REVIEW

Educational paper

Abusive Head Trauma Part I. Clinical aspects

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Abstract Abusive Head Trauma (AHT) refers to the combination of findings formerly described as shaken baby syndrome. Although these findings can be caused by shaking, it has become clear that in many cases there may have been impact trauma as well. Therefore a less specific term has been adopted by the American Academy of Pediatrics. AHT is a relatively common cause of childhood neurotrauma with an estimated incidence of 14–40 cases per 100,000 children under the age of 1 year. About 15–23% of these children die within hours or days after the incident. Studies among AHT survivors demonstrate that approximately one-third of the children are severely disabled, one-third of them are moderately disabled and one-third have no or only mild symptoms. Other publications suggest that neurological problems can occur after a symptom-free interval and that half of these children have IQs below the 10th percentile. Clinical findings are depending on the definitions used, but AHT should be considered in all children with neurological signs and symptoms especially if no or only mild trauma is described. Subdural haematomas are the most reported finding. The only feature that has been identified discriminating AHT from accidental injury is apnoea. *Conclusion*: AHT should be approached with a structured approach, as in any other (potentially lethal) disease. The clinician can only establish this diagnosis if he/she has knowledge of the signs and symptoms of AHT, risk factors, the differential diagnosis and which additional investigations to perform, the more so since parents seldom will describe the true state of affairs spontaneously.

Keywords Abusive head trauma · Child abuse · Head injury, closed · Haematoma, subdural

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Introduction

With an estimated incidence of 14–40 per 100,000 children under the age of 1 Abusive Head Trauma (AHT) is probably as prevalent in young children as neonatal meningitis (25–32 per 100,000 live births [21,36,44]) and lymphatic leukaemia (28.7–36.6 per 100,000 children <1 [23]). Still, clinicians feel uncomfortable establishing the diagnosis and performing the right additional investigations. The term 'Abusive Head Trauma' is recommended by the American Academy of Pediatrics (AAP) for the combination of findings formerly described as shaken baby syndrome [9]. It has, among other things, also been described as Inflicted Traumatic Brain Injury (ITBI), Non-Accidental Head Injury (NAHI) and whiplash shaken infant syndrome. AHT can present with cerebral, cervical or cranial injuries that result from inflicted head injury, which can be shaking, impact trauma or a combination of both.

The term 'shaken baby syndrome' implies the underlying mechanism has to be shaking. Although shaking can cause severe head injury, in some of these children there also has been impact trauma or a combination of shaking and impact. Therefore, the AAP recommends using Abusive Head Trauma, a term that does not refer to the exact accident mechanism (shaking) but to the harming action in broader perspective (abuse).

The aim of this educational paper is to give the paediatrician, facing a possible case of AHT, a comprehensive overview of the clinical findings and differential diagnoses. The role of radiology in establishing the diagnosis of AHT will be discussed later in this journal, in 'Educational paper: Abusive head trauma, Part II: Radiological aspects'.

Incidence and consequences

Incidence

For several reasons it is difficult to determine the actual incidence of AHT. First of all, not all abused infants need/seek medical help and come in contact with the medical system. In a Dutch study among primary health care visitors, 5.6% of the parents of 6-month-old babies reported to have shaken, slapped or smothered their baby at least once [34]. In an American study, 2.6% of the children under the age of 2 years were reported by their mothers to have been shaken [38]. Most of these children do not need medical attention and are therefore not represented in incidence numbers. Secondly, not all cases presented in hospitals are recognised as cases of AHT. Abuse cases may be diagnosed with other conditions, e.g., accidental trauma or Sudden Infant Death Syndrome. Finally, different definitions for AHT are in use, making comparison of incidence rates reported by different institutions difficult.

Several prospective studies have been performed to try to establish the incidence of AHT. Most of these studies collect data from both hospitals and (forensic) pathology departments. In the first population-based American study, an incidence of 29.7 per 100,000 person years was found for children under the age of 1 year [25]. In a Scottish study, the incidence of children with AHT, based on hospital visits, was 24.6 per 100,000 children younger than 1 year [1]. In British and Estonian studies, the incidence rates were 14.2 and 40.0 per 100,000 in children under the age of 1, respectively [20,37]. In a prospective Swiss study an incidence of 14 per 100,000 was found in children under the age of 6 years [17].

Most incidence studies describe a higher proportion of boys being affected; range 62–77% [1,17,20,25,37]. Most

cases manifest before the age of 6 months; median age ranges from 2.2 to 5.9 months [1,17,20,25,37].

