

Acute Subdural Hematoma in Infants with Abusive Head Trauma: A Literature Review

Hiroshi KARIBE,¹ Motonobu KAMEYAMA,¹ Toshiaki HAYASHI,¹
Ayumi NARISAWA,¹ and Teiji TOMINAGA²

¹*Department of Neurosurgery, Sendai City Hospital, Sendai, Miyagi*

²*Department of Neurosurgery, Tohoku University Graduate School of Medicine,
Sendai, Miyagi*

Abstract

The number of cases with child abuse is increasing in Japan, and abusive head trauma (AHT) is a major cause of death in abused children. Child abuse has been recognized by the late 19th century, and widely accepted as battered child syndrome in the middle of the 20th century. As terms, there had been considerable mechanistic controversies between shaken-baby and -impact syndrome until the beginning of the 21st century. In recent years, AHT has been utilized as a less mechanistic term. Most of the characteristics of AHT in Japan have been similar to those in the United States as follows: infant is the most common victim, acute subdural hematoma (SDH) is the most common intracranial lesion, and retinal hemorrhage is often complicated. On the other hand, several characteristics have been different as follows: mother is the most common perpetrators, impact is a more common mechanism of trauma than shaking, and external trauma is more common reflecting the existence of impact. Since AHT as well as child abuse is a social pathological phenomenon influenced by victims, perpetrators, socioeconomic circumstances, and so on, various aspects of AHT as well as child abuse can be changed with times. Actually, a recent paper suggests such changes in infants with acute SDH due to AHT. In this review article, AHT, abusive infantile acute SDH in particular, are reviewed from the aspect of neurosurgical perspectives, including its mechanisms of trauma, biomechanics, clinical features, management, and prognosis, to update the trend in Japan.

Key words: acute subdural hematoma, abusive head trauma, infant

Introduction

Abusive head trauma (AHT) is a major cause of severe head injury in infants,¹ as 64% of all head injuries or as 95% of serious intracranial injuries.² From another point of view, physical abuse is common among child abuse. In physical abuse, the most common cause of mortality is head injury represented by acute subdural hematoma (SDH).³ In addition to acute SDH, AHT has been classically characterized in North America as follows: infant is the most common victim, shaking or shaking-impact is the most common mechanism of trauma, retinal hemorrhages are commonly complicated, male, including fathers and mother's boyfriends, are more common perpetrators than female, and so on.⁴

In Japan, it has been estimated that the number of cases with child abuse is increasing. According

to a statistic report from the Japan Ministry of Health, Labor, and Welfare, approximately 66,000 cases of child abuse were consulted at child welfare institutions in 2013. The number of child abuse was 5.7 times larger since the proclamation of the Child Abuse Prevention Act in 2000. Most of the clinical characteristics of AHT in Japan have been similar to those in North America, however, several characteristics have been different as follows: mother is the most common perpetrators, impact is a more common mechanism of trauma than shaking, and external trauma is more common reflecting the existence of impact.⁵ Child abuse may be a social pathological phenomenon influenced by the victims, the perpetrators including parents and other caregivers, socioeconomic circumstances, and so on, suggesting that various aspects surrounding child abuse as well as AHT have potentials to change with time.⁶ Actually, a recent paper suggests such changes in infants with acute SDH due to AHT.⁷ These facts

suggest that participants for the management and treatment of abused children may have to update the trends of the characteristics concerning AHT.

In this review article, child abuse is reviewed from the aspect of neurosurgical perspectives, including its mechanisms, clinical features, management, and prognosis, including the trends of infantile acute SDH due to AHT in Japan.

Historical Overview and Terminology

Child abuse has been recognized by the late 19th century, at the latest. Caffey reported six cases with multiple long-bone fractures in infants suffering from chronic SDH, to provide attention to arousing child abuse as a cause of trauma.⁸⁾ In 1962, Kempe et al. reported multiple cases of abused child as battered child syndrome.⁹⁾ Since then, the concept of child abuse has become widely recognized. In 1971, Guthkelch reported five cases with infantile SDH caused by acceleration/deceleration force by shaking.¹⁰⁾ In 1974, Caffey proposed the concept of whiplash shaken infant syndrome,¹¹⁾ the so-called shaken baby syndrome (SBS). The concept was that shaking an infant's head by holding the arms or body trunk caused SDH and retinal hemorrhage without signs of external trauma to the head. In 1990, American Academy of Pediatrics gave warning for violent shaking of the head to be prevented, since it caused intracranial injuries and subsequent serious impairments.¹²⁾

It was also suggested that not only shaking but also sudden deceleration associated with forceful striking of the head against a surface, was responsible for most of the severe inflicted brain injuries, the so-called shaken impact syndrome (SIS).^{4,13)} Considerable controversies might exist between SBS and SIS in their biomechanical concepts, however, general consensus might have been obtained in most part of this matter as follows: (1) SBS/SIS was seen more commonly in infants. (2) SBS/SIS was diagnosed by the presence of diffuse brain swelling, subdural hemorrhage, and retinal hemorrhages in the absence of documented extraordinary blunt force. Subarachnoid hemorrhage, diffuse axonal injury (DAI), and progression toward computed tomography (CT) images of "black brain" were seen in some cases, in addition to the classic triad above. (3) Cerebral edema and resultant neurological symptoms commence within a short time after shaking. (4) Primary injuries to the brain caused the signs and symptoms to determine the course of SBS/SIS. (5) Retinal hemorrhages occurred in the vast majority cases of SBS/SIS. (6) Signs of external trauma to the head and body might or might not be present.

