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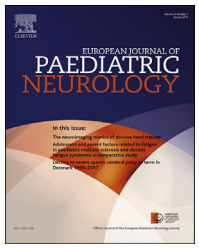

Review article

The neuroimaging mimics of abusive head trauma

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ABSTRACT

Abusive head trauma (AHT) is a significant cause of morbidity and mortality in the paediatric population, typically in children under the age of two years. Neuroimaging plays a key role in the diagnostic work up of these patients as information regarding the mechanism of injury is often lacking and the findings on examination can be nonspecific.

A number of conditions, both traumatic and atraumatic can mimic AHT based on neuroimaging features alone. The repercussions associated with a diagnosis or misdiagnosis of AHT can be severe and radiologists therefore need to be aware of and familiar with the imaging differentials of AHT.

In this paper we review the imaging findings of the radiological mimics of AHT and focus on features that can help differentiate these entities from AHT.

Contents

Abbreviations: AHT, Abusive head trauma; AAP, American Academy of Pediatrics; AVM, Arteriovenous malformation; BESS, Benign enlargement of the subarachnoid space; CSF, Cerebrospinal fluid; CT, Computed tomography; DWI, Diffusion weighted imaging; EPI, Echo planar imaging; GA1, Glutaric aciduria 1; HASTE, Half Fourier acquisition single-shot turbo spin echo; MRI, Magnetic resonance imaging; NAI, Non-accidental injury; OI, Osteogenesis imperfect; RH, Retinal haemorrhage; RCR, Royal College of Radiologists; SBS, Shaken baby syndrome; SDH, Subdural haemorrhage; VST, Venous sinus thrombosis; DD, Vitamin D insufficiency and deficiency.

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1. Introduction

The term AHT encompasses the spectrum of neurological injury that is associated with shaking or blunt trauma in young children, typically under the age of two years.[1] It has also been described as non-accidental head injury (NAHI) and shaken baby syndrome (SBS) in the literature.

The incidence of AHT is reported to be in the range of 14.7–39.8 cases per 100,000 children.[2,3] This figure is however likely to be an underestimation of the true prevalence of AHT.

Research has led to a number of proposed but conflicting pathophysiological theories and hypotheses pertaining to the biomechanical (shaking, whiplash and/or impact), histopathological (primary and secondary injury, including hypoxic injury) and clinical features associated with AHT. This, in combination with a lack of consistent and established diagnostic criteria means that AHT remains a topic that attracts significant controversy from both a diagnostic and medicolegal perspective.[4]

Brain injury is the leading cause of mortality in the context of inflicted trauma.[5] The outlook and prognosis associated with AHT is poor. There is a higher overall mortality and incidence of neurological impairment in this subset of patients when compared to those with accidental head trauma.[6]

A retrospective study of 173 cases of confirmed AHT by Jenny et al. identified a 13% rate of initial misdiagnosis based on misinterpretation of neuroimaging alone.[7] In the context of the potential repercussions associated with a diagnosis or misdiagnosis of AHT this is a significant figure.

An incorrect diagnosis can be associated with long lasting stigma for the child and the family and can have medicolegal implications for the clinicians and non-clinical care workers involved with the case. Numerous case reports, published papers and a relatively recent high-profile court case from the United Kingdom (UK) highlight this specific point.[8,9]

The triad of acute encephalopathy and retinal haemorrhage (RH) in conjunction with subdural haemorrhage (SDH) on neuroimaging was previously perceived to be pathognomonic for diagnosing AHT. This view is controversial as the presence of the triad does not enable a diagnosis of AHT to be confidently made in the absence of a detailed history and examination. Neither can a diagnosis of AHT be excluded on the ground that one or more of these findings is absent.

Relevant history and information regarding the mechanism of injury associated with AHT is often lacking. The clinical manifestations of AHT are variable and depending on the mechanism of injury, evidence of significant external injury may be absent in up to 40% of cases.[5] These factors add to the complexity of imaging interpretation from the perspective of the radiologist.

