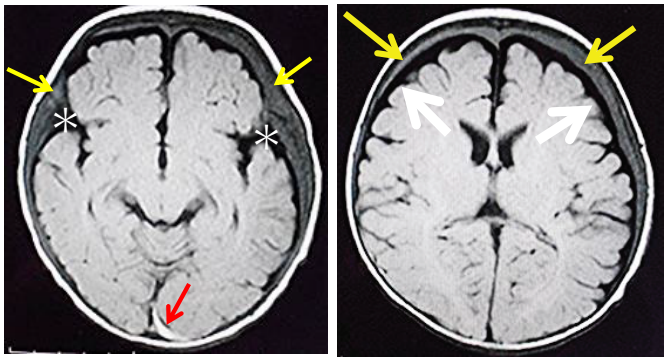

Figure 4. MRI (fluid-attenuated inversion recovery) 9 days after the seizure, at the age of 3 months

Left : Bilateral subdural hematohygroma on frontal regions (yellow arrows). Large sylvian fissure on both sides (asterisks). Subdural hematoma on the left occipital region (red arrow), showing gravitational sedimentation of hematohygroma.
Right : Subdural hematohygroma on both frontal regions (yellow arrows). Bilateral enlargement of subarachnoid spaces, coinciding with benign enlargement of subarachnoid spaces (white arrows).
Note, no cerebral parenchymal abnormality (including other sequences, not shown)

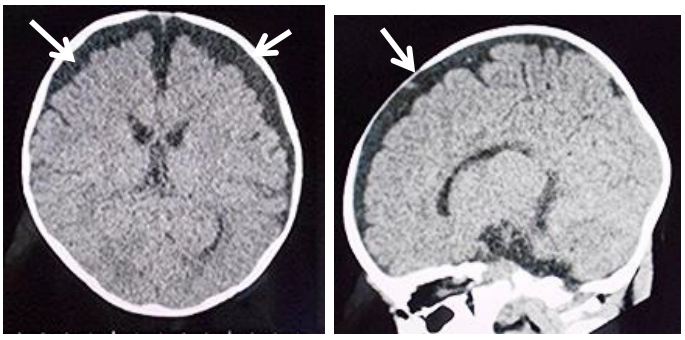

Figure 5. CT at the age of 4 months

Left: Axial view Right : Sagittal view
Bilateral enlargement of subarachnoid spaces on frontotemporal regions (white arrows)
Note, disappearance of hematohygroma

of subarachnoid space with LSF on both sides (Figure 3). Ophthalmological examination revealed no retinal hemorrhage on both sides. MRI 9 days later disclosed disappearance of ASDH and newly developed SDHy (Figure 4). Her further clinical course was uneventful, and follow-up CT at the age of 4 months showed enlargement of subarachnoid spaces on both frontotemporal regions, and disappearance of SDHy (Figure 5)

Her 3 years follow-up neurological examinations confirmed to have normal developmental milestones.

Discussion

As noted earlier, the immaturity of CSF dynamics induce age-regulated accumulation of CSF in the SAS [1,2]. This phenomenon is mostly observed in infants between the ages of 6 and 10 months as observed in IASDH after mild head impact [3-5]. On the other hand, SDHy has been revealed in early infancy within a few months after birth, and most of them are non-traumatic in origin and associated with favorable outcome [6,7]. In the present case, clinical course and serial neuroimaging findings, particularly CT and MRI at the age of one month taken as routine sepsis workup without neurological events, are invaluable to elucidate the patho-etiological similarity between IASDH and SDHy.

Second CT taken after a seizure showed thin film-like ASDH associated with enlargement of SAS, showing typical features seen in mild-type IASDH [4]. On the other hand, second MRI 9 days after CT revealed disappearance of ASDH, and disclosed SDHy on both sides associated with BESS [6,7]. This phenomenon may indicate early disappearance of mild-type IASDH together with progressive enlargement of SAS probably leading to development of SDHy.

Both clinical entities (i.e., IASDH and SDHy) may be commonly originated from progressive accumulation of CSF in the subarachnoid space. First, in early infancy mainly at the age of 1 to 3 months, SDHy is prone to occur due to rapid progression of CSF collection by which arachnoid membrane destined to rupture even without head impact [7]. Then, second, at the age of 6 to10 months, IASDH are observed mostly due to mild head impact causing arachnoid tear together with rupture of bridging veins [3-5].

Both subdural collections are composed of CSF mixed with blood in different ratio and develop in different age groups, and are identically followed by benign clinical course.

Conclusion

As speculated in the present case, it is plausible to consider that IASDH and SDHy are commonly originated from age-regulated immaturity of the CSF dynamics causing progressive accumulation of CSF in SAS (i.e., cranio-cerebral disproportion; CCD). Intracranial structural vulnerabilities secondary to CCD may be followed by IASDH in middle aged infants (peak 8 -10 months) after mild had trauma [3-5], and SDHy in early infancy even without head trauma [7].

Author contribution

Nobuhiko Aoki (corresponding author with no coauthors) conceived the study, conducted a search of the literature, and drafted the original manuscript. The author has reviewed the manuscript draft, revised it critically for intellectual content, and approved the final version for submission.

Availability of data and materials

None of the data or material can be provided

Declarations, ethics approval, and consent to participate

This article was approved by the ethics committee of Bethlehem Garden Hospital and Tokyo Metropolitan Tama Medical Center. No funding was obtained for this study. The parents/legal guardians of the patient provided their consent to publish the details of this case.

Consent to publish

The author consents to the publication of all identifiable details, which may include photographs, case history, and other details.

Conflict of Interests

None

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