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REGULAR ARTICLE

Re-evaluation of medical findings in alleged shaken baby syndrome and abusive head trauma in Norwegian courts fails to support abuse diagnoses

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Abstract

Aim: The criteria for diagnosing abusive head trauma (AHT) are not well defined and this condition might be diagnosed on failing premises. Our aim was to review criminal AHT cases in Norwegian courts by scrutinising the underlying medical documentation.

Methods: Cases were identified in the data registry for Norwegian courts from 2004 to 2015. Documentation was obtained from relevant health institutions. The medical co-authors first made independent evaluations of the documentation for each child, followed by a consensus evaluation.

Results: A total of 17 children (11 boys) were identified, all diagnosed as AHT by court appointed experts, 15 were infants (mean age 2.6 months). A high proportion (41.2%) was born to immigrant parents and 31.3% were premature. The medical findings could be explained by alternative diagnoses in 16 of the 17 children; 8 boys (7 infants – mean age 2.9 months) had clinical and radiological characteristics compatible with external hydrocephalus complicated by chronic subdural haematoma. Six children (five infants with mean age 2.1 months) had a female preponderance and findings compatible with hypoxic ischaemic insults.

Conclusion: The medical condition in most children had not necessarily been caused by shaking or direct impact, as was originally concluded by the court experts.

KEYWORDS
abusive head trauma, benign external hydrocephalus, child abuse, shaken baby syndrome, subdural haematoma

1 | INTRODUCTION

A triad of medical findings has been regarded as almost pathognomonic for shaken baby syndrome (SBS) or abusive head trauma (AHT). For decades, this triad of subdural haematoma (SDH), retinal haemorrhages (RH) and encephalopathy has been accepted as proof of violent shaking in court cases. However, the validity of this triad as judicial proof has been questioned, mainly because of

Abbreviations: AHT, abusive head trauma; BEH, benign external hydrocephalus; BESS, benign enlargement of the subarachnoid spaces; CT, computerised tomography; HC, head circumference; ICP, intracranial pressure; MRI, magnetic resonance imaging; RH, retinal haemorrhages; SBS, shaken baby syndrome; SDH, subdural haematoma.

circular reasoning.[1,2] Solid scientific evidence for a causal relationship between the triad and violent shaking has never been demonstrated in the literature This was shown by an extensive literature review as far back as 2003[1] and in a 2016 review by the Swedish Agency for Health Technology Assessment and Assessment of Social Services,[3] summarised in a paper published in 2018.[2] These stated that: 'There is limited scientific evidence that the triad and therefore its components can be associated with traumatic shaking (low-quality evidence). There is insufficient scientific evidence on which to assess the diagnostic accuracy of the triad in identifying traumatic shaking (very low-quality evidence'.[2] Neither of these reviews was able to identify articles based on observed shaking and only two studies in the literature were based on confessed shaking.[3,4] However, these confessions came long time after the diagnosis – during police custody or judicial investigations. Confessions obtained under such circumstances are hampered by uncertainties.[5]

Thus, one may question whether the diagnoses of SBS/AHT are based on scientific evidence that in judicial context is solid enough to prove guilt beyond any reasonable doubt. We still need to know if some of these presumptive AHT diagnoses may in fact have been caused by non-traumatic causes, for instance, benign external hydrocephalus (BEH), which has been shown to predispose for SDH in infancy.[6-20]

In 2021, Feld et al[21] reviewed the legal outcome of criminal SBS/AHT cases in Germany, but without analysing the underlying medical documentation. The aim of the present study was to review recent SBS/AHT cases in Norwegian criminal courts by scrutinising the underlying medical documentation, including the neuroimaging. Furthermore, we wanted to find out how this information had been interpreted by the medical experts in court, and finally, how the experts' advice had been received and treated by the court, as expressed in the verdict.

Key Notes

- As criteria for diagnosing abusive head trauma (AHT) are not well defined, this condition may have been overdiagnosed and a re-evaluation of the medical documentation in AHT cases is therefore warranted.
- A 12-year nationwide study revealed that most children in court cases had findings compatible with medical conditions not caused by violence.
- Medical teams investigating suspected AHT should contain clinicians experienced in practical handling of head injured infants.

2 | MATERIAL AND METHODS

This study was approved of by The National Committee for Medical and Health Research Ethics (NEM) in Norway in 2016. The study was carried out by a multi-professional team of three medical professionals: one paediatric neurosurgeon, one professor of neurosurgery (KW), a professor of neuroradiology (JW) and two professors of civic law (US, AS). The court cases were identified by going through the Lovdata data registry for the Norwegian courts for 2004–2015, searching for the law paragraphs that cover such criminal acts. In addition, we searched the registry for the following Norwegian search word compatible with 'Violent shaking' in addition to 'Shaken baby', a diagnosis (in English) that is used also in Norwegian courts.

This search revealed a total of 17 cases with intracranial conditions that had caused suspicion of SBS/AHT and subsequent prosecution. These patients are listed as #1–17 in the present article. An article emphasising the judicial aspects of these court cases has already been published in Norwegian.[22] In this judicial article, we used a different numbering of the patients (4.2–4.18), but they are presented and discussed in the same order in both articles, patient #1 corresponds to 4.2 and patient #17 corresponds to 4.18. All the medical investigators were specialised in neuro-medicine with only general medical competence regarding injuries outside the head. A few infants with only extracranial injuries were therefore not included.

All relevant medical documentation was requested from the hospitals and other health institutions that had been in contact with the children. Both acute CT and MRI of the brain had been performed in 13 children, only CT in 3, and only MRI in1.

The medical investigators first evaluated independently the medical documentation for each child. Their findings and tentative diagnoses were written down on a standardised recording sheet, including presenting symptoms and signs, head size and growth, presence or absence of subdural or subarachnoid haematoma and parenchymal lesions. Further, measurements were made of three different extra-cerebral distances, assumed to be characteristic for the BEH diagnosis. These distances were the inter-hemispheric distance (IHD), the craniocortical width (CCW) and the sinocortical width (SCW). The measurements, above which these distances are considered abnormal, vary considerably in the literature, as stated in Wiig et al[23] After this initial round, a consensus evaluation was performed. The two neurosurgeons had no formal training in neuroimaging, but a rather extensive clinical experience in reading radiological images. The final decision on how to interpret the neuroimaging was, however, left to the neuroradiologist.

During the process described above, the medical authors had no access to the legal papers, including the reports from the medical experts in court or the verdicts. That information was kept with the two law professors and not made available for the medical authors until they had made up their minds on the diagnoses of the children.

After independent medical diagnoses had been reached by the medical investigators, the legal authors compared these diagnoses with the diagnoses given by the court experts and concluded regarding the final conviction.

3 | RESULTS

The medical experts in court concluded that all included children had an inflicted head injury, either caused by violent shaking alone (11), by a combination of shaking and blunt trauma (4), by unspecified 'forceful violence' (1) or by a blunt head trauma alone (1). No alternative diagnoses were discussed, except for BEH, which was discussed in one case, but later abandoned (#13), see below.