AHT is as prevalent in very young children as other potential lethal diseases e.g. neonatal meningitis.

Mortality

According to Duhaime et al. [14], 'trauma is the most common cause of death in childhood, and inflicted head injury is the most common cause of traumatic death in infancy' [14]. Reported mortality rates for AHT in the studies mentioned above are quite similar: 15–23% of all recognised cases of AHT die before or shortly after presentation [17,20,25,37]. In the Scottish cohort, the mortality was lower than in other studies, namely 11% [2]. This is probably due to selection bias; studies including only patients admitted to a Paediatric Intensive Care Unit (PICU) will probably find worse outcomes than studies including patients with AHT from all hospital departments. The latter will probably be a better representative of mortality in the full spectrum of AHT. This means that although AHT accounts for a substantial number of paediatric deaths, it is not exactly known what percentage of the affected children die. Besides the short-term mortality, a significant amount of AHT survivors are severely handicapped and will have a lower life expectancy. Death in these children is unlikely to be registered as the result of AHT.

Morbidity

In the Swiss AHT follow-up study by Fanconi and Lips [17], only 36% of survivors had a good outcome, and 64% were disabled. Roughly half of these children were moderately disabled, e.g., had significant reduction in cognitive functioning, motor deficiencies, or were referred to outpatient rehabilitation therapy. The other half were severely disabled, e.g., had cognitive scores in the deficient range, severe motor deficits, or were referred to inpatient rehabilitation [17]. In the Scottish incidence study, the same distribution was found; roughly one-third had a good outcome, one-third were mildly to moderately disabled and one-third were severely disabled [2]. In a small sample of AHT survivors (only 14 of 62 AHT survivors could be contacted), Duhaime et al. [13] also found that 36% had a good outcome, 14% were moderately disabled and 50% were severely disabled or vegetative. Because of the young age of the patients and the prolonged developmental course of the brain, it has been said that the final neurological prognosis cannot be given before they reach school age. Bonnier et al. found that children who initially appear to be symptom-free can have neurological problems after this

symptom-free interval [5]. Ewing-Cobbs et al. [16] found that nearly 50% of children with early traumatic brain injury (both AHT and other causes) had IQ's below the 10th percentile.

Recognising AHT

Because of (relatively) common appearance and severe consequences, all paediatricians should be able to recognise AHT. Establishing the diagnosis AHT can be very difficult, especially if no fractures are found. In a retrospective study among children with AHT by Jenny et al. [24], 31% of abuse cases were not initially recognised. AHT was more likely to be unrecognized in very young children, white children, children from intact families and in children without respiratory problems or convulsions. The authors estimate that 80% of deaths in the missed AHT group could have been prevented by earlier recognition of abuse. In the majority of the cases of AHT, parents describe a relatively small trauma, e.g., fall from arms, or no preceding trauma at all [19]. Having knowledge about the expected range of injury seen following common accidents can be useful in differentiating between accidental and non accidental injury. Child abuse needs intervention; missing the diagnosis can have severe consequences for the patient and the siblings. On the other hand, wrongfully concluding that a child has been mistreated will have devastating effects on the family as well. It is therefore essential that, in each case of suspected AHT, a multidisciplinary approach is chosen. Paediatricians, radiologists, ophthalmologists, social workers and child protection workers have to work closely together, preferably in an institutional child abuse and neglect team (CAT), in order to collect as much information as possible on the facts that led to clinical manifestations. The clinical abnormalities found during physical examination have to be sorted out in detail in order to formulate an accurate differential diagnosis. It is of great importance that the right kind of additional investigation is performed to reach the right diagnosis.

Clinical findings and differential diagnosis

As AHT is the most common cause of neurotrauma in children younger than 2 years, it should be considered in *all* children presenting with neurotrauma, unless the trauma is without any doubt accidental, e.g., a car accident.