(7) Classic metaphyseal lesions of the long bones and posterior rib fractures might be seen in some cases. (8) Injuries to the neck muscles or cervical vertebrae were distinctly uncommon. (9) The injuries were caused by violent assault on the infant by the perpetrator and are not the result of rough play or short falls. (10) Esoteric disease states, such as osteogenesis imperfecta and glutaric acidemia, could be distinguished from inflicted injury. (11) SBS/SIS could be a single event in a child's life or it could be a series of events. (12) Based on limited information on outcome, 15–30% of SBS/SIS victims died, while the survivors were normal in less than 10% of the cases.¹⁴⁾

In recent years, it has been suggested that AHT can occur not only by violent shaking of the head but also by other various mechanisms.¹⁵⁾ American Academy of Pediatrics recommends that a less mechanistic term AHT, should be embraced, when describing an inflicted injury to the head and its contents.¹⁶⁾

Epidemiology

It has been reported that the AHT is largely restricted to children under 3 years of age. The majority of cases occur during the first year of life.^{4,17,18)} In a study of admitted children under 2 years of age with head injuries, 24% of injuries resulted from inflicted trauma.^{2,19,20)} Such children commonly have evidence of previous abuse.²¹⁾ In cases with traumatic intracranial hemorrhage, 68.6% of them occur in infants, 25.7% occur in 1–3 y.o., and 5.7% occur in 4 y.o. or older.

In Japan, AHT had been sparsely reported until 1990s.²²⁾ Since then, the number of cases with child abuse increased rapidly to exceed more than 10,000 cases in 2000, becoming a serious social problem. According to the recent statistic report from the Japan Ministry of Health, Labor, and Welfare, the number of cases with child abuse, processed at child consultation center in Japan, is 66,701 in 2012.

The types of abuse are classified into four types: physical abuse, sexual abuse, emotional maltreatment, and neglect. According to a recent report from the Japan Ministry of Health, Labor, and Welfare, emotional maltreatment is the most frequent as 38.4%, physical abuse is the second as 32.9%, neglect is the third as 26.1%, and sexual abuse is the fourth as 2.1%. Recently, the rate of physical abuse is decreasing, and the rate of both emotional maltreatment and neglect is increasing in Japan, as well as in North America. AHT is the most common in physical abuse, and is the most important factor which influences on survival and neuro-functional

prognosis.^{4,5,23,24)} It has been reported that intracranial injury caused 49.2% of abused death in children.⁵⁾ In a recent report, child abuse represented 38% of traumas, it was the cause of 71% of death and 90% of severe disability.²⁵⁾

Most of the clinical characteristics of AHT in Japan have been similar to those in North America. However, several characteristics have been different as follows: mother is the most common perpetrators, impact is a more common mechanism of trauma than shaking, and external trauma is more common reflecting the existence of impact.⁵⁾ Although restricted in infants with acute SDH due to AHT, composition ratio of the perpetrators is suggested to be changing in Japan.⁷⁾

According to the annual report from the Japan Ministry of Health, Labor, and Welfare, the number of death from child abuse were approximately 50–60 cases/year, unchanged during the period of 2003–2012. In the same period, total number of child abuse increased 2.5 times fold from 26,569 cases in 2003 to 66,701 cases in 2012. Thus, it has been suggested that the recent increase of child abuse is brought by the spreading recognition, early detection, and early intervention for child abuse, leading to prevent the occurrence of serious cases.⁷⁾

Biomechanics

Studies of the biomechanics of brain injury have established that forces applied to the head that result in a rotation of the brain cause diffuse brain injuries. It is this type of movement that is responsible for the DAI and SDH. In contrast, forces that result in a translation movement are generally less injurious to the brain, with the effects largely determined by the specific focal contact forces. The type and severity of the injury are determined both by the type of deceleration and its magnitude. In infants and young children, household falls causing head injuries mainly involve low-velocity translational forces. Rotational deceleration is distinctly uncommon.¹⁹⁾

The most common cause of AHT may be shaking in North America. Rapid, impulsive, emotional, and violent shaking often occur, when an infant is crying, suckling, or training for excretion.^{26,27)} Violent shaking affects the intracranial contents, to produce rapid and repeated acceleration-deceleration movement as well as angular movement. Another common cause may be impact, often complicated with shaking. The magnitude of angular deceleration is 50 times as great when the head of an infant model held by the trunk forcefully strikes a surface as when shaking alone.^{28,29)} When the surface is soft, the force

of the impact is widely dissipated and may not be associated with visible signs of surface trauma, even though the brain itself decelerates rapidly.⁴⁾ This angular force is distinct from the forces generated in most cases of accidental trauma in infants. It is supported by the fact that the majority of abused infants have clinical, radiologic, or autopsy evidence of blunt impact to the head.^{4,13,21)}

Such AHT occurs most commonly in infant because of anatomical and physical characteristics, such as large size of the head compared to the body trunk, large amount of water content of the brain, immature cervical spine and insufficient strength of cervical muscles, large subarachnoid space, and immature myelination.^{13,17,30)} Shaking and/or shaking-impact force under such circumstances expose brain to various movements against skull and dura mater, resulting in shearing of bridging veins.³¹⁾

In Japan, impacts may be the most common mechanism of AHT. Miki et al. summarized the traumatic mechanisms of AHT in Japan as follows: direct blow is the most frequent as 37%, thrown is the second as 31%, dropped is the third as 14%, and shaking is the next as 10%.¹⁵⁾ In contrast, SBS has been more common in the United States than Japan, as shaking related 68% or caused 38% of abusive injuries.^{4,10,11,17,32)} Concerning the mechanism of AHT, it is interesting that the most common intracranial lesion of AHT is acute SDH in both the United States and Japan, despite the most common mechanism of injury is quite different between Japan and the United States. However, no reasonable explanation has been established concerning this issue.