The medical authors found that the clinical and radiological documentation could be explained by an alternative diagnosis to SBS/AHT in 16 of the 17 children. The most important finding was that there appeared to be two distinct groups of children in our material; the largest comprised eight children – all boys – with clinical and radiological characteristic compatible with external hydrocephalus (BEH/BESS), hereafter referred to as the 'BEH-like group' (patients #2, #7, #10, #12–#16) (Figures 1 and 2).

The other group had a female preponderance – four girls and two boys – and were characterised by clinical and radiological findings compatible with a failure of blood and/or oxygen supply to the brain hypoxic-ischaemic injury (HII), below referred to as the 'HII-like group' (#3, #4, #6, #9, #11, #17) (Figures 3-5).

For details, including the three remaining children, see Table 1.

3.1 | Epidemiological characteristics

There was a marked male preponderance (64.7% boys). This skewed gender distribution was caused solely by the fact that all eight children in the BEH-like group were boys. In the HII-like group, there was an opposite sex distribution, four girls and only two boys. This difference in sex distribution between the two groups is statistically significant ($p = 0.015$, Fisher's exact test). Five infants – all boys with a mean age of 3.3 months not corrected for prematurity – were born prematurely, this constitutes 31.3% of the 16 children with known gestation length. The Norwegian national pre-term delivery rate during the first part of our study period was 6.4%.[24] This difference between the national prematurity rate and the rate in the present material is statistically significant ($p = 0.005$, Fisher's exact test). Three of these pre-terms belonged to the BEH-like group. The most premature infant (31 weeks) belonged to the HII-like group.

With two exceptions, all children were 6 months or younger; the two exceptions were a 15-month-old girl with rickets (#8) and a boy aged 3.5 years (#16) with an undiagnosed BEH. The remaining 15 infants had an age range of 18 days–6 months (mean age 2.6, median age 2.5 months). Six infants (40% of the 15 infants) were 1.5 months old or younger. If age is corrected for prematurity, a total of seven infants (46.7%) were that young.

Infants in the HII-like group had a mean age of 2.1 months (median 1, range 1–6 months). Infant boys in the BEH-like group (not including the 3.5-year-old boy) had a mean age of 2.9 months (median 2.5, range 1–5 months). The age difference between these two groups was not statistically significant.

The ethnic background of the included children differed from that of the general population, as seven children (41.2%), including five infants, were born to immigrant parents, 10 children were born to non-immigrants. The proportion of immigrants in the general Norwegian population during the study period varied between 7.6% in 2004[25] and 15.6% in 2015.[26] The difference between immigrant and non-immigrant children is statistically significant ($p = 0.007$, Fisher's exact test).

3.2 | Initial symptoms and clinical findings

For an overview of symptoms and findings, see Table 1.

Seizure was the most frequent presenting symptom; 8 of the 17 children (47.1%) had initial seizures and three more infants had possible seizures, described as 'opistotonus' (#13) and 'sudden stiffening' (#12, #14) as initial symptoms. Four infants in the HII-like group and four in the BEH-like group had initial epileptic seizures.

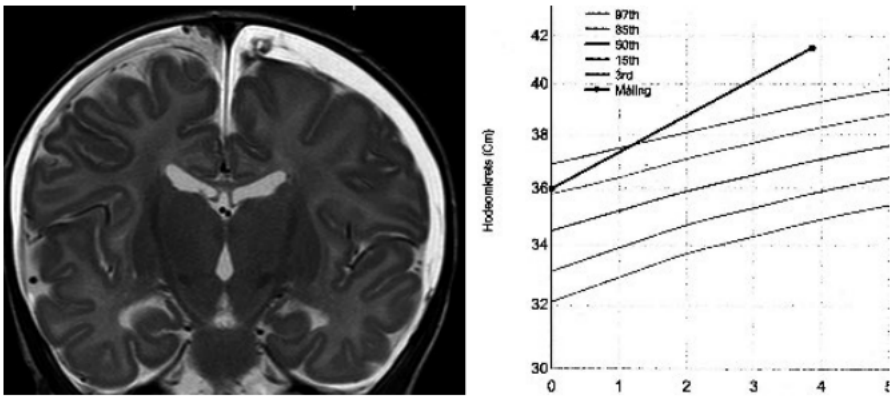

FIGURE 1 Patient #10. Left: Coronal T2-weighted image at 1 month of age shows bilateral cerebrospinal fluid signalling subdural fluid collections, compatible with chronic SDH or subdural hygroma. On the right side, there is a portion with signal suggesting more recent hematoma (arrow). There is no compression of underlying cerebral cortex. Right: Head circumference (HC) growth curve showing rapid increase from birth to 4 weeks of age (age in weeks)

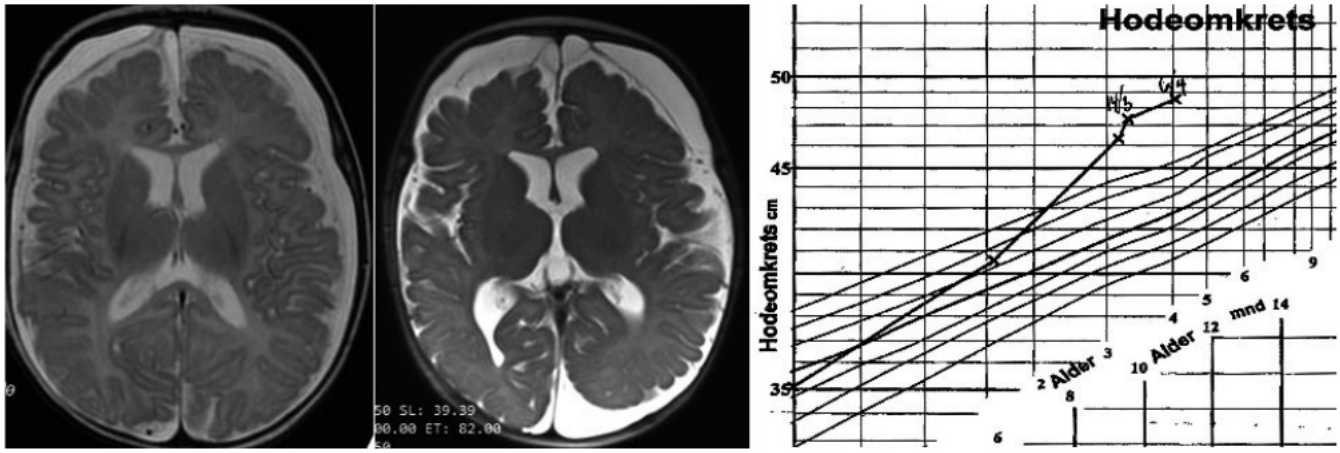

FIGURE 2 Patient #13. T2-weighted MRI at 2.5 months shows bilateral subdural hygroma or chronic SDH. There is no compression of underlying gyri and clearly visible subarachnoid space, suggesting an underlying BEH. MRI at 4 months (middle) shows slight increase in volume of subdural fluid, with compression of cerebral gyri. Right: HC growth curve showing a gradual increase over several weeks. 'Hodeomkrets': head circumference, 'Alder': age in months