Due to the non-specific nature of presentation a high index of suspicion is required when considering a diagnosis of AHT. Imaging plays a key role in the assessment of the pattern of injury, providing corroborative diagnostic and prognostic information.[10–12] A recent comprehensive review of the neuroradiological findings associated with AHT has been published by Wright.[13]

In conjunction with clinical examination, neuroradiological investigations, predominantly computed tomography (CT) and magnetic resonance imaging (MRI) play a complementary role with each modality having its own benefits and limitations.[14]

2. Current guidance on investigation of AHT and imaging protocols (Table 1)

Occult cranial, intracranial and spinal injuries are often associated with extracranial physical abuse and thus neurological imaging should be performed in the cases where there is evidence of such trauma.[15,16]

Current guidance from the American Academy of Pediatrics (AAP) outlines the requirement for both laboratory and neuroradiological testing in cases of suspected AHT. In addition to routine laboratory tests, there are specific recommendations for the evaluation of clotting (including specific

Table 1 – Proposed laboratory and neuroradiological tests in cases of suspected AHT.

Proposed clinical and imaging diagnostic work-up for suspected AHT		
	AAP (USA)	RCR (UK)
Laboratory Tests	Haematological testing to include a complete blood count, clotting screen, factor VIII/IX levels, fibrinogen, d-dimer Review of newborn screen Urinary organic acids for GA1	
Neuroradiological Tests	CT Head in the acute setting MRI brain and whole spine	CT Head in the acute setting MRI brain and whole spine at days 2–5

factor assays), a review of the newborn screen and evaluation for urinary organic acids. Imaging recommendations include cranial CT and MR imaging of the brain and whole spine.[17]

Current guidance from the Royal College of Radiologists (RCR) of the United Kingdom on investigation of suspected physical abuse in children advises unenhanced cranial CT imaging as the first line neuroradiological investigation. This should be performed routinely for children under one year of age presenting with suspected abusive injury and should be performed in children over the age of one who have external evidence of head trauma or abnormal neurological findings on clinical examination.

MRI is recommended at days 2–5 following presentation when there is CT confirmed intracranial haemorrhage, parenchymal injury or skull fracture. Furthermore MRI is also recommended in cases where there are ongoing neurological symptoms despite a normal CT scan. MR imaging of the whole spine should be performed at the same time as cranial imaging.[18]

Whilst more time consuming, standard MRI has been shown to have superior sensitivity in detecting intracranial pathology when compared with CT and ultrafast MRI sequences, limited to T2 HASTE axial and coronal, DWI and EPI T2*.[19] Limited sequences are therefore not recommended. In cases of suspected AHT we advocate the inclusion of T2* or susceptibility weighted sequences in addition to routine sequences. FLAIR (Fluid Attenuated Inversion Recovery) sequence may be particularly useful in detecting subtle subdural collections. In terms of spinal imaging, fat suppressed sequences can improve detection of subtle vertebral trauma, as well as subdural collections.

3. Differential diagnoses and their imaging phenotype (Table 2)

3.1. *Accidental or non-abusive head trauma (Fig. 1)*

The primary differential diagnosis of AHT is accidental head trauma.[20] There is an overlap of the spectrum of radiological findings between these two entities. Intracranial haemorrhage and skull fractures are commonly seen with both forms of injury.

Based on case series and systematic reviews there are certain patterns of intracranial haemorrhage which are significantly associated with AHT and therefore may enable more accurate radiological discrimination between abusive and non-abusive head trauma.

SDH is the hallmark of AHT, present in up to 89% of cases based on prospective studies.[21] Proposed pathophysiological theories for this finding is that haemorrhage occurs as a result of rupture of superficial cerebral bridging veins at the point of dural insertion or effacement of the venous sinuses as a result of brain swelling.[22,23] The 'lollipop' and 'tadpole' signs have been used to describe the finding of thrombosed cortical veins on MR imaging. Specifically, interhemispheric, convexity and posterior fossa SDH is significantly associated with AHT, particularly when combined with findings of brain parenchymal injury and retinal haemorrhage on clinical examination.[24] The characteristics of SDH associated with AHT, both acute and subacute has been reviewed comprehensively recently.[13]

Findings from systematic reviews have demonstrated that the presence of epidural haemorrhage in the context of an isolated skull fracture and scalp swelling is significantly associated with accidental head trauma.[24,25] Subarachnoid haemorrhage as a solitary finding is non-discriminatory.[25]

Parenchymal contusions and diffuse axonal injury are uncommon findings in children with AHT.[26]

3.2. *Birth related intracranial haemorrhage*

This entity is associated with various forms of delivery, both instrumented and non-instrumented.