Clinical signs and symptoms seen in children with AHT depend of the type of AHT and accompanying injuries. Neurological manifestations include altered state of consciousness (77%), seizures (43–50%), vomiting (15%), and delayed development (12%) [20,37]. Subdural haemorrhages, described in 77–89% of the patients, are the most

common neuroradiological finding [17,37] (Fig. 1). In autopsy series, subdural haematoma (SDHs) have been described in approximately 83–90% of all children diagnosed with AHT [6,12]. Other neuroradiological findings include subarachnoidal haemorrhages (12–25%), intracerebral haemorrhages (8%), epidural haemorrhage (4%), parenchyma lesions (37%), and hygroma (11%) [17,37]. In a systematic review by Maguire et al. [29], only apnoea was found to be a critical distinguishing feature for AHT compared to accidental head injury, having a positive predictive value (PPV) of 93%. Rib fractures and retinal haemorrhage were strongly associated with AHT, having a PPV of 73% and 71%, respectively. Though seizures are more common in AHT, this trend did not achieve statistical significance. Long bone fractures were not significantly discriminative, although the 'classical metaphyseal lesion' was not separated out in this analysis, and has been found to be highly correlated with abuse. Skull fracture and bruising on the head were more common in accidental injury, but this trend, too, did not achieve significance. Correlation between skull fractures and intracranial pathology is limited. Skull fractures can occur with and without intracranial bleeding. On the other hand, intracranial injury can be present without a skull fracture [4].

In a recent systematic review on ocular signs in AHT intraocular haemorrhages were seen in 74% (range 51%–100%) of 560 combined cases of AHT and in 82% (range 63–100%) of cases in autopsy series [3]. Retinal haemorrhages are bilateral in 85% of AHT cases [27]. Injuries

Fig. 1 Two-month-old boy, victim of abusive head trauma. The CT shows an interhemispheric subdural haematoma along the falx cerebri. Subdural haematomas in children often extend along the falx cerebri and this should not be confused with calcification, which is seen at an older age

outside the head, e.g., bruises and fractures, are found in 35–54% and 33–48%, respectively [17,37]. This means that in a substantial number of AHT patients no other traumatic injuries are detected, although severe traumatic brain injury exists. The absence of fractures and bruises is therefore never an argument against AHT.

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Minns and Busutill distinguish four different types of AHT [31]

Hyperacute encephalopathy

Approximately 6% of all children with AHT presents with this form of AHT in their study. These children are young at presentation (2–3 months of age) and present with acute respiratory failure and cerebral oedema; the majority of them are dead or die shortly after presentation. Hyperacute encephalopathy results from an injured brainstem after hyperflexion and hyperextension. At autopsy localised axonal damage at the craniocervical junction, in the corticospinal tracts, and in the cervical cord roots is seen, in combination with brain swelling and hypoxic injury.

Acute encephalopathy

This is the most common form of AHT, affecting approximately 53% of cases, and has been described as the classic 'shaken baby syndrome'. Children present with the findings mentioned above: a low level of consciousness, increased cranial pressure, convulsions, apnoea, hypotonia, anaemia, and/or shock. Trauma in body parts other than the brain are found in 35–54% of the cases [37]. The cause can be shaking injury, impact trauma or a combination of both. Extended injury is seen on MRI, with SDHs, oedema, contusions and lacerations and white matter shearing.

Subacute non-encephalopathic presentation

Approximately 19% of all patients presents with sub-acute non-encephalopathic AHT. This is a less severe form of acute encephalopathy. SDH's and retinal haemorrhages are found, but other brain injuries are less recognisable. Coexistent injuries are common.

Chronic extra-cerebral presentation

The chronic extra-cerebral form of AHT (22% of cases) is the hardest to relate to abuse because of the time interval between the incident, development of complaints and establishing a diagnosis. Children present with expanding head circumference and/or signs of raised intracranial pressure, e.g., irritability, vomiting, failure-to-thrive, hypotonicity or convulsions. An isolated subdural haemorrhage is often found, but retinal haemorrhages can have disappeared already. If other signs of abuse can be detected, e.g., rib fractures, it is easier to relate the SDH to abuse. If the SDH is the only sign, it is highly plausible that a trauma has happened, but limitation in the dating of SDHs makes conclusive diagnosis difficult.

AHT should be part of the differential diagnosis in children with a variety of non-neurologic presentations, such as increasing head circumference, children with failure-to-thrive, vomiting, crying excessively, poor drinking, developmental delay, children who present with other forms of physical abuse, and children with siblings who suffered severe physical abuse.

Multiple factors contribute to the recognition and diagnosis of AHT. A major determination that must be made is whether the described trauma can explain the child's injuries. The identification of other, often non-acute injuries can be of great importance to support the diagnosis of inflicted injury, although the absence of other traumatic injuries does not rule out AHT. The child's past history can contribute to suspicion of inflicted injury. In one study approximately 20% of the children who died as a result of Child Abuse had had contact with the health care system in the month before their dead. Some of these presentations were suspicious for CAN and provided a missed opportunity for intervention [26]. Oral et al. [32] found that 8% of children who died of AHT had a medical history of abuse that was missed by health care providers.