Pathophysiology

I. Acute SDH

The most common intracranial lesion has been proposed as the acute SDH in AHT. From the view point of SDH, it was reported that abuse was confirmed in 59%, unintentional injury in 23%, and indeterminate cause in 18% in cases with acute SDH, who are 36 months old or younger.³³⁾ The mean age of abused children with acute SDH was 8.7 ± 8.1 months old, as with unintentional injuries was 19.1 ± 10.0 months. Chronic or mixed acute and chronic SDHs were found only in 44% of abused children and 67% of cases with indeterminate injury, but not in cases with unintentional injury. Long bone and/or rib fractures were found in 51% of abused children, but in only one unintentionally injured child. They concluded that one-fifth of infant SDH resulted from unintentional trauma. Of those without obvious unintentional trauma, 76% were corroborated to have been abused. Acute SDHs commonly

occur along parietal or occipital falx and tentorium. In general, the amount of acute SDHs are small and rarely causes death. Acute SDHs often cause brain swelling and intracranial hypertension.¹⁴⁾

Compared to unintentionally injured children, abused children were younger, more likely to have chronic SDHs and multiple associated injuries.³³⁾ It has been reported that 44% of infants with chronic SDH were thought to have sustained previous abusive injuries.³⁴⁾ Concerning chronic SDH, it has been reported that 46% of neonates have SDH that formed in the perinatal period during labor and its potential relationship to the misdiagnosis of abused head trauma even during infancy as chronic SDH.³⁵⁾

Differential diagnosis includes coagulopathies, meningitis, metabolic diseases such as glutaric aciduria, osteogenesis imperfecta, and accidental infantile SDH. Accidental infantile SDH has been proposed to be caused by a short-distance fall (fall from a height less than 5 feet). Accidental infantile SDH are characterized as: the patients are predominantly male, the mechanism of trauma is mild such as fall on the ground, most patients develop generalized tonic seizure, all patients have retinal hemorrhages, patients at the age of 7–10 months old are the most common, most of the patients result in good outcome and not recurred.^{36,37)} Since SDHs are rarely caused by accidental low-energy short-distance falls,^{36–39)} the existence of accidental infantile SDH has been mostly denied in the United States.

II. Intracranial lesions other than acute SDH

Traumatic subarachnoid hemorrhages, cerebral contusions, diffuse brain injuries, diffuse brain swellings are also involved as intracranial lesions of AHT other than acute SDH. Acute epidural hematomas are rarely involved. When shaking is the main cause of AHT injury, shearing injuries^{24,40,41)} or cervical cord injuries¹⁷⁾ are also included. In chronic stage, intracranial AHT lesions include chronic SDHs and diffuse hemispheric atrophy.

III. Skull fracture

Skull fracture is usually produced by an impact, and the fractures in AHT may not be suffered from a mild and simple trauma such as a fall. The severity of the skull fractures are various in AHT. In AHT, severe force are often inflicted, the fractures often have multiple fracture lines, widely dissected, and bilateral. In cases with depressed fractures, the fractures are often opened and broad area is involved. Parietal and occipital convexity are common site of fractures as well as skull base.^{31,42–44)} Coexistence

with an old fracture is thought to be another characteristic of AHT skull fracture.

Clinical Features

Common symptoms include lethargy, irritability, meningismus, seizure, increased or decreased tone, consciousness disturbance, vomiting, poor feeding, breathing abnormalities, and apnea. Approximately half of the patients with AHT have severe impairment, are unresponsive, have opisthotonos, or are moribund.⁴⁵⁾ The fontanelle may be full. Seizures are in 40–70% of patients.^{18,46)} The patients are often complicated with hypotension and hypoxemia, leading to diffuse cerebral ischemia and subsequent diffuse hemispheric atrophy of the brain. An anemia can occur in thick acute SDH. Even cases with thin acute SDH may result in poor outcome, when epileptic seizures are abandoned to delay receiving appropriate treatments. Transport to an appropriate medical facility is often delayed.

General physical findings include bruising, swelling, a pattern of cutaneous marks, and burns. In some patients, no extracranial injuries are detected. Some cutaneous injuries become visible only after admission. In some patients, soft-tissue injuries such as scalp hemorrhages, are noted only at autopsy.²¹⁾ In cases with AHT, intracranial hematomas are the most common as 53.4%, blows and scalp hematomas are the second as 28.2%, skull fractures are the third as 15.3%, lacerations are the fourth as 3.1%, in order.

I. Victims, perpetrators, and social circumstances

It has been reported that risk factors for non-accidental injuries in children include young parents, unstable family situations, low socioeconomic status, and disability or prematurity of the child.^{47,48)} Early papers suggested that females were the most common perpetrators of fetal child abuse in the United States.^{49,50)} Bergman et al. found that, in severe child cases resulting in permanent injury or death, fathers and boyfriends were the most common perpetrators.⁵¹⁾ Starling et al. also revealed fathers and boyfriends to be the most common abusers.⁴⁸⁾ In their report, fathers are the most common perpetrators as 37%, mothers' boyfriends are the second as 20.5%, female baby sitters are the third as 17.5%, and the mothers are the fourth as 12.6%. Lazowitz et al. reported that the fathers were the most common perpetrators as 33%, mothers' boyfriends as 20%, and the mothers as 6.5%.⁶⁾ The rate of baby sitter is varied ranging from 4% to 30%.^{48,52,53)}

In Japan, it has been proposed that the mothers are the most common perpetrators as 53.7%, and the fathers are the second as 28.4%. Although

restricted in abused infants with ASDH, a recent report from Japan demonstrates changes in the composition rate of the perpetrators as: fathers are the most common perpetrators as 39%, mothers are the second as 27%, and mothers' boyfriends are the third as 17%.⁷⁾ In addition to changes in the perpetrators, abused infants without external trauma is also demonstrated.

II. Retinal hemorrhage

In AHT, the retinal hemorrhages are frequently complicated as 50–100%.^{2,4,18)} They are numerous, can be unilateral or bilateral, and occupy multiple layers of the retina extending to the periphery.¹⁴⁾ Laterality of the retinal hemorrhage does not always meet the laterality of the SDHs. The rate of retinal hemorrhage is much higher in AHT (11.8–73.2%) than in adult severe head injury (1.18–3.33%).⁵⁴⁾ The presence of dense vitreous hemorrhage in infants with AHT has a poor visual prognosis, due to the frequent concomitant occurrence of significant retinal pathology. In contrast, infants with only intraretinal or subhyaloid hemorrhage tend to have a much better prognosis, with less disruption of the intraocular structure.⁵⁵⁾ Recent report indicated that SDHs were significantly correlated with retinal hemorrhage and child abuse.²⁵⁾ The sensitivity of retinal hemorrhage for the diagnosis of child abuse was 75%, and the specificity of it was 93.2%. Retinal hemorrhages associated with accidental trauma were mild, and the specificity of more severe retinal hemorrhage was 100%, although the grading of retinal hemorrhage requires the expertise of a trained neuroophthalmologist.