In a series of 101 asymptomatic term neonates imaged by MRI within the first 72 h of life, Rooks et al. identified a 46% incidence of SDH, typically small volume (up to 4 mm) and located posteriorly. The distribution of birth related SDH in these cases was demonstrated to be variable, typically multifocal and involving both the supra and infratentorial compartments and best appreciated on coronal imaging. Subarachnoid, epidural and parenchymal haemorrhage was not a feature. On follow-up imaging, performed in 39% of cases of confirmed SDH at index imaging there was complete resolution of haemorrhage at 3 months.[27]

The exact pathophysiology of this entity is not clear. Injury or tears of the cortical bridging veins, tears of the falx and tentorium, remodelling and compressive forces during vaginal delivery are all proposed mechanisms leading to SDH in this population.[28–30]

It should be noted that the distribution of SDH associated with birth can overlap with haemorrhage seen in the context of AHT as reported by Holden et al. in a case of interhemispheric SDH in an asymptomatic neonate. Distribution of blood alone is therefore an unreliable indicator for the mechanism of haemorrhage.[31]

Whilst a small minority of birth related SDH identified on early imaging persisted beyond one month in the study performed by Rooks et al., the majority of SDH resolved by one month. Resolution of birth associated SDH within 4 weeks was noted in all 9 cases in a study performed by Whitby et al.[32] We

Table 2 – Patterns of intracranial haemorrhage associated with AHT and its mimics and distinguishing features on neuroimaging.

Distinguishing neuroimaging features of intracranial haemorrhage in AHT and mimics	
Entity	Pattern and features of associated haemorrhage
AHT	Subdural distribution of haemorrhage. Specifically, • Interhemispheric •Convexity • Posterior fossa Review imaging for • 'Tadpole sign' • Retinal haemorrhage
Accidental head injury	Intra and extra-axial distribution of haemorrhage. The extra-axial component is typically extradural. Subarachnoid haemorrhage may also be present, although non-discriminatory in isolation Review imaging for • Isolated skull fracture • Scalp swelling
Birth-associated SDH	Subdural distribution of haemorrhage, typically posterior distribution. Tends to resolve within 4 weeks. Temporally associated with the delivery of the child. Typically asymptomatic.
Infection	Intra or rarely extra-axial haemorrhage may be present. Review imaging for •Cerebritis • Venous sinus thrombosis • Extra-axial collections
Vitamin D deficiency	Intracranial haemorrhage is uncommon. The presence of other fractures which bear a high specificity for non-accidental injury should raise concern for AHT. Review imaging for • Sutural diastasis • Other fractures on skeletal survey
Osteogenesis imperfecta	Subdural distribution of haemorrhage Review imaging for • Wormian bones • Other features on skeletal survey - platyspondyly, gracile bones and fractures
Haemorrhagic disease of the newborn/bleeding diathesis	Variable pattern of haemorrhage which requires correlation with laboratory testing. Vitamin K may not be given at birth.
Benign enlargement of the subarachnoid spaces	Subdural distribution of haemorrhage Review imaging for • Macrocephaly • Enlargement of the subarachnoid space over the frontal lobes, frontoparietal areas with a prominent interhemispheric fissure and normal or mildly enlarged ventricles.
Glutaric aciduria (GA1)	Subdural distribution of haemorrhage in conjunction with subdural collections Review imaging for • Widening of the Sylvian fissures with poor opercularization. • Expansion of the cerebrospinal fluid space anterior to the temporal lobes, widening of the basal cisterns. • Diffuse white matter signal abnormality and variable basal ganglia changes on MRI.
Menkes disease	Subdural distribution of haemorrhage in conjunction with subdural collections Review imaging for • Wormian bones and skull fractures • Tortuous arteries • White matter lesions which may be tumefactive or non-tumefactive, focal parenchymal DWI hyperintensity, basal ganglia abnormalities and parenchymal atrophy.
Venous sinus thrombosis	Intraparenchymal or extra-axial haemorrhage