It is therefore of great importance that history taking be done very carefully and that information be gathered about not only this child and this event, but also about the lifelong, and family medical history [27]. Both parents and other health care professionals can give important information that can support or weaken the diagnosis of AHT. History taking should at least include the following:

Detailed history

Any reported trauma event should be made absolutely clear to the treating physician. If an accident is described, the doctor should exactly be aware of the timeline and exact circumstances of the incident, i.e., of which height the child fell, what material was on the floor, who was present, how

the child reacted, what did the caregivers do at that moment, how many time elapsed between the incident and seeking medical help. If no accident was described but the child was found or suddenly appeared to be in a bad condition it should be clear what the first manifestations were: what was the last time the child was seen in good condition, how many time elapsed between this moment and the clinical symptoms.

Medical history

The history should document prior presentations to any hospitals, to the GP, and to routine primary health care visits. Prior traumas or signs and symptoms attributable to injury must be sought out.

Growth curve

All former measurements should be retrieved to construct a growth curve. The growth curve of the skull is most important, as the first sign of deviation from the patient's growth curve can indicate a possible event that led to an increase in head circumference.

Medical history of siblings

Unusual medical contacts of brothers and/or sisters may indicate a hereditary disorder or a history of family violence.

Family known at Child Protection Services (CPS)

Many children who present with AHT have a prior history with CPS. The team must determine if this child and/or siblings are known at the CPS, or if there are siblings placed in foster care.

Assessing risk factors

In close collaboration with, e.g., a social worker an assessment of risk factors should be made (Table 1). AHT also occurs in families with little or no risk factors at all, but

the presence of risk factors should increase the physicians' awareness.

> AHT should be part of the differential diagnosis in children with a variety of nonneurologic presentations, such as increasing head circumference, children with failure-to-thrive, vomiting, crying excessively, poor drinking, developmental delay, children who present with other forms of physical abuse, and children with siblings who suffered severe physical abuse.

Differential diagnosis

Although AHT is the most common cause of SDH in children <1 year [30], the differential diagnosis of intracranial haemorrhage is extensive (Table 2, adapted from David) [10]. After formulating a differential diagnosis, additional investigations have to be performed to confirm or rule out alternative diagnoses. The role of imaging will be discussed in part II, to be published later in this journal. We will not address blood examination, ophthalmologic examination and genetic analysis for rare diseases.

All medical conditions, both congenital and acquired, should be ruled out before trauma can be considered to cause the (combination of) findings. If trauma is the only option, it should be judged carefully whether accidental trauma is a possible explanation for the abnormalities found. If no accidental trauma has been described that can cause these findings this option is excluded as well and AHT is the only remaining diagnosis. Most diagnoses listed in this table can be ruled out easily because they should have been known from medical history (medical interventions, ingestion), are accompanied by other signs and symptoms (genetic disorders) or can be rejected after simple laboratory results (coagulation disorders, infectious disorders). Furthermore, all diseases listed above are extremely uncommon. Some groups of diagnoses are more likely to cause SDH's, namely birth trauma, coagulation disorders, metabolic disorders and accidental trauma.

Birth trauma has been described as a common cause of SDH's. In prospective studies, 10% to 46% of newborns

Table 1 Risk factors for child abuse [41]

Parents	Environment	Child
Psychiatric problems	Partner violence	Ex-prematures
Substance or alcohol abuse	Large family	Dysmatures
Suffered from abuse in youth	Stepchildren	Physically disabled
Lack of pedagogic capacity	Poverty/financial problems	Mentally disabled
Very low level of education	Residential instability	Excessive crying
Single parent	Social isolation	Chronically ill
Young mother	Refugee families	Unwanted
Unemployment		Behavioural problems

Table 2 Differential diagnosis of intracranial haemorrhage in children adapted from David [10]