The mechanisms of retinal hemorrhage have not been well established yet, but several mechanisms are supposed to be related, including Purtscher's retinopathy due to intrathoracic pressure increase, intracranial hypertension, increased retinal venous pressure, extravasation of subarachnoid blood, and traction of retinal vessels at the vitreoretinal interface due to angular deceleration.^{14,30,56,57)} Since AHT is strongly associated with retinal hemorrhages, it is strongly suspected in infants with retinal hemorrhage.⁵⁶⁾ However, retinal hemorrhages are not always specific for the diagnosis of abusive trauma.²⁹⁾ Differential diagnosis includes either severe or mild accidental trauma suffering SDHs,^{19,36,39,58,59)} non-traumatic subarachnoid hemorrhage, sepsis, coagulopathy, galactosemia, severe hypertension, and so on.^{60,61)} In addition, retinal hemorrhages are seen in up to 25% of vaginally delivered newborns.⁶²⁾ The diagnosis of AHT cannot rest on the findings of retinal hemorrhage alone, but the findings of severe bilateral retinal hemorrhages with retinal

folds or detachments is particularly suggestive of the diagnosis. The importance of fundoscopic examination has been suggested to detect not only fresh retinal hemorrhages but also old traumatic fundus changes. It has been stressed the importance of ophthalmologists participation from the first medical contact, since physicians other than ophthalmologists misdiagnosed 29% of retinal hemorrhages.⁴⁴⁾

Diagnosis

I. Radiological features

A CT may be essential to detect intracranial lesions in abusive head trauma. Subdural or subarachnoid hemorrhage can nearly always be detected on CT.²⁹⁾ Hemorrhages most often appear as unilateral or bilateral high density collections of fresh blood that are thin but extensive; a particular propensity for the interhemispheric fissure, especially posteriorly, is well documented.^{63,64)} In addition, extensive loss of gray-white matter differentiation and diffuse hemispheric hypodensity have been known as a unique CT finding associated with SDH in infancy. This finding can be unilateral or bilateral. The basal ganglia and posterior fossa structures are relatively spared and thus appear hyperdense as compared with the surrounding cerebrum, the so-called "reversal sign" or "black brain."⁴⁵⁾ In unilateral cases, an additional wedge-shaped area of hypodensity in the contralateral frontal lobe is noted, reflecting subfalcine herniation.²⁹⁾ Diffuse hypodensity is not always apparent on the initial CT, but appears in 38% of cases.⁶⁵⁾ It has been also demonstrated that such hypodensities appear on initial CT scans performed as early as 1.2 hours after the report of injury, and become evident within 27 hours in all cases.⁶⁵⁾ This finding is not specific for abuse, but seen most often in association with abuse.²⁹⁾

Magnetic resonance imaging (MRI) is useful to detect white matter injury such as DAI, traumatic acute and subacute subarachnoid hemorrhage, and upper cervical spinal cord injury due to SBS. DAI is generally proven by detecting cerebral microhemorrhages (MHs), which are often associated with DAI. Localized axonal injury or DAI manifested petechial tissue-tear MH have been readily identified on T_2^* gradient recalled echo (GRE) and susceptibility-weighted imaging (SWI).^{66,67)} It has been shown that SWI depicts 4–6 times as many MH compared to standard T_2^* GRE imaging.⁶⁷⁾ Ultra-high field (7T) SWI is more sensitive than 3T SWI for the depiction of MH in DAI.⁶⁸⁾ It has been demonstrated that the presence of intraparenchymal MH in children with nonaccidental trauma, as detected on SWI, correlates with poor long-term neurologic

outcomes.⁶⁹⁾ In addition, MRI is also useful to detect heterochronic intracranial hemorrhage such as chronic SDH. Diffusion-weighted MRI is now being used to study the direct effects of traumatic injury, as well as detecting secondary ischemic and hypoxic changes.^{40,67,70,71)} Diffusion-tensor imaging, which allows evaluation of white matter fiber tracts by taking anisotropy of water diffusion, has been shown to be useful in identifying white matter abnormalities after DAI when conventional imaging appears normal.⁶⁷⁾ Magnetic resonance spectroscopy acquires metabolites information reflecting neuronal integrity and function, providing early prognostic information regarding outcome.⁶⁷⁾

Plain skull films are useful to detect skull fractures, which are found most commonly in the occipital or parieto-occipital regions. Multiple or complex skull fractures have been associated with abuse.^{63,72,73)} A skeletal survey is essential in the evaluation of abused children, since extracranial abnormalities are detected in 30–70% of abused children with head injury.^{6,63)} A variety of skeletal injuries have been thought to be characteristic such as multiple posterior or lateral rib fractures and metaphyseal fractures, by detecting periosteal reaction and callus formation. Repeated skeletal survey or radionuclide bone scans are rarely useful to detect sites of subtle injury.^{74,75)}

II. Diagnostic tools

With inflicted head injuries, an accurate history is rarely provided.²⁹⁾ Classical diagnosis depends on a high index of suspicion and the physical findings of a bulging fontanelle, head circumference greater than the 90th percentile, and retinal hemorrhage, but these signs and symptoms are nonspecific.¹⁸⁾ Radiological and ophthalmological examination may provide useful information, however, to suspect and to achieve the diagnosis of child abuse may not be always easy. In recent years, various scoring systems, tools, and/or algorithms have been reported for the diagnosis of the abused child to differentiate from other types of traumatic brain injury in children. Hoskote et al. has reported the child abuse risk factor scoring system. As the abuse risk factors, five factors are presented in cases with acute SDH in children: (1) age less than 12 weeks, (2) inconsistent history, (3) retinal hemorrhage, (4) positive skeletal survey, (5) unexplained bruising.⁷⁶⁾ When three or more factors are met, probability of child abuse is as 100%, two as 93%, one as 82%. Aoki has reported judgment criteria of abused head trauma.³⁶⁾ Dashti et al. has reported algorithm for the diagnosis of AHT.⁷⁷⁾ These diagnostic criteria or the algorithms are not established yet as the clinical diagnostic criteria

for AHT, however, they may be useful tools to protect the children who are suspected as victims of AHT. In addition, for an appropriate diagnosis of child abuse, history taking, careful physical examination, neuroradiological survey with CT and MRI, skeletal survey, ophthalmological examination, and social workers' cooperation as well as systematic survey may be necessary. It may be useful to establish a child protective service, which consists of social workers, pediatricians, radiologists, ophthalmologists, and neurosurgeons.²⁹⁾