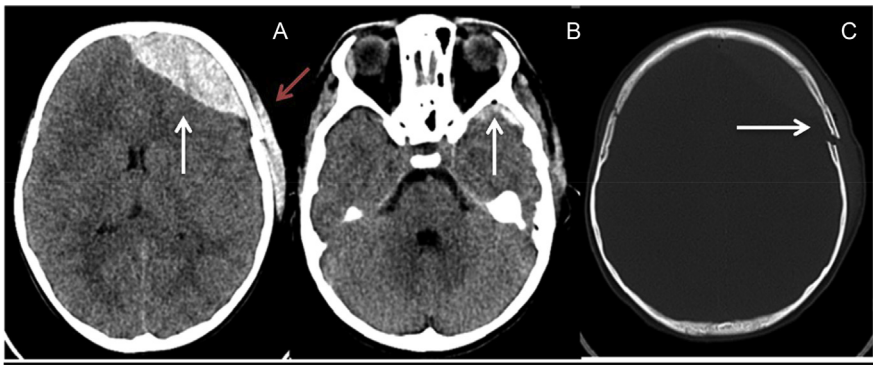

Fig. 1 – Accidental head trauma. CT of 3 year old female patient who fell out of a first floor window demonstrates the typical findings associated with accidental trauma. Incidentally this child was also found to have left sided retinal haemorrhage. There is a large hyperacute left frontal epidural hematoma with mass effect (A, white arrow). This is associated with a left temporal subdural hematoma, small volume pneumocephalus (B, white arrow) and a left subgaleal haematoma (A, red arrow). There is a linear fractures of the left parietal bone which extends into the widened left coronal suture (C, white arrow). (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

therefore conclude that SDH in a child over the age of one month should not be wholly attributed to birth related trauma and warrants further investigation with a view to excluding AHT as a potential cause.

3.3. *Haemorrhagic disease of the newborn and bleeding diathesis*

Spontaneous intracranial haemorrhage may occur in newborns who have not received vitamin K at the time of birth.[33]

In children with an underlying bleeding diathesis intracranial haemorrhage may occur as a spontaneous phenomenon or following minor trauma.[34,35] The haemorrhage associated with coagulopathy may be intra- or extra-axial in distribution, is often associated with cerebral oedema and can mimic AHT (Fig. 5).

Imaging alone is not pathognomonic in these cases due to the variable pattern of haemorrhage associated with this entity. There is therefore a higher reliance upon clinical features, history and laboratory findings to reach the correct diagnosis.

As noted previously, laboratory testing in suspected AHT should include work up for bleeding diathesis.

3.4. *Infection*

Infection is overwhelmingly common in the paediatric population, both bacterial and viral. Clinically, intracranial infection may mimic AHT as infants present with encephalopathy. Radiologically, brain oedema and SDH can be present on neuroimaging.[4] Other less common patterns of intracranial haemorrhage, such as intraparenchymal haemorrhage have been described in the context of herpes simplex virus associated encephalitis in infancy.[36] All sequences should therefore be thoroughly interrogated to assess for evidence of cerebritis.

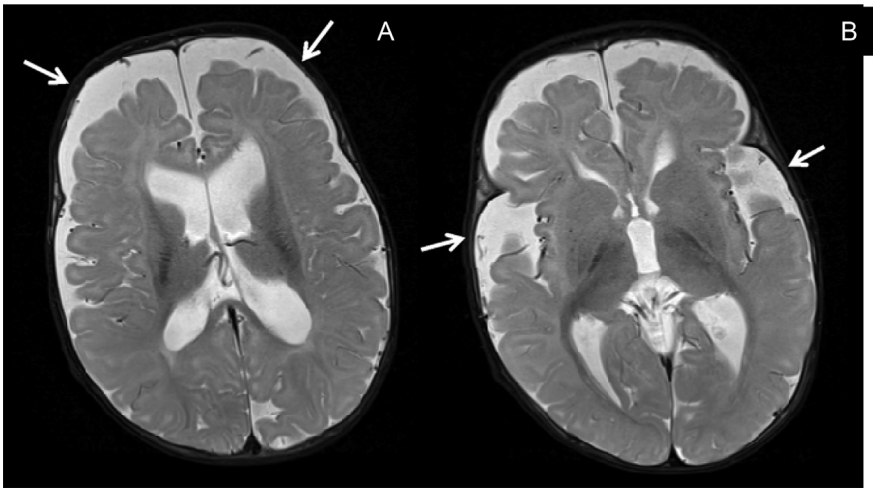

Fig. 2 – Benign enlargement of the subarachnoid spaces. Axial T2-weighted MRI of a 6 month old female patient with asymptomatic macrocrania clinically demonstrates prominent subarachnoid spaces anterior to the frontal and temporal lobes and a widened interhemispheric fissure (white arrows). The ventricular system is prominent. The brain parenchyma is within normal limits. A diagnosis of BESS was made.