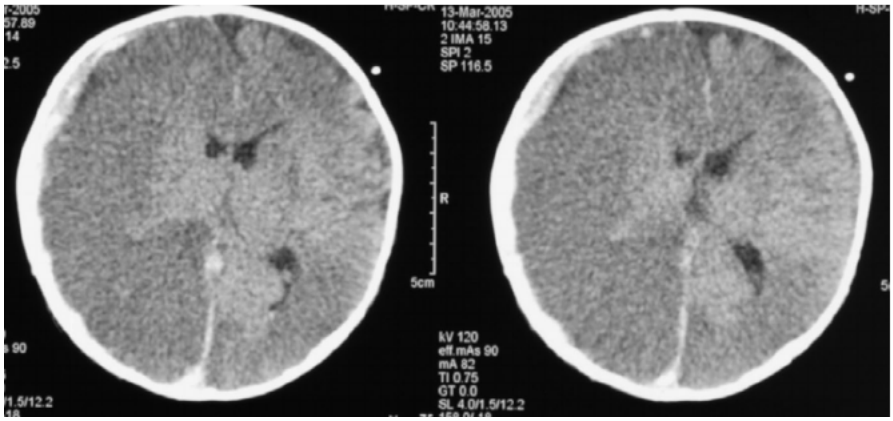

FIGURE 3 Patient #3. CT scan on admission shows widespread hypoxic/ischaemic changes (low attenuating areas) in the bilateral cerebral parenchyma, with sparing of central grey matter structures. There is also a thin acute SDH on the right side

Vomiting was noted as the initial symptom in two infants (#10, #15), in both cases being increasingly present over the last days/weeks before hospital admission.

Seven children, all boys in the BEH-like group, had large and/or rapidly increasing head circumference (HC). For one boy (#16), infantile HC development was not available, as he came to Norway after infancy. His large head was not noticed until he died, and autopsy was performed, as described below. Of the remaining seven infants, one (#12) was born with a large HC that remained at that level (97 percentile); the others had an HC within normal range (34–36 cm) at birth, which started to grow too rapidly in the following months. Six children (35.3%) had an HC at or above the 97% (percentile); five of these were infants (#2, #10, #12–#14) including three of the preterms, with a mean age of 2.3 months. A 5-month-old boy (#15) in the BEH-like group had an HC >90% and a 3-month-old girl (#9) had a rapidly increasing HC, crossing 2–3 percentile lines (from 10% to 50%–75%), thus complying with the national guidelines for when to suspect development of hydrocephalus.

Nine children had no signs of impact to the head. Three had blue marks in the *face*, but not on the neurocranium, one had a small blue mark on the forehead, one had scalp swelling (caused by a witnessed fall – patient #16), and three had swelling in association with an underlying cranial fracture (#1, #8, #17).

The intracranial pressure (ICP) was not measured in any of the children. Clinical signs of elevated ICP noted in the medical records include tense fontanelle and sunset gaze. Nine infants (#3, #4, #7, #9, #10, #13–#15, #17) were recorded as having a tense or bulging fontanelle, in one (#10) sunset gaze was also described. Three of the eight infants with a tense fontanelle also had an HC above 97% (#10, #13, #14), one had an HC above 90% (#15), and one (#9) had an HC that crossed 3 percentile lines. In the remaining four infants, the HC was within normal limits (#7, #17) or no HC measurement was noted in the medical records (#3, #4).

Fourteen of the children (82.4%) had RH; nine had bilateral RHs (#3, #5–#9, #11, #14, #17), five had unilateral or only small RHs (#2, #4, #10, #12, #15), and three had none.

3.3 | Radiological findings

In nine cases, encephalopathy was diagnosed based on neuroimaging HII signs (Figure 5). In severe cases (#3, #6, #8, #11, #14, #15,

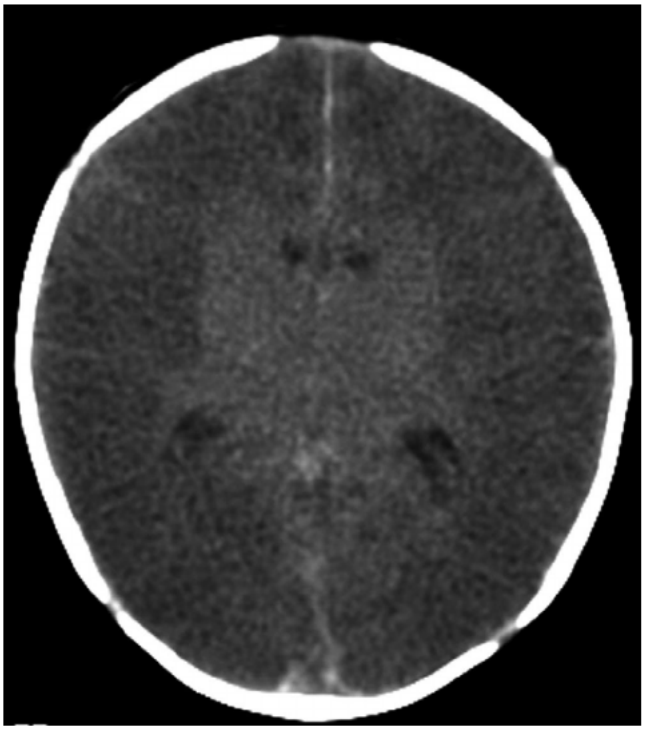

FIGURE 4 Patient #11. Axial CT shows global low attenuating in the cerebral cortex and subcortical white matter, with sparing of central grey matter. The findings are typical of hypoxic-ischaemic encephalopathy

#17), it later revealed itself as a marked delay in the head growth (#17) or even as a reduction of the HC (#15) (Figure 6).

Nine infants had neuroimaging compatible with chronic subdural haematoma (chronic SDH)/hygroma. In one of these (#2), the chronic SDH was asymptomatic and was diagnosed only because a long limb fracture caused suspicion of SBS/AHT. In the other eight infants, the chronic SDH caused symptoms. Two of these infants (#5, #14) also had small volumes of acute, coagulated blood in the chronic SDH – often referred to as 'acute on chronic' in radiology reports. [27]

An acute SDH was found in only one child, a 6-month-old prematurely born (week 31) boy (#3). The 3.5-year-old boy with an undiagnosed BEH (#16) had an isodense SDH.

With two exceptions (#5, #8), the subdural fluid collections did not compress the cortical surface, the ventricles, or cause a midline shift. With the same two exceptions, MR imaging showed a layer of normally looking cerebrospinal fluid between the cortex and the subdural fluid collection/haematoma.

Five infants (#1, #2, #6, #10, #13), aged 1–3 months (1–2 months corrected for prematurity in #1 and #13), had asymptomatic rib fractures. Three infants with rib fractures were born prematurely and nearly all rib fractures showed callus formation, indicating an ongoing healing process of some duration.

A clavicle fracture with callus formation was found in two children, a 1-month-old girl (#17 – vaginal delivery) and an old, completely healed fracture in the 3.5-year-old boy (#16).

Four children had a cranial fracture, one in a 15-month-old girl with rickets disease after an established head injury; she had hypocalcaemia and low values of phosphorus and magnesium. All three remaining cranial fractures were linear and asymptomatic. They were found in a boy (#1) aged 3 months (1.5 months corrected for prematurity) and two 1-month-old girls (#4 and #17), see Figure 7.