III. The role of physicians, including neurosurgeons

As mandated reporters of suspected child abuse, neurosurgeons carry the burden of recognizing and responding to medical manifestations in AHT. To protect abused children from further repeated AHT, neurosurgeons must remain cognizant of the possibility of AHT in children who present with both subtle and overt neurologic symptoms, when a witness's report is hardly obtained from a third person. It is important to hospitalize an abused child under cooperation with a pediatrician to secure safety preventing future additional AHT, even on occasions that the diagnosis of AHT is less certain. In addition, neurosurgeons must take seriously the ethical and legal mandates to report suspected child abuse to governmental agencies for investigation.¹⁶⁾ Such a report to the governmental agency should be done systematically by a child protective serviceteam, but not personally. On occasion that a parent is a perpetrator, such systematic management may prevent belligerent behavior of the perpetrator against a medical staff.

Management

In an infantile AHT, surgical management may be indicated in cases with acute epidural hematoma and SDH, and chronic SDH. The surgical indication and techniques for them may not be different from those in cases with accidental injury. As the surgical managements for infantile acute SDHs, fontanelle puncture, trepanation, and hematoma removal via craniotomy have been reported. In cases with large acute SDH, decompression by the fontanelle puncture and subsequent craniotomy has been utilized. During the surgical managements, caution should be paid to avoid systemic hypotension. Pre-operative systemic anemia should be corrected by blood transfusion. As the post-operative managements, respiration, circulation, intracranial pressure (ICP), and cerebral perfusion pressure (CPP) should be managed to avoid secondary ischemia, in addition to anti-convulsant measures.

In severe cases, respiratory management with ventilator, blood gas, and systemic blood pressure monitoring under securing the arterial line, central venous pressure monitoring, ICP and CPP monitoring may be also utilized. Barbiturate coma therapy or hyperosmolar agents may be utilized for ICP and CPP management as necessary. For the management of epileptic seizures, oral, suppository, and transvenous administration of diazepam may be useful, as well as continuous intravenous administration of barbiturate. However, the value of aggressive management of ICP has not been established on the basis of outcome studies, which show infants who present with poor prognostic indicators, especially bilateral diffuse hypodensity on CT, have dismal outcome regardless of treatment.²⁹⁾

Conservative management may be also utilized in cases with thin acute SDH, traumatic subarachnoid hemorrhage, cerebral contusion, or diffuse brain injury. In such situations, status epileptics and hypoxia should be avoided. Even in cases with initially mild epileptic seizure, the brain results in secondary ischemia due to intracranial hypertension of respiratory suppression in case of status epileptics. In infant, prophylactic administration of anticonvulsant may be useful under tissue oxygen saturation monitoring.

As the surgical management for infantile chronic SDH, repeated dural puncture other than fontanelle, hematoma removal and irrigation via trepanation, and subduro-peritoneal shunt have been reported to be useful.

On occasion that a parent (or another caregiver) is a potential perpetrator, it is important to put both the child and the parent (or another caregiver) under surveillance without isolating. The role of physician, including neurosurgeon, is not to apportion blame or investigate potential criminal activity, but to identify the medical problems, treat the child's injuries, and offer accurate medical information to parents, families, child protective services, and law enforcements.¹⁶⁾ Interaction with the parent in such an impassive manner, as well as systematic management, may rarely make a parent aggressive, to focus on the treatment of abused child.

The goal of the management for an abused child is to return the child to home safely and to restore a peaceful daily living. In Japan, since real mothers are the most common perpetrators, it seems not easily achieved to return the victims to home. On such occasions, the role of a neurosurgeon is to provide medical information (e.g., the timing of discharge, medical follow-up schedule, and so on) to child protective services or law enforcement,

interpreting nonmedical professionals in an understandable manner.¹⁶⁾

Prognosis

It has been reported that patients with AHT present with motor, visual, and auditory impairments, and mental retardation as they grow.¹¹⁾ In 80% of cases with SBS, follow-up CT reveals severe atrophy of brain parenchyma, as well as various neurofunctional deficits.^{23,78,79)} It is well known that the outcome and the prognosis of acute SDH are poor in abused infants. Miki reported the mortality as 32%, and the severely disabled including chronic vegetative state as 34%.⁵⁾ In the United States, the mortality has been proposed to be ranged as 12–30%, and many of the survivors are severely disabled.

Haviland et al. reported the mortality as two out of 15 cases (13.3%). Of the survivors, nine (69.2%) showed major neurological handicaps and three (23.1%) showed moderate. Only one survivor was considered normal at discharge.⁸⁰⁾ Ludwig et al. reported the mortality of SBS as 15%, and the morbidity as 50%.¹⁸⁾

Motor weakness is well known as a sequela caused by cerebral atrophy subsequent to primary traumatic and secondary ischemic injuries, as well as intellectual and developmental impairment. In addition, visual impairment is specifically complicated to limit social adaptation. It has been known that cognitive and/or behavioral disorders, neurological deficits, and visual impairments are often revealed by long-term follow-up even in cases with good outcome at the time of discharge.