Category	Cause
Non accidental trauma	Shaking, impact or a combination
Accidental trauma	For example, falls, motor vehicle accident
Medical and surgical interventions	Known from the medical record
Prenatal/perinatal conditions	Birth trauma
	Intrauterine trauma, e.g., domestic violence to mother
	Idiopathic intrauterine subdural haematoma
	Intrauterine isoimmune thrombocytopaenic purpura
	Maternal pre-eclampsia
Coagulation disorders	Haemophilia A and B
	von Willebrand disease
	Factor V deficiency
	Factor XII deficiency
	Factor XIII deficiency
	Haemorrhagic disease of the newborn (vitamin K deficiency)
	Disseminated intravascular coagulation (DIC)
	Hermansky–Pudlak syndrome (albinism)
	Alpha 1-antitrypsin deficiency
Congenital malformations	
Genetic disorders	Osteogenesis imperfecta
	Sickle cell anaemia
	Alagille syndrome
	Ehlers–Danlos syndrome
	Menkes kinky hair syndrome
Metabolic disorders	Glutaric aciduria type 1
	Pyruvate carboxylase deficiency
Infectious disorders	Meningitis
	Kawasaki disease
	Herpes simplex encephalitis
	Congenital toxoplasmosis
Intoxication	Lead poisoning
	Cocaine
	Anticoagulant therapy

without clinical signs or symptoms had SDH. All of these SDH's were resolved after 4 weeks to 3 months [28,35,43]. These haematomas were differently located compared to SDH's with clinical manifestations. Typically, they present as a thin film of blood occipital or infratentorial overlying the cerebellar hemispheres. It has been hypothesized that these clinically silent SDH's may present with delayed symptoms due to catastrophic re-bleeding, or growing chronic SDH [10]. No studies except for case reports have demonstrated the existence of these categories. It is well known that some SDH's present after birth with major clinical symptoms. These space-occupying SDH's give immediate signs and symptoms post-partum, e.g., seizures, hypotonia and coma [10].

Coagulation disorders are an important alternative cause of intracranial haemorrhage in young children. It is

uncommon for bleeding disorders to present with intracranial haemorrhage except for vitamin K deficiency [7,45]. It is mostly seen in breastfed babies who do not receive supplemental vitamin K. In some cases an underlying disease is present [15,33,40,42]. Although most Western countries have vitamin K guidelines [11], there is never a 100% adherence. It is important to note that an abnormal coagulation time can be caused by a large haemorrhage and does not necessarily reflect an underlying problem with coagulopathy [22].

Although certain genetic and metabolic disorders can cause SDHs, most of the affected children will show other features as well. These diseases are less common than AHT and the presence of a rare disease does not rule out child abuse. In children with SDH's without signs of trauma, glutaric aciduria type 1 should be ruled out as children with

glutaric aciduria type 1 can develop normally the first 6–18 months of life [18].

AHT is the most common cause of SDH's in young children.

Falls from limited height and AHT

An extensive overview of literature on short-distance falls and accompanying injuries has been provided in 'Forensic Aspects of Pediatric Fractures' [4]. Short distance falls (<1.5 m.) usually take place from a couch, crib or caregivers arms. Eight studies describe 3451 children who experienced a short distance fall. Child abuse was highly unlikely in most studies, e.g., because only falls during hospital admissions were included. In 25 of these children (0.7%), a skull fracture was found. Skull fractures were never seen after a fall from a couch. It has to be noted that not all children underwent (skull) radiography, so fractures without clinical signs or symptoms could have been missed. No life-threatening events or intracranial pathology have been reported in these types of falls. Serious intracranial injuries and fatalities have been reported after a fall from a baby walker and perambulator. Literature is not conclusive on potential serious or lethal consequences after falls from baby bouncers, bunk beds, high chairs, staircases, shopping trolleys and trampolines. Very few studies have been published on these specific subjects. It is clear that falls are very common in young children; about 50% of all children will experience a short distance fall in the first year of life. It has been calculated that approximately 1 in 250,000 children <1 year will die from a short-distance fall. Another literature study found that the population based risk of dying after a short distance fall for young children is less than 1 per million per young children per year [8].