3.4 | Clinical outcome

Two patients died: a 1-month-old girl (#4) with an intracerebral haematoma and a 3.5-year-old boy (#16) with clinical and radiological characteristics of BEH, who sustained a large SDH, possibly after a seizure that resulted in a fall from own height with impact to the head.

Seven children survived with severe brain injury. One of these is the 15-month-old girl (#8) with rickets disease. She sustained a cranial fracture and a left-sided acute SDH with a cytotoxic oedema of the underlying hemisphere (Figure 8). Another six infants also had a bad outcome due to severe brain damage – four boys and two girls (mean age after birth 2.8 months, corrected for prematurity 2.3 months, median age after birth 1.0 month). Two of these boys (#14, #15) aged 3.5 (corrected age 2.5 months) and 5 months, respectively, had clinical and radiological characteristics compatible with BEH; in both, an objectively recorded, long-lasting respiratory arrest was the cause of the global brain injury. The remaining four infants with a severe brain injury were a prematurely (week 31) born 6-month-old boy (#3), two 1-month-old girls (#6, #17), and a boy aged 0.5 months (#11).

Thus, a total of nine children died or survived with a severe brain injury. Four of these, three girls and one boy, were 1-month old or younger and two of the infants were born prematurely.

Four infants with uncomplicated chronic SDH/hygroma/mixed SDH (#5, #7, #9, #10) had good outcome despite bilateral RH. Two infant boys with bilateral RH and a BEH condition complicated by (cardio-) respiratory arrest had a severe brain damage (#14, #15). All infants with failed circulation had bilateral RH apart from patient #4, who only had a unilateral RH.

3.5 | The BEH-like group

Seven infant boys and a 3.5-year-old boy had findings supporting a diagnosis of BEH, with normal or slight-to-moderately enlarged lateral ventricles without ventricular compression or midline shift, except for patient #7, who had a midline shift of 3 mm. In all these infants, MR imaging revealed a widened extra-cerebral compartment, consisting of an inner compartment with signal compatible with cerebrospinal fluid and an outer layer of fluid with signal compatible with chronic SDH or a hygroma. The IHD was 5 mm or larger in six infants and the CCW and the SCW in all infants ranged from 4 to 11 mm and 5 to 9 mm, respectively. The average measurement for all three distances in all infants was ≥5 mm (Table S1).

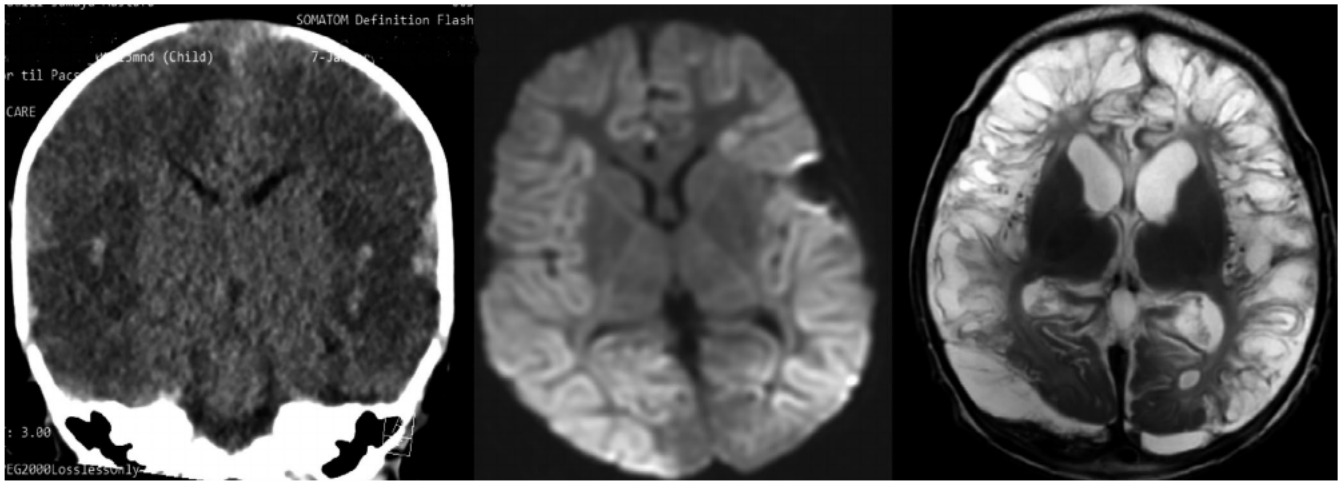

FIGURE 5 Patient #17. Coronal CT scan upon admission (left) shows widespread low attenuating areas with preserved central grey matter structures, indicating a recent event of hypoxia/ischaemia. There is also fresh blood in the subdural and subarachnoid spaces. Diffusion-weighted MR image obtained the day after admission (middle) shows extensive cortical and subcortical cytotoxic oedema, confirming hypoxic/ischaemic lesions. Right: T2-weighted image from a follow-up examination performed 3 weeks later shows widespread parenchymal loss corresponding to previous areas of hypoxia/ischaemia

Moreover, the cortical surface was not compressed, and MR imaging showed a visible layer of cerebrospinal fluid between the subdural fluid collection and the brain surface. The poor quality of the CT of the 3.5-year-old boy (#16) did not allow refined measurements.

In only one case (#13) did the medical experts in court notice and discuss the large head and the possibility of BEH but concluded against this possibility because they believed the rapid and sudden HC increase made that diagnosis unlikely (see Discussion).

3.6 | The HII-like group

All six infants in this group had radiological signs of severe brain damage compatible with HII, characterised by cytotoxic oedema. This comprised the entire cortex and subcortical tissue, but not the basal ganglia or the diencephalon in two infants (#4, #17) (Figure 5), whereas some cortical and subcortical areas were spared in the four remaining infants (Figure 3). Additional findings were acute SDH in one case, chronic SDH in one case, SAH and SDH in one case, and ICH in one case (Table 1).

3.7 | Medical expert witnesses in court

A total of 14 medical expert witnesses had been appointed by the court in these cases. For an overview, see Table S2. In at least four cases (#1, #12, #13, #17), the same specialists in forensic medicine had served as a medical advisor for the police from the beginning of the investigation at the hospital and later in both court levels. The medical experts were mostly recruited from forensic medicine (7), ophthalmology (3) and paediatrics (2).