Risk factors

As a long-term outcome in infant with shaken/impact syndrome, Duhaime et al. reported the acute risk factors for poor outcome as unresponsiveness on admission, need for endotracheal intubation, age less than 6 months, epileptic seizures, apnea, uni- or bi-lateral diffuse hypodensity on CT.⁸¹⁾ Patients who demonstrate such findings result in encephalomalacia in chronic stage, and are often complicated by chronic SDH. Foster et al. reported that hemispheric hypodensity on CT was associated with Pediatric Intensity Level of Therapy (PILOT) scores and daily maximal intracranial pressure, but not with age, initial Glasgow Coma Scale score, or mortality.⁸²⁾ The presence of skull fracture was more likely in the children who did not develop hemispheric hypodensity on CT.⁸²⁾ Johnson et al. stated that pre-hospital dyspnea/apnea occurred in 57% of patients with AHT, and that cerebral ischemia induced by traumatic apnea could be a major cause

of poor outcome.⁴⁶⁾ Diffuse brain injury, as well as upper cervical cord injury, has also been a major cause of mortality and neurological handicaps.^{17,24,41)} Relationship between the acute ophthalmological lesions and the outcome/prognosis has been also reported. The severity of the retinal hemorrhage^{32,83)} or the dense vitreous hemorrhage⁵²⁾ has been reported as a risk factor for poor visual and neurofunctional outcome.

Conclusion

AHT, infants with acute SDH in particular, is a serious head injury and has been well characterized. Since it is a social pathological phenomenon influenced by the victims, perpetrators including parents and other caregivers, socioeconomic circumstances, and various aspects of it have potentials to change with time. Participants for the management and treatment of AHT should persevere to update the trends, knowledge, and characteristics of them.

Conflicts of Interest Disclosure

None of the authors have any conflicts of interest (COI) associated with this study. All authors who are members of The Japan Neurosurgical Society (JNS) have registered online self-reported COI Disclosure Statement Forms through the website for JNS members.

References

- 1) Durkin MS, Olsen S, Barlow B, Virella A, Connolly ES Jr: The epidemiology of urban pediatric neurological trauma: evaluation of, and implications for, injury prevention programs. *Neurosurgery* 42: 300–310, 1998
- 2) Billmire ME, Myers PA: Serious head injury in infants: accident or abuse? *Pediatrics* 75: 340–342, 1985
- 3) Graupman P, Winston KR: Nonaccidental head trauma as a cause of childhood death. *J Neurosurg* 104: 245–250, 2006
- 4) Duhaime AC, Gennarelli TA, Thibault LE, Bruce DA, Margulies SS, Wiser R: The shaken baby syndrome. A clinical, pathological, and biomechanical study. *J Neurosurg* 66: 409–415, 1987
- 5) Miki T: Child abuse with head injury in Japan. *Neurotraumatology* 25: 71–77, 2002 (Japanese)
- 6) Lazoritz S, Baldwin S, Kini N: The Whiplash Shaken Infant Syndrome: has Caffey's syndrome changed or have we changed his syndrome? *Child Abuse Negl* 21: 1009–1014, 1997
- 7) Karibe H, Hayashi T, Kameyama M, Tominaga T: Infantile abusive head trauma with acute subdural hematoma: changes in clinical characteristics in the last 20 years. *Nervous System in Children* 39: 313–319, 2014 (Japanese)
- 8) Caffey J: Multiple fracture in long bones of infants suffering from chronic subdural hematoma. *Am J Roentgenol Radium Ther* 56: 163–173, 1946
- 9) Kempe CH, Silverman FN, Steele BF, Drogemueller W, Silver HK: The battered-child syndrome. *JAMA* 181: 17–24, 1962
- 10) Guthkelch AN: Infantile subdural hematoma and its relationship to whiplash injury. *Br Med J* 22: 430–431, 1971
- 11) Caffey J: The whiplash shaken infant syndrome: manual shaking by the extremities with whiplash-induced intracranial and intraocular bleedings, linked with residual permanent brain damage and mental retardation. *Pediatrics* 54: 396–403, 1974
- 12) Childhood injuries in the United States. Division of Injury Control, Center for Environmental Health and Injury Control, Centers for Disease Control. *Am J Dis Child* 144: 627–646, 1990
- 13) Hahn YS, Raimondi AJ, McLone DG, Yamanouchi Y: Traumatic mechanisms of head injury in child abuse. *Childs Brain* 10: 229–241, 1983
- 14) Reece RM: Controversies in shaken baby/shaken impact syndrome, in Palusci VJ, Lazoritz S (eds): *The Shaken Baby Syndrome. A Multidisciplinary Approach*. Binghamton, The Haworth Press, 2001, pp 367–388
- 15) Minns RA, Brown JK: Neurological perspectives of non-accidental head injury and whiplash/shaken baby syndrome; an overview, in Minns RA, Brown JK (eds): *Shaking and Other Non-Accidental Head Injuries in Children*. London, Cambridge University Press, 2005, pp 1–105
- 16) Christian CW, Block R; Committee on Child Abuse and Neglect; American Academy of Pediatrics: Abusive head trauma in infants and children. *Pediatrics* 123: 1409–1411, 2009
- 17) Hadley MN, Sonntag VK, ReKate HL, Murphy A: The infant whiplash-shake injury syndrome: a clinical and pathological study. *Neurosurgery* 24: 536–540, 1989
- 18) Ludwig S, Warman M: Shaken baby syndrome: a review of 20 cases. *Ann Emerg Med* 13: 104–107, 1984
- 19) Duhaime AC, Alario AJ, Lewander WJ, Schut L, Sutton LN, Seidl TS, Nudelman S, Budenz D, Hertle R, Tsiaras W: Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. *Pediatrics* 90: 179–185, 1992
- 20) Goldstein B, Kelly MM, Bruton D, Cox C: Inflicted versus accidental head injury in critically injured children. *Crit Care Med* 21: 1328–1332, 1993
- 21) Alexander R, Sato Y, Smith W, Bennett T: Incidence of impact trauma with cranial injuries ascribed to shaking. *Am J Dis Child* 144: 724–726, 1990
- 22) Waga S, Fujimoto K, Okada M, Miyazaki M, Tanaka Y: Caudate hemorrhage. *Neurosurgery* 18: 445–450, 1986
- 23) Miki T, Ito H, Sengoku Y, Ikeda Y, Endou T, Mukai T: Mechanism of brain injury in child abuse: Case