As (intra)cranial pathology is a necessary condition for AHT, this is described in all studies on AHT. As mentioned before injuries other than intracranial pathology, e.g., fractures, are found in approximately 40% of all cases. The only diagnoses, in the differential, that cause both

intracranial haemorrhage and fractures are severe accidents like motor vehicle accidents, and osteogenesis imperfecta [39]. As a result the combination of these findings is highly suggestive for AHT (Fig. 2a, b). More difficult are the cases where intracranial pathology is the only abnormal finding. AHT is the most common cause of SDHs in young children, but in absence of any other signs of trauma establishing the diagnosis is more difficult for clinicians. Of all neurologic symptoms, only apnoea has been found to be a critical distinguishing feature for AHT compared to accidental head trauma. After ruling out underlying disease with relatively simple additional investigations, history taking, including all the items mentioned above, is of great diagnostic value. A presenting history of no or low-impact trauma has a specificity of 0.97 and a PPV of 0.92 for AHT. In patients with neurological symptoms at discharge, no or low-impact trauma histories' specificity and PPV are both 1.0 for AHT. In the same study injuries were blamed on resuscitation in 12% of AHT cases compared to 0% in not definite abuse cases. The initial history of trauma described by caregivers was changed in 9% of definite AHT cases compared to 0% in not definite abuse cases [19].

Conclusion

AHT is as prevalent in very young children as other potential lethal diseases, e.g., neonatal meningitis. It is the most common cause of neurotrauma in children younger than 1 year, and should therefore be considered in all children presenting with neurotrauma or unexplained neurological symptoms. The diagnosis should also be considered in children presenting with non-traumatic (aspecific) symptoms and signs such as increasing head circumference, children with failure-to-thrive, vomiting, crying excessively, poor drinking, developmental delay, as well as children who present with other forms of physical abuse and children with siblings who suffered severe physical abuse. About one-fifth of the children die and two-thirds of survivors are handicapped. The true extent of

Fig. 2 a Four-month-old boy admitted with a large subdural haematoma with clinical symptoms. No trauma was described. A bite mark was found on the left shoulder. Unfortunately no measuring tape has been used while taking the picture. b Chest radiograph obtained 2 weeks after the initial CT scan shows a series of posterior rib fractures on the left side with callus formation (arrow)
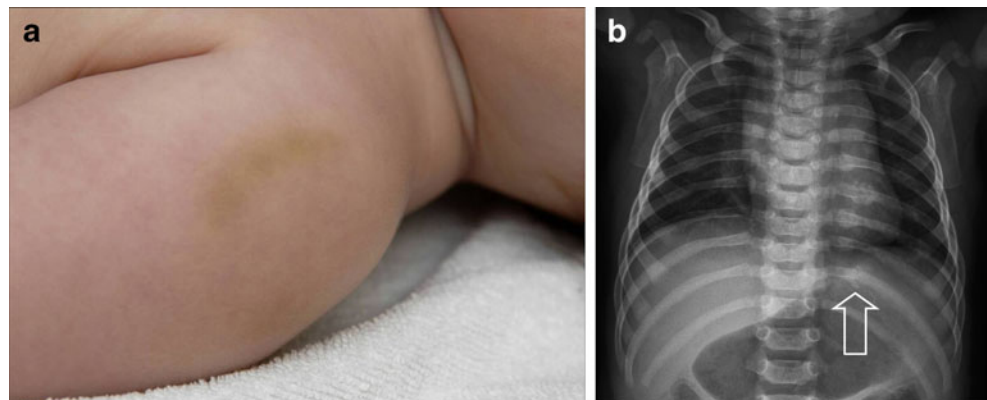
the damage cannot be assessed before school age, as neurological problems and low IQs can become apparent after a symptom-free interval. The clinician can only establish this diagnosis if he/she has knowledge on the signs and symptoms of AHT, risk factors, the differential diagnosis and which additional investigations to perform, as parents seldom will describe the true state of affairs spontaneously. AHT should be approached with as structured an approach as any other (potentially lethal) disease. History taking should be done extremely carefully and include contact with other health care providers, medical history of siblings, skull circumference growth curve and checking for CPS involvement. Physical examination should always be performed to look for additional signs of trauma. A differential diagnosis should be formulated and additional investigations should be performed to rule out or confirm alternative diagnoses. Birth trauma, coagulation disorders, metabolic disorders and accidental trauma are, besides AHT the most common causes of SDHs. All medical conditions should be ruled out before considering trauma to be the cause of the signs and symptoms. If trauma is the only option, it should be judged carefully whether accidental trauma is a possible explanation for the findings. If no accidental trauma has been described that can cause the signs and symptoms, this option is excluded as well and AHT is the only remaining diagnosis.