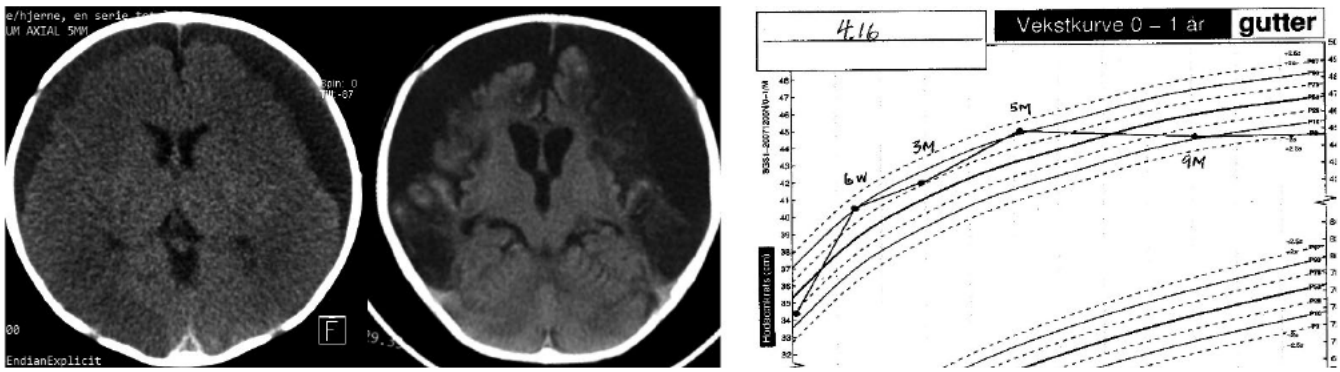

FIGURE 6 Patient #15. The patient developed radiological and clinical characteristic of BEH followed by a secondary brain damage Left: axial CT scan at 5 months shows bilateral low attenuating cerebral parenchyma indicating hypoxic/ischemic damage, with sparing of central grey matter. There is also bilateral subdural hygroma or chronic SDH. Middle: axial MRI scan at 5 months and 3 weeks showing marked brain atrophy. Right: HC growth curve showing an initial rapid growth from birth to 6 weeks of age, followed by an apparent normalisation around the 90% line and a dramatic HC reduction from 5 months of age. Handwritten notes indicate ages in weeks (W) and months (M)

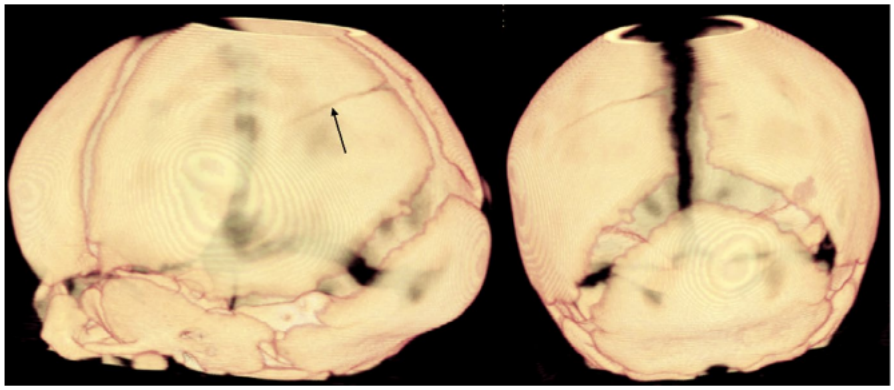

FIGURE 7 Patient #4. 3-D reconstruction (volume rendering technique) shows a non-displaced linear skull fracture in the left parietal region (arrow) and abnormal widening of the skull sutures, indicating high intracranial pressure

Forensic medicine is not a formal medical specialty in Norway, but some nevertheless regard themselves as such specialists and are counted as specialists here.

One paediatrician and two specialists in forensic medicine served in six, seven and six cases, respectively. One of the three was present in 16 of the 17 cases.

In short, a total of seven specialists in forensic medicine served as expert witnesses 18 times, two specialists in paediatrics served nine times with one of them serving in six cases, the remaining medical experts serve 1–2 times each. Physicians with clinical experience in managing infantile head injuries were not appointed to serve as medical experts in court, except for the first author of the present article, who served once.

3.8 | The judicial outcome of the court cases

In only 12 of the cases was a person convicted for violence. In one case, the court was convinced by the experts that violent shaking had taken place but could not decide which of the family members was responsible for the crime; in three cases, including the one above, a person was convicted for not having brought the baby to hospital in time, but not for having caused a criminal act. For an overview, see Table S3.

4 | DISCUSSION

The diagnosis of inflicted head trauma is different from nearly all other diagnoses; it is not solely a medical diagnosis, but also an important background for the court's decision in such cases – in fact the most important premise. In criminal cases involving SBS/AHT, neither the investigators, nor the prosecution authorities or the judges, possess any relevant medical knowledge or insight; consequently, they are completely dependent on advice from medical experts. In principle, the verdict is delivered by the courts, but as the experts' opinion provides important premises for the court decisions, the medical experts in court play a decisive role in the cases. So, to say, the deciding judges can be regarded as lay people in this type of question.

As discussed in the Introduction, it is important to realise that a diagnosis or conviction of SBS/AHT cannot be based on the triad alone. The findings in the triad may occur as caused by shaking or completely independent of shaking. It therefore seems difficult to both prove and disprove SBS/AHT based on medical evidence that is gathered after the alleged crime.

The medical experts in these 17 cases had, with little reservation, concluded that all children had been subjected to an inflicted head trauma, either by violent shaking, blunt head trauma or other forms of violence to the head. The aim of the present study was

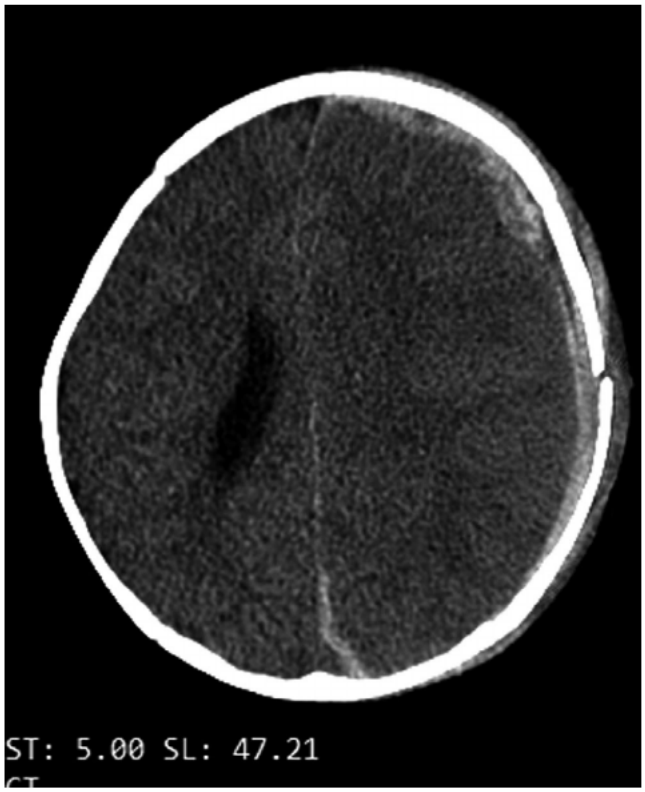

FIGURE 8 Patient #8, a 15-month-old girl with rickets disease. CT shows a left-sided minimally displaced parietal fracture with an underlying acute subdural hematoma. The haematoma causes substantial compression of the left lateral ventricle, effacement of the sulci and dislocation of midline structures to the right

TABLE 1 Overview of all included children. Age in months; (corrected for prematurity)