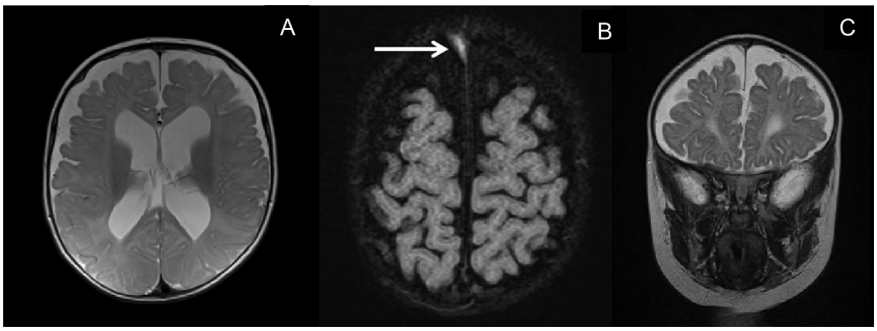

Fig. 3 – Benign enlargement of the subarachnoid spaces. Axial T2-weighted (A), FLAIR (B) and coronal T2-weighted (C) imaging of a 7 month old male with asymptomatic macrocrania demonstrates prominent subarachnoid spaces anterior to the frontal lobes and a widened interhemispheric fissure. A small SDH is incidentally noted (B, white arrow). There was no history of trauma or clinical features to suggest AHT. A diagnosis of BESS was made.

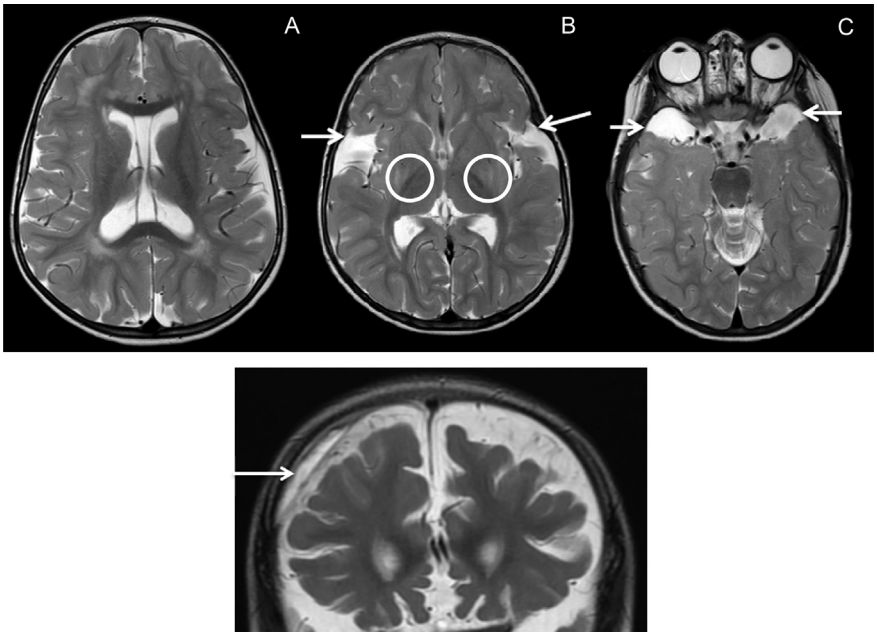

Fig. 4 – Glutaric aciduria 1. Axial T2-weighted MRI of a 31 month old female patient with afebrile convulsions and developmental delay demonstrates under opercularization and small frontal lobes (A and C, white arrows). Patchy areas of signal change seen in the subcortical and deep white matter particularly of the frontal lobes bilaterally as well as mild delay in the maturation of myelin. Subtle increase in signal is seen within the posterior putamen (B, white circles). Metabolic screen confirmed a diagnosis of glutaric aciduria I. Follow up imaging in the same patient demonstrates the presence of an incidental shallow right convexity subdural collection which could be misinterpreted as AHT.

3.5. *Fractures and anatomical variants*

No single or particular pattern of skull fractures has been found to be specific for AHT.[37] Furthermore, based on the mechanism of inflicted trauma there may be significant intracranial injury in the absence of skull fracture, for example in the context of shaking.[38]

Accessory sutures are a common anatomical variant which occur as a result of anomalous fusion and separation of ossification centres.[39] A number of accessory sutures have been described, both within the calvarium and skull base. A complete discussion of all the possible accessory sutures is beyond the scope of this article however there are numerous articles in the literature which cover this topic in detail.[39–41]

Differentiating a fracture from an accessory suture can pose a diagnostic challenge as evidence by numerous case reports available in the literature. In terms of differentiating these entities, fractures tend to be sharply lucent with non-sclerotic edges and cross sutures. Associated soft tissue swelling may be present particularly in the context of trauma.