- report and review of the literature. *Neurotraumatology* 20: 126–132, 1997 (Japanese)
- 24) Shimura T, Nakazawa S, Takahashi H, Kobayashi S, Node Y, Suzuki H, Mukai T, Ohno Y: Neuro-pathological study of battered child syndrome: eight autopsy cases. *No Shinkei Geka* 22: 23–28, 1994 (Japanese)
 - 25) Vinchon M, Defoort-Dhellemmes S, Desurmont M, Dhellemmes P: Accidental and nonaccidental head injuries in infants: a prospective study. *J Neurosurg* 102(4 Suppl): 380–384, 2005
 - 26) Caffey J: On the theory and practice of shaking infants. Its potential residual effects of permanent brain damage and mental retardation. *Am J Dis Child* 124: 161–169, 1972
 - 27) Dykes LJ: The whiplash shaken infant syndrome: what has been learned? *Child Abuse Negl* 10: 211–221, 1986
 - 28) Bruce DA, Alavi A, Bilaniuk L, Dolinskas C, Obrist W, Uzzell B: Diffuse cerebral swelling following head injuries in children: the syndrome of “malignant brain edema.” *J Neurosurg* 54: 170–178, 1981
 - 29) Duhaime AC, Christian CW, Rorke LB, Zimmerman RA: Nonaccidental head injury in infants—the “shaken-baby syndrome.” *N Engl J Med* 338: 1822–1829, 1998
 - 30) Levin AV: Ocular manifestation of child abuse. *Ophthalmology Clinics of North America* 3: 249–264, 1990
 - 31) Lancon JA, Haines DE, Parent AD: Anatomy of the shaken baby syndrome. *Anat Rec* 253: 13–18, 1998
 - 32) Kivlin JD, Simons KB, Lazoritiz S, Ruttum MS: Shaken baby syndrome. *Ophthalmology* 107: 1246–1254, 2000
 - 33) Feldman KW, Bethel R, Shugerman RP, Grossman DC, Grady MS, Ellenbogen RG: The cause of infant and toddler subdural hemorrhage: a prospective study. *Pediatrics* 108: 636–646, 2001
 - 34) Parent AD: Pediatric chronic subdural hematoma: a retrospective comparative analysis. *Pediatr Neurosurg* 18: 266–271, 1992
 - 35) Gabaëff SC: Investigating the possibility and probability of perinatal subdural hematoma progressing to chronic subdural hematoma, with and without complications, in neonates, and its potential relationship to the misdiagnosis of abusive head trauma. *Legal Med (Tokyo)* 15: 177–192, 2013
 - 36) Aoki N: [Infantile acute subdural hematoma: caused by abuse or minor head trauma?]. *Nervous System in Children* 36: 326–330, 2011 (Japanese)
 - 37) Aoki N, Masuzawa H: Infantile acute subdural hematoma. Clinical analysis of 26 cases. *J Neurosurg* 61: 273–280, 1984
 - 38) Gardner HB: A witnessed short fall mimicking presumed shaken baby syndrome (inflicted childhood neurotrauma). *Pediatr Neurosurg* 43: 433–435, 2007
 - 39) Plunkett J: Fatal pediatric head injuries caused by short-distance falls. *Am J Forensic Med Pathol* 22: 1–12, 2001
 - 40) Chen CY, Zimmerman RA, Rorke LB: Neuroimaging in child abuse: a mechanism-based approach. *Neuroradiology* 41: 711–722, 1999
 - 41) Vowles GH, Scholtz CL, Cameron JM: Diffuse axonal injury in early infancy. *J Clin Pathol* 40: 185–189, 1987
 - 42) Hobbs CJ: ABC of child abuse. Head injuries. *Br Med J* 298: 1169–1170, 1989
 - 43) Nimkin K, Kleinman PK: Imaging of child abuse. *Pediatr Clin North Am* 44: 615–635, 1997
 - 44) Stoodley N: Neuroimaging in non-accidental head injury: if, when, why and how. *Clin Radiol* 60: 22–30, 2005
 - 45) Han BK, Towbin RB, De Courten-Myers G, McLaurin RL, Ball WS Jr: Reversal sign on CT: effect of anoxic/ischemic cerebral injury in children. *AJNR Am J Neuroradiol* 10: 1191–1198, 1989
 - 46) Johnson DL, Boal D, Baule R: Role of apnea in nonaccidental head injury. *Pediatr Neurosurg* 23: 305–310, 1995
 - 47) Sills JA, Thomas LJ, Rosenbloom L: Non-accidental injury: a two-year study in central Liverpool. *Dev Med Child Neurol* 19: 26–33, 1977
 - 48) Starling SP, Holden JR, Jenny C: Abusive head trauma: the relationship of perpetrators to their victims. *Pediatrics* 95: 259–262, 1995
 - 49) Kaplun D, Reich R: The murdered child and his killers. *Am J Psychiatry* 133: 809–813, 1976
 - 50) Simons B, Downs EF, Hurster MM, Archer M: An epidemiological study of medically reported cases of child abuse. *NY State J Med* 66: 2783–2788, 1966
 - 51) Bergman AB, Larsen RM, Mueller BA: Changing spectrum of serious child abuse. *Pediatrics* 77: 113–116, 1986
 - 52) Kunz J, Bahr S: A profile of parental homicide against children. *Journal of Family Violence* 11: 347–362, 1996
 - 53) Margolin L: Child abuse by mothers’ boyfriends: why the overrepresentation? *Child Abuse Negl* 16: 541–551, 1992
 - 54) Betz P, Puschel K, Miltner E, Lignitz E, Eisenmenger W: Morphometrical analysis of retinal hemorrhages in the shaken baby syndrome. *Forensic Sci Int* 78: 71–80, 1996
 - 55) Matthews GP, Das A: Dense vitreous hemorrhages predict poor visual and neurological prognosis in infants with shaken baby syndrome. *J Pediatr Ophthalmol Strabismus* 33: 260–265, 1996
 - 56) Greenwald MJ, Weiss A, Oesterle CS, Friendly DS: Traumatic retinoschisis in battered babies. *Ophthalmology* 93: 618–625, 1986
 - 57) Levin AV: The ocular findings in child abuse. Focal points. *American Academy of Ophthalmology* 14: 1–13, 1998
 - 58) Gilliland MG, Luckenbach MW: Are retinal hemorrhages found after resuscitation attempts? A study of the eyes of 169 children. *Am J Forensic Med Pathol* 14: 187–192, 1993
 - 59) Goetting MG, Sowa B: Retinal hemorrhage after cardiopulmonary resuscitation in children: an etiologic reevaluation. *Pediatrics* 85: 585–588, 1990
 - 60) Levy HL, Brown AE, Williams SE, de Juan E Jr: Vitreous hemorrhage as an ophthalmic complication of galactosemia. *J Pediatr* 129: 922–925, 1996