Pat #	Age	Sex	Premat	Twin	Symptoms and findings	Onset	HC
1	3 (1.5)	M	34 w	No	Small swelling head, focal seizure, irritability, moderate hypocalcaemia	Sudden	2.5%
2	1.5 (0.5)	M	35 w	No	Tender swelling right thigh	Gradual	>97%
3	6 (4)	M	31 w	Yes	Seizures, opistotonus, tense fontanelle, anisocoria	Sudden	No records
4	1	F	Term	No	Unilateral pupillary dilatation, severe hypopotassaemia and acidosis	Sudden	No records
5	3	F	Term	No	Short fall, LOC, bulging fontanelle, pale	Sudden	25%
6	1	F	Term	No	Light head trauma, irritability, seizures, gaze deviation	Sudden	32 cm at birth – <3%
7	2.5	M	Term	Yes	Apathy, reduced consciousness, tense fontanelle	Sudden	25%–50%
8	15	F	Term	No	Fever, LOC, right hemiparesis, seizures, marked hypocalcaemia and low magnesium/ phosphorus	Sudden	From 10–25% to 75%
9	3	F	Term	No	Irritability, reduced consciousness, seizures, hyper-tension, tense fontanelle	Sudden	Rapid increase from 3% to 50–75%
10	1	M	Term	No	Frontal bossing, head lag, sunset gaze, tense fontanelle, hypotonia vomiting	Gradual	>97%
11	0.5	M	Term	No	Irritability, apnoea, O^2 sat 40%, bulging fontanelle, reduced general condition	Sudden	75%, only one measure-ment
12	4	M	Term	No	LOC sudden stiffening	Sudden	97%
13	2.5	M	37 w	No	Short fall, cried, opistotonus, tense fontanelle	Sudden	Rapid growth to >97%
14	3.5 (2.5)	M	35 w	Yes	LOC, sudden stiffening, cardiorespiratory arrest 15 min, Severe hypoxia, tense fontanelle	Sudden	>97%
15	5	M	Term	No	Apnoea, reduced consciousness, bulging fontanelle vomiting, seizures, status epilepticus, O^2 sat. 50%	Gradual	>90%, crossing 3 percentile lines
16	3.5 years	M	?	No	Seizure, fall, cardiorespiratory arrest, GCS 3	Sudden	>97%
17	1	F	Term	No	Somnolence, seizure, tense fontanelle	Sudden	50%–85%

Abbreviations: ASDH, acute subdural haematoma; BEH, benign external hydrocephalus; CSDH, chronic subdural haematoma; F, female; HII, hypoxic ischaemic injury; LOC, loss of consciousness; M, male; O^2 sat, O^2 saturation; SDH, subdural haematoma; SDHy, subdural hygroma.

therefore to go through the medical documentation to see if alternative diagnoses existed.

4.1 | Epidemiological characteristics

There was a striking male preponderance, almost two-thirds were boys, and most of these boys had clinical and radiological characteristics compatible with external hydrocephalus – BEH/BESS – complicated by a chronic SDH or hygroma. Such haematomas or hygromas are common complications to BEH. Numerous studies have shown a similar gender distribution in BEH populations as in the present material.[9,11,28-37] In the only population-based epidemiological study on BEH, the Norwegian male preponderance was even higher – 86.4%.[38] Adamsbaum et al[3] and Vinchon et al[4] reported large numbers of allegedly abused children, with a pooled population of 157 infants. This population had a similar marked male preponderance (73%) as in populations of infants diagnosed with BEH. This similarity indicates that BEH may have been misdiagnosed as AHT/SBS. Such cases have indeed been reported, and several authors have warned that BEH may

RH	Signs of impact to skull	Fractures	Radiological findings	Outcome
No	Head swelling overlying linear skull fracture	Left temporal cranial fracture, three rib fractures with callus	Small local SDH	Moderately delayed psychomotor development
Unilat	Small blue mark forehead	Femur fracture, Rib fractures with callus	BEH-like Bilateral CSDH/SDHy	Good
Bilat	None	None	ASDH/brain oedema, HII	Severe brain damage
Unilat		Small linear skull fracture	Intracerebral haematoma, suture diastasis, HII	Died
Bilat	None	None	Mixed SDH (CSDH/ASDH), 4 mm midline shift	Good
Bilat	Blue marks on face and body	Clavicle, old rib fractures with callus	Large left hemisphere infarction, HII	Severe brain damage
Bilat		None	BEH-like CSDH/SDHy	Good
Bilat	Skin swelling over cranial fracture	Cranial fracture	ASDH, cortical vein thromboses? Skeletal signs of rickets	Severe brain damage
Bilat	None	None	Bilateral CSDH, early signs of hypoxia and ischemia, HII	Probably good
2 and 3 small RH	Small blue mark left cheek	2 rib fractures with callus	BEH-like CSDH	Good
Bilat	None	None	No SDH, bilat. extensive cyto-toxic and vaso-genic oedema, HII	Severe brain damage
Small unilat	Small blue mark left cheek	None	BEH-like CSDH, thrombosed veins	Good
None	None	Rib fractures with callus	BEH-like CSDH	Good
Bilateral	None	None	BEH-like mixed SDH, mostly CSDH	Severe brain damage
One small unilateral	None	None	BEH-like CSDH Severe hypoxic brain damage	Severe brain damage
None	Swelling left side head	Healed old clavicle fracture	BEH-like Isodense CSDH/ mixed SDH	Died
Bilateral	Swelling left side head over skull fracture	Clavicle fracture with callus, Skull fracture	SAH/SDH suture diastasis, small linear cranial fracture, HII	(Moderate to) severe brain damage

represent a pitfall in the diagnosis of inflicted head injury.[17,19,39] Also, Vinchon et al[20] have demonstrated this predisposition for SDH in BEH.

There was a rather high proportion of prematurely born infants in our population, nearly five-fold that found in the general population. Nearly half – three – of the boys in the BEH-like group were prematurely born. Prematurity is a relatively frequent feature in BEH, reported in up to 20%.[40-42] In our material, the proportion of prematurity was even higher. Thus, the age and sex distributions, as well as the high proportion of prematurity, all point to BEH as a probable cause of the subdural fluid collections.

All infants were 6 months or younger and the infant boys in the BEH-like group had a mean age of 2.9 months (median age 2.5 months). This low age is rather similar to the age at which Norwegian infants with BEH become symptomatic and are diagnosed as such.[38] Mean age of symptom onset in that study was 3.4 months.

Thus, the similarity in gender and age distribution between the BEH-like group and that in a nationwide study of BEH infants adds further doubt about the correctness of the abuse diagnosis in these boys.

The four girls within the HII-like group had a median age of only 1.5 month. The very low age in these girls – and in the BEH-like boys as well – makes one wonder if the birth trauma or some immaturity-related factor could be a common denominator for these conditions.