References

1. Barlow KM, Minns RA (2000) Annual incidence of shaken impact syndrome in young children. Lancet 356:1571–1572
2. Barlow KM, Thomson E, Johnson D, Minns RA (2005) Late neurologic and cognitive sequelae of inflicted traumatic brain injury in infancy. Pediatrics 116:e174–e185
3. Bhardwaj G, Chowdhury V, Jacobs MB, Moran KT, Martin FJ, Coroneo MT (2010) A systematic review of the diagnostic accuracy of ocular signs in pediatric abusive head trauma. Ophthalmology 117:983–992
4. Bilo RAC, Robben SGF, Rijn van RR (2010) Forensic aspects of pediatric fractures. Springer
5. Bonnier C, Nassogne MC, Evrard P (1995) Outcome and prognosis of whiplash shaken infant syndrome; late consequences after a symptom-free interval. Dev Med Child Neurol 37:943–956
6. Brennan LK, Rubin D, Christian CW, Duhaime AC, Mirchandani HG, Rorke-Adams LB (2009) Neck injuries in young pediatric homicide victims. J Neurosurg Pediatr 3:232–239
7. Brousseau TJ, Kissoon N, McIntosh B (2005) Vitamin K deficiency mimicking child abuse. J Emerg Med 29:283–288
8. Chadwick DL, Bertocci G, Castillo E, Frasier L, Guenther E, Hansen K et al (2008) Annual risk of death resulting from short falls among young children: less than 1 in 1 million. Pediatrics 121:1213–1224
9. Christian CW, Block R (2009) Abusive head trauma in infants and children. Pediatrics 123:1409–1411
10. David TJ (2008) Non-accidental head injury—the evidence. Pediatr Radiol 38(Suppl 3):S370–S377
11. de Winter JP, Joosten KF, Ijland MM, Verkade HJ, Offringa M, Dorrius MD et al (2011) New Dutch practice guideline for administration of vitamin K to full-term newborns. Ned Tijdschr Geneeskd 155:A936
12. Duhaime AC, Gennarelli TA, Thibault LE, Bruce DA, Margulies SS, Wiser R (1987) The shaken baby syndrome. A clinical, pathological, and biomechanical study. J Neurosurg 66:409–415
13. Duhaime AC, Christian C, Moss E, Seidl T (1996) Long-term outcome in infants with the shaking-impact syndrome. Pediatr Neurosurg 24:292–298
14. Duhaime AC, Christian CW, Rorke LB, Zimmerman RA (1998) Nonaccidental head injury in infants—the "shaken-baby syndrome". N Engl J Med 338:1822–1829
15. Ertekin V, Selimoglu MA, Gursan N, Ozturk CF (2005) Image and diagnosis. Intracranial haemorrhage due to vitamin K deficiency, as the first symptom of extrahepatic biliary atresia. West Indian Med J 54:392–397
16. Ewing-Cobbs L, Prasad MR, Kramer L, Cox CS Jr, Baumgartner J, Fletcher S et al (2006) Late intellectual and academic outcomes following traumatic brain injury sustained during early childhood. J Neurosurg 105:287–296
17. Fanconi M, Lips U (2010) Shaken baby syndrome in Switzerland: results of a prospective follow-up study, 20022007. Eur J Pediatr 169:1023–1028
18. Hedlund GL, Longo N, Pasquali M (2006) Glutaric acidemia type 1. Am J Med Genet C Semin Med Genet 142C:86–94
19. Hettler J, Greenes DS (2003) Can the initial history predict whether a child with a head injury has been abused? Pediatrics 111:602–607
20. Hobbs C, Childs AM, Wynne J, Livingston J, Seal A (2005) Subdural haematoma and effusion in infancy: an epidemiological study. Arch Dis Child 90:952–955
21. Hristeva L, Booy R, Bowler I, Wilkinson AR (1993) Prospective surveillance of neonatal meningitis. Arch Dis Child 69:14–18
22. Hymel KP, Abshire TC, Luckey DW, Jenny C (1997) Coagulopathy in pediatric abusive head trauma. Pediatrics 99:371–375
23. Incidentie van kanker bij kinderen naar geslacht en morfologisch type in de periode 1990–2009 (ESR per 1.000.000). 2011. Integrale kankercentra. Last accessed 26-5-2011.http://www.ikcnet.nl/uploaded/docs/Landelijk/cijfers/incidentie%202009/Leeftijdspecifieke%20incidentie%20invasieve%20tumoren%20bij%20kindernen%200_17jaar.xls
24. Jenny C, Hymel KP, Ritzen A, Reinert SE, Hay TC (1999) Analysis of missed cases of abusive head trauma. JAMA 281:621–626
25. Keenan HT, Runyan DK, Marshall SW, Nocera MA, Merten DF, Sinal SH (2003) A population-based study of inflicted traumatic brain injury in young children. JAMA 290:621–626
26. King WK, Kiesel EL, Simon HK (2006) Child abuse fatalities: are we missing opportunities for intervention? Pediatr Emerg Care 22:211–214
27. Kivlin JD, Simons KB, Lazoritz S, Ruttum MS (2000) Shaken baby syndrome. Ophthalmology 107:1246–1254
28. Looney CB, Smith JK, Merck LH, Wolfe HM, Chescheir NC, Hamer RM et al (2007) Intracranial hemorrhage in asymptomatic neonates: prevalence on MR images and relationship to obstetric and neonatal risk factors. Radiology 242:535–541
29. Maguire S, Pickerd N, Farewell D, Mann M, Tempest V, Kemp AM (2009) Which clinical features distinguish inflicted from non-inflicted brain injury? A systematic review. Arch Dis Child 94:860–867