- 61) Wetzel RC, Slater AJ, Dover GJ: Fatal intramuscular bleeding misdiagnosed as suspected nonaccidental injury. *Pediatrics* 95: 771–773, 1995
- 62) Watts P, Maguire S, Kwok T, Talabani B, Mann M, Wiener J, Lawson Z, Kemp A: Newborn retinal hemorrhages: a systematic review. *J AAPOS* 17: 70–78, 2013
- 63) Merten DF, Osborne DR, Radkowski MA, Leonidas JC: Craniocerebral trauma in the child abuse syndrome: radiological observations. *Pediatr Radiol* 14: 272–277, 1984
- 64) Zimmerman RA, Bilaniuk LT, Bruce D, Schut L, Uzzell B, Goldberg HI: Computed tomography of craniocerebral injury in the abused child. *Radiology* 130: 687–690, 1979
- 65) Bradford R, Choudhary AK, Dias MS: Serial neuroimaging in infants with abusive head trauma: timing abusive injuries. *J Neurosurg Pediatr* 12: 110–119, 2013
- 66) Tong KA, Ashwal S, Obenaus A, Nickerson JP, Kido D, Haacke EM: Susceptibility-weighted MR imaging: a review of clinical applications in children. *AJNR Am J Neuroradiol* 29: 9–17, 2008
- 67) Ashwal S, Wycliffe ND, Holshouser BA: Advanced neuroimaging in children with nonaccidental trauma. *Dev Neurosci* 32: 343–360, 2010
- 68) Moenninghoff C, Kraff O, Maderwald S, Umutlu L, Theysohn JM, Ringelstein A, Wrede KH, Deuschl C, Altmeppen J, Ladd ME, Forsting M, Quick HH, Schlamann M: Diffuse axonal injury at ultra-high field MRI. *PLoS One* 10: e0122329, 2015
- 69) Colbert CA, Holshouser BA, Aaen GS, Sheridan C, Oyoyo U, Kido D, Ashwal S: Value of cerebral microhemorrhages detected with susceptibility-weighted MR imaging for prediction of long-term outcome in children with nonaccidental trauma. *Radiology* 256: 898–905, 2015
- 70) Suh DY, Davis PC, Hopkins KL, Fajman NN, Mapstone TB: Nonaccidental pediatric head injury: diffusion-weighted imaging findings. *Neurosurgery* 49: 309–318; discussion 318–320, 2001
- 71) Zimmerman RA: New detection in neuroradiology of child abuse, in Reece RM, Ludwig S (eds): *Child Abuse, Medical Diagnosis and Management*. Philadelphia, Lippincott Williams & Wilkins, 2001, pp 81–96
- 72) Cohen RA, Kaufman RA, Myers PA, Towbin RB: Cranial computed tomography in the abused child with head injury. *AJR Am J Roentgenol* 146: 97–102, 1986
- 73) Meservy CJ, Towbin R, McLaurin RL, Myers PA, Ball W: Radiographic characteristics of skull fractures resulting from child abuse. *AJR Am J Roentgenol* 149: 173–175, 1987
- 74) Kleinman PK: Diagnostic imaging in infant abuse. *AJR Am J Roentgenol* 155: 703–712, 1990
- 75) Smith FW, Gilday DL, Ash JM, Green MD: Unsuspected costo-vertebral fractures demonstrated by bone scanning in the child abuse syndrome. *Pediatr Radiol* 10: 103–106, 1980
- 76) Hoskote A, Richards P, Anslow P, McShane T: Subdural haematoma and non-accidental head injury in children. *Childs Nerv Syst* 18: 311–317, 2002
- 77) Dashti SR, Decker DD, Razzaq A, Cohen AR: Current patterns of inflicted head injury in children. *Pediatr Neurosurg* 31: 302–306, 1999
- 78) Frank Y, Zimmerman R, Leeds NM: Neurological manifestations in abused children who have been shaken. *Dev Med Child Neurol* 27: 312–316, 1985
- 79) Sinal SH, Ball MR: Head trauma due to child abuse: serial computerized tomography in diagnosis and management. *South Med J* 80: 1505–1512, 1987
- 80) Haviland J, Russell RI: Outcome after severe non-accidental head injury. *Arch Dis Child* 77: 504–507, 1997
- 81) Duhaime AC, Christian C, Moss E, Seidl T: Long-term outcome in infants with the shaking-impact syndrome. *Pediatr Neurosurg* 24: 292–298, 1996
- 82) Foster KA, Recker MJ, Lee PS, Bell MJ, Tyler-Kabara EC: Factors associated with hemispheric hypodensity after subdural hematoma following abusive head trauma in children. *J Neurotrauma* 31: 1625–1631, 2014
- 83) Wilkinson WS, Han DP, Rappley MD, Owings CL: Retinal hemorrhage predicts neurologic injury in the shaken baby syndrome. *Arch Ophthalmol* 107: 1472–1474, 1989

Address reprint requests to: Hiroshi Karibe, MD, Department of Neurosurgery, Sendai City Hospital, 1-1-1 Asuto-Nagamachi, Taihau-ku, Sendai, Miyagi 982-8502, Japan.
e-mail: karibe@nsg.med.tohoku.ac.jp