A large proportion (41.2%) of the children in our material had immigrant parents, about four times higher than the proportion of immigrants in the general population. Possible explanations for this disproportionally high number of immigrants are that these parents are more prone to use violence against their own offspring, that infants in immigrant families for some reason more easily get intracranial pathological conditions, or that being immigrant parents represents a social stigma that raises suspicion and further investigations of child abuse in cases of intracranial conditions with unknown causes. The latter possibility receives some support from a recent study by Dror et al who conducted an experiment in which forensic pathologists were tested whether knowledge of irrelevant non-medical information, such as skin colour and who the caretaker was, would influence how they determined the manner of death. Forensic pathologists were more likely to rule 'homicide' rather than 'accident' for deaths of coloured children relative to white children in this hypothetical setting when all *medical* information was kept identical.[43]

4.2 | Initial symptoms and clinical and radiological findings

Nearly half of the included children had macrocephaly or a rapidly increasing HC; this was especially the case in seven of the eight boys in the BEH-like group. A nationwide study has shown that an abnormal HC growth most commonly is caused by hydrocephalus.[44] As most children in our material were hospitalised very soon (hours) after an acute event/worsening, family members were consequently suspected of having performed an act of violence shortly before this hospitalisation. After such a sequence of events, one would expect to find neuroimaging compatible with an acute incident, such as an acute SDH; one would not expect a chronic condition, such as a hygroma or a chronic SDH. That is, however, exactly what the neuroimaging shows in all infants with an increased HC – a chronic SDH or a hygroma. An acutely acquired intracranial expansive process, such as acute SDH, will not cause an abnormal head growth; only if an intracranial expansion is present over time, will it eventually result in an abnormally increased HC.

It follows from this that it is rather unlikely that the eight infants with a large or rapidly growing head had been subjected to an act of violence shortly before the acute, clinical worsening. More likely, these infants had a chronic condition with increasing ICP that ultimately caused seizures or precipitated an intracranial bleeding, for instance, an acute re-bleeding in a chronic SDH, see below.

Most of the included children had seizures as an initial symptom; seizure was thus the most frequent symptom. Seizures are frequently reported in infants with BEH, especially in combination with this condition's most common complication – SDH.[7,9,12,17,28,37,45-49]

Vomiting is a non-specific symptom that also can be caused by increased ICP. In the two infants where vomiting was noted, it was not an acute event, but a gradually increasing phenomenon over the last days or weeks before hospital admission, indicating a gradual build-up of the ICP. Both these infants had a bulging or tense fontanelle as a sign of increased ICP with a gradual, not sudden, symptom onset.

4.3 | Clinical findings

The majority (12) of the infants had no signs of impact to the neurocranium. The impact signs in the remaining five were a blue mark on the forehead in one infant, scalp swelling after a documented fall in another child, and scalp swelling in combination with an underlying skull fracture in three, two of these were asymptomatic fractures in very young infants – possibly birth-related and disclosed only because suspicion precipitated whole body X-ray. A symptomatic skull fracture was found in a 15-month-old girl (#8) with rickets disease – marked hypocalcaemia and low magnesium/ phosphorus (see Table 1). In addition, blue marks outside the neurocranium were noted in three infants.

Most of the infants had a tense or bulging fontanelle as a sign of increased ICP. In a substantial portion of these cases, the HC measurements reflected a build-up of ICP over time, as it is a common clinical observation that acute intracranial expansive processes usually do not cause macrocephaly, see discussion about macrocephaly above.

Retinal haemorrhages were also common, mostly bilateral – in nine; three had none. Only three infants with bilateral RHs had a good outcome, six had a bad outcome; this finding thus appears to be an alarming sign.

Retinal haemorrhages has for decades been regarded a cardinal symptom of the SBS triad and has been assumed to be caused by intraocular shearing or traction forces acting directly on the retina. According to the medical reports from the court experts, the presence of RHs was regarded as a very strong indication of violent shaking.

However, another explanation is also possible: that the retinal bleedings are caused by an increased ICP being mediated via the optic nerve sheaths to the ocular fundi. Intraocular bleedings can be seen in patients with a rapidly increasing ICP – the so-called Terson's syndrome.[50] A recent study shows that observed or spontaneously admitted violent shaking did not cause bilateral RHs in healthy infants, in other words: shaking alone does not cause RH,[51] and the presence of RH can therefore not be used to decide whether abuse is the cause of the intracranial pathology. In this context, the study by Finnie et al[52] is of some interest; they subjected young lambs to shaking – none of them developed RH (and only minor SDH).

4.4 | Radiological findings

Signs of HII were detected in six infants. HII is caused by a global hypoxic-ischaemic insult that does not affect all cerebral structures

in the same way, by complex pathophysiologic processes.[53-56] One typical radiological pattern is that cerebral cortex and subcortical white matter are affected, and that deep brain structures, such as the basal ganglia, the thalamus and the cerebellum are spared. This is the predominant imaging pattern in our patients.

HII has also by some authors been explained as the result of abusive head injury. A relation between HII and injury to the brain stem, causing apnoea, has been proposed and pathological studies have found evidence supporting this in some cases.[57,58] Similar HII has also been described after apnoea caused by ALTE/BRUE and BEH.[39,59] However, HII is a pattern of injury, and not specific for the cause of hypoxia or ischaemia; it is not possible, with present imaging techniques, to ascertain the cause of the hypoxic/ischaemic event.

Except for two 1-month-old girls (#6, #17), none of the infants in this group had any signs of impact to the skull or other external signs of trauma. Both girls had clavicle fracture with callus formation, one had also a small linear cranial fracture and the other had old rib fractures with callus. Four of the infants (#4, #6, #11, #17) were 1-month-old or even younger, an age that indicate a causal relationship with the birth process.

Although the radiological findings in this group were typical of the HII condition,[60] none of the medical experts mentioned this condition in their reports or during their testimony in court, with one exception (#17). On the contrary, they concluded that the brain damage in all these infants was caused by violent shaking and/or blunt traumas.

In short, all infants in this group had neuroimaging compatible with a HII, a condition which is non-specific and does not prove a traumatic genesis. The medical experts in court did not discuss or mention that there are alternative causes for this injury pattern. In this respect, it is of some interest that the child abuse literature often emphasises that abuse is not diagnosed until all possible alternative conditions have been excluded, see the statement given by Choudhary et al[61] who wrote that the workup must exclude medical diseases that can mimic AHT. It seems questionable whether the court experts followed this advice.

According to the medical experts in court, all the infants with subdural fluid collections had been subjected to violence shortly before hospitalisation. The initial neuroimaging studies in the included cases were performed in close relation to the initial symptoms, and one would therefore expect to find an acute SDH in these cases, with compression of the ipsilateral brain and ventricle and a midline shift when unilateral, and compression of both hemispheres and lateral ventricles if bilateral, as usually seen in traumatic acute SDH. However, these infants' neuroimaging failed to reveal features of an acutely acquired haematoma caused by external traumatic forces. Acute, coagulated blood was not predominant, and the extra-cerebral fluid seemed more compatible with chronic subdural hygroma or chronic SDH, with scattered small amounts of coagulated blood in the chronic fluid. Moreover, these chronic subdural fluid collections did not exert any mass effect on the cortical surface or the lateral ventricles and did not cause a midline shift when unilateral. Choudhary et al[61] claim that mixed density SDH is a frequent finding in AHT: 'Mixed-attenuation subdural hematomas are found with greater prevalence in AHT than in accidental head trauma.' Maybe this consensus statement is the reason for the discrepancy discussed above.