30. Matschke J, Voss J, Obi N, Gorndt J, Sperhake JP, Puschel K et al (2009) Nonaccidental head injury is the most common cause of subdural bleeding in infants <1 year of age. Pediatrics 124:1587–1594

31. Minns RA, Busuttil A (2004) Patterns of presentation of the shaken baby syndrome: four types of inflicted brain injury predominate. BMJ 328:766

32. Oral R, Yagmur F, Nashelsky M, Turkmen M, Kirby P (2008) Fatal abusive head trauma cases: consequence of medical staff missing milder forms of physical abuse. Pediatr Emerg Care 24:816–821

33. Puckett RM, Offringa M (2000) Prophylactic vitamin K for vitamin K deficiency bleeding in neonates. Cochrane Database Syst Rev CD002776

34. Reijneveld SA, van der Wal MF, Brugman E, Sing RA, Verloove-Vanhorick SP (2004) Infant crying and abuse. Lancet 364:1340–1342

35. Rooks VJ, Eaton JP, Ruess L, Petermann GW, Keck-Wherley J, Pedersen RC (2008) Prevalence and evolution of intracranial hemorrhage in asymptomatic term infants. AJNR Am J Neuroradiol 29:1082–1089

36. Synnott MB, Morse DL, Hall SM (1994) Neonatal meningitis in England and Wales: a review of routine national data. Arch Dis Child 71:F75–F80

37. Talvik I, Metsvaht T, Leito K, Poder H, Kool P, Vali M et al (2006) Inflicted traumatic brain injury (ITBI) or shaken baby syndrome (SBS) in Estonia. Acta Paediatr 95:799–804

38. Theodore AD, Chang JJ, Runyan DK, Hunter WM, Bangdiwala SI, Agans R (2005) Epidemiologic features of the physical and sexual maltreatment of children in the Carolinas. Pediatrics 115:e331–e337

39. Tokoro K, Nakajima F, Yamataki A (1988) Infantile chronic subdural hematoma with local protrusion of the skull in a case of osteogenesis imperfecta. Neurosurgery 22:595–598

40. van Hasselt PM, de Koning TJ, Kvist N, de Vries E, Lundin CR, Berger R et al (2008) Prevention of vitamin K deficiency bleeding in breastfed infants: lessons from the Dutch and Danish biliary atresia registries. Pediatrics 121:e857–e863

41. van Rooijen K, Berg T (2010) Risicofactoren en beschermende factoren voor kindermishandeling. Nederlands Jeugdinstituut. Last accessed 18-6-2011. http://www.nji.nl/nji/dossierDownloads/Risicofactoren_kindermishandeling.pdf

42. Van Winckel M, De Bruyne R, Van De Velde S, Van Biervliet S (2009) Vitamin K, an update for the paediatrician. Eur J Pediatr 168:127–134

43. Whitby EH, Griffiths PD, Rutter S, Smith MF, Sprigg A, Ohadike P et al (2004) Frequency and natural history of subdural haemorrhages in babies and relation to obstetric factors. Lancet 363:846–851

44. Wiswell TE, Baumgart S, Gannon CM, Spitzer AR (1995) No lumbar puncture in the evaluation for early neonatal sepsis: will meningitis be missed? Pediatrics 95:803–806

45. Yilmaz C, Yuca SA, Yilmaz N, Bektas MS, Caksen H (2009) Intracranial hemorrhage due to vitamin K deficiency in infants: a clinical study. Int J Neurosci 119:2250–2256