An acute SDH was found in only two children; a *chronic* subdural haematoma (chronic SDH) or hygroma on the other hand, was much more common, present in 10 infants and with a male preponderance. This discrepancy between the radiological appearance of a chronic subdural fluid collection and the short time lapse from the assumed act of violence is indeed intriguing; one possible explanation could be that the fluid in many of these cases simply was described as a 'subdural haematoma' by the radiologist, without any further specifications regarding age of the SDH and that the medical experts assumed this meant an acute subdural haematoma.

All the seven infants in the BEH-like group had radiological characteristics compatible with BEH complicated by a chronic SDH/hygroma. The predisposition for SDH/hygroma is well-known in infants with BEH.[6-20] Lee et al[11] demonstrated this predisposition in almost 10% of their infants with benign extracerebral fluid collection. Several studies show that BEH complicated by SDH/ subdural hygroma has been misdiagnosed as SBS/AHT, often with the most severe medical and above all, social/legal consequences.[15,17,39,62]

In only one case did the medical experts in court notice and discuss the large head and the possibility of BEH (#13), but concluded against this possibility because the HC increase had occurred suddenly, which they believed made that diagnosis unlikely; however, at that time it had been known for some years that the HC growth in BEH usually does not start immediately at birth, but after some months.[37,41,63] Most infants in the BEH-like group had a similar development; they were born with a normal HC that started to grow too rapidly in the following months.

Further, nearly half of these infants were prematurely born, which is a well-known predisposing factor for developing BEH.[40-42] In all other settings than the present, where AHT is suspected, external hydrocephalus would have been the diagnosis. This applies also to the 3.5-year-old boy who died with an isodense SDH after a fall.

Thus, it can be concluded that findings supporting a diagnosis of BEH were made in these eight boys. Similarly, it can be concluded that the medical experts in court disregarded this diagnosis and misdiagnosed the condition as a result of abuse.

The designation 'benign' in BEH or BESS is unfortunately misleading, as it predisposes for serious complications, such as SDH, mostly chronic SDH and seizures as discussed above.

With only two exceptions (#2, #8), the fractures in the present material were asymptomatic and *found only because inflicted head trauma was suspected* for other reasons. This applies to all rib fractures, two cranial fractures and clavicle fractures with callus.

Three infants had asymptomatic linear cranial fractures: a prematurely (week 35) born boy (#1) aged 3 (corrected 1.5) months and two 1-month-old girls (#4, #17). The low age, at least of the latter two, indicates birth trauma as a possible mechanism behind the fracture. Dynamic MRI studies of the birth process have shown dramatic

distortions/misconfiguration of the head during its passage through the birth canal[64]; that these forces are strong enough to cause an asymptomatic fracture of one or more of these thin bony plates may not be surprising. This assumption is supported by the estimates of Nachtergaele et al[65] that between 2.9% and 10% of all newborns have a cranial fracture.

None of the rib fractures had given any symptoms; they were found only because abuse was suspected for other reasons. Three of the five infants with rib fractures were born prematurely, and the fact that nearly all fractures had callus formation and the infants had a mean age of 1.8 months, indicates that these fractures could have been caused by the birth process. Most likely, rib fractures are clinically silent in newborns and consequently, the incidence of rib fractures underestimated.[66]This renders it likely that birth-related rib fractures are much more common than previously assumed; they are however not looked for routinely – only when inflicted head trauma is suspected.

The only symptomatic fracture was the femur fracture in a moderately premature (35 weeks) 1.5-month-old boy. This fracture was discovered by the parents because they had observed a tender swelling of the thigh two days after an Ortolani's test for hip dysplasia. It is possible that this manoeuvre, in combination with bone fragility/low calcium levels that has been reported in prematures,[67,68] caused this fracture.

Clavicle fracture is the most common birth-related fracture – in 5% of newborns.[66] The two infants with a clavicle fracture in our material were only one month of age and the fracture had callus; thus, most likely these fractures were caused by a birth trauma.

4.5 | Clinical outcome

Two patients died: a 1-month-old girl (#4) with an intracerebral hematoma and a 3.5-year-old boy (#16) with clinical and radiological characteristics of BEH, who sustained a large isodense SDH that did not compress the hemispheres, probably after a seizure that resulted in a fall from own height and impact to the head.

A total of seven children survived with a severe brain injury: a 15-month-old girl (#8) with a manifest rickets disease with hypocalcaemia and low values of phosphorus and magnesium. She sustained a cranial fracture and an acute SDH with a cytotoxic oedema of the underlying hemisphere, possibly after an unwitnessed fall.

Another six infants also had a bad outcome due to severe brain damage – four boys and two girls (mean age 2.8 months, median age 2.3 months). Two of these boys (#14, #15) aged 3.5 and 5 months, respectively, had clinical and radiological characteristics compatible with BEH; in both, an objectively recorded, long-lasting respiratory arrest was the cause of the global brain injury. The remaining four infants with a severe brain injury were a prematurely (week 31) born 6-month-old boy (#3), two 1-month-old girls (#6, #17) and a boy aged 0.5 month (#11).

Thus, a total of nine infants died or survived with severe brain injury. Four of these, three girls and one boy, were 1-month-old or younger.

Four infants with uncomplicated chronic SDH/hygroma/mixed SDH (#5, #7, #9, #10) had good outcome despite bilateral RH. All infants with HII had bilateral RH, apart from patient #4, who only had a unilateral RH.

4.6 | Expert witnesses appointed by the court

Three expert witnesses served in most of the included cases. It seems as if very few, if any, of the medical experts in court had practical, clinical experience with handling of severe head injuries in infants. In at least four cases, the same specialists in forensic medicine had served as medical experts for the investigating authorities and later in both court levels. Such a practice is associated with bias, for example, confirmation bias, and has therefore been heavily criticised.[69]

4.7 | Strengths and limitations

This report is based on cases within the Norwegian legal system, in which a small number of court-appointed medical experts serve in most cases. The applicability of our findings to other legal systems may therefore be limited. The strength of the present study is that it is nationwide and thus covers all judicial regions over a 12-year period, with good access to medical, as well as judicial documentation at an individual level. A limitation may be that we cannot guarantee that all cases are court cases have been included in the national court system's data base. However, it appears quite unlikely that this could have caused any bias in the selection of cases.

5 | CONCLUSION

Our results indicate that the head injuries in the majority of the investigated children possibly, or even probably, had a non-traumatic cause, and consequently, that these children not necessarily had been subjected to an inflicted head injury caused by shaking or direct impact. We emphasise that we cannot, based on the present study, rule out that these children had been shaken, in the same way as it is impossible to prove that any of them had been shaken, based only on clinical and radiological findings made *after* the alleged violence.

There appears to be a need for a strict investigation protocol for cases of alleged SBS/AHT. In addition, we believe that information on HC development should be available and that any multidisciplinary team to analyse such cases should include colleagues with practical clinical experience in handling and diagnosing head injuries in children. In the present study, such professionals were virtually non-existent. Most experts in court were specialists in forensic medicine or paediatrics without such clinical experience.

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CONFLICT OF INTEREST

KW has served as an expert witness for the court (paid) and the defence (unpaid) in a few cases of suspected abusive head injury in Norwegian courts. JW has served as a mostly unpaid expert witness for the prosecution and the defence in a few cases in Norway and Sweden. AS and US declare no conflict of interest.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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