In contrast, sutures typically have an interdigitated margin with sclerotic borders. A suture does not tend to extend across another suture, and is not associated with diastasis of the other sutures that it approaches. Symmetry or bilaterality may also help discriminate an accessory suture from a fracture.[41]

Wormian bones are accessory bones that occur within a suture. They may be normal variants but can be seen in

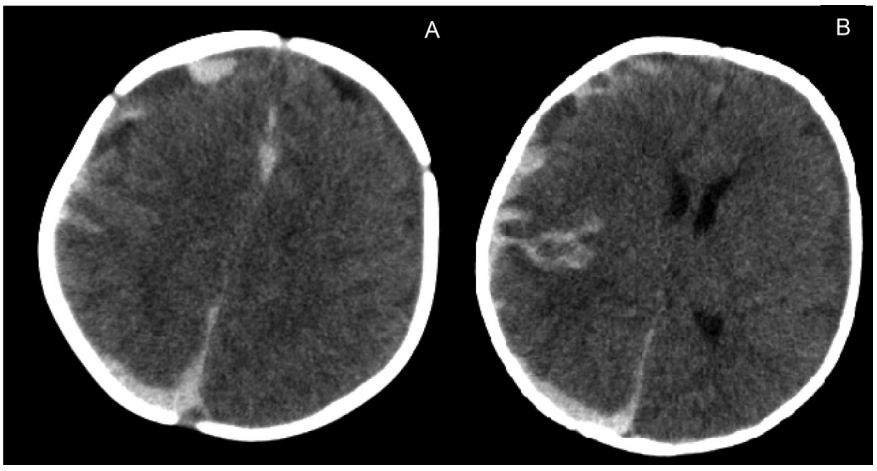

Fig. 5 – Haemorrhagic disease of the newborn. CT of a 6 week old male patient demonstrates a mixed density right cerebral convexity subdural hematoma. Hyperdense subdural blood is seen within the interhemispheric fissure and along the right side of the tentorium cerebelli. There is also an intermediate density left convexity subdural collection. Focal acute subarachnoid haemorrhage is seen particularly within the right sylvian fissure. On haematological testing the vitamin K level was undetectable. There were no features clinically to suggest abusive injury. The haemorrhage was attributed to a bleeding diathesis.

association with a number of conditions including cleidocranial dysplasia, pycnodysostosis and osteogenesis imperfecta.

Congenital skull depressions are a rare, self-limiting entity which have been described in the literature with an incidence of 1 in 10,000 cases. The proposed theory is that the foetal skull is susceptible to deformity and compression from foetal limbs or the maternal pelvis due to its cartilaginous nature. Typically there is no associated fracture.[42]

Surface-rendered 3D CT reconstructions can be an invaluable aid in detecting and discriminating between fractures, accessory sutures and wormian bones.[43]

3.6. *Metabolic bone disease, skeletal dysplasias and connective tissue diseases*

Vitamin D insufficiency and deficiency (DD) is not uncommon in pregnant women although there is conflicting evidence surrounding the link between maternal vitamin D deficiency and impaired bone mineralization in childhood.[44,45]

Rickets, or DD remains a controversial entity in the context of non-accidental injury. There are numerous case reports and series of children with DD presenting with imaging features typical for inflicted injury such as multiple fractures of varying chronicity, metaphyseal fractures, rib fractures and apparent cranial sutural diastasis.[9,46,47]

There is however no definitive evidence to explain or account for the fact that DD in isolation results in the same pattern of fractures that is highly specific for non-accidental injury.[48–50] Similarly, there is no definitive evidence to associate DD with unexplained intracranial haemorrhage (Fig. 7).

Therefore, whilst vitamin D testing should be considered as part of the workup of children presenting with intracranial haemorrhage in the context of suspicious fractures we propose that unexplained intracranial haemorrhage cannot be solely attributed to DD.[51]

Osteogenesis imperfecta (OI) is a common skeletal dysplasia. This condition is caused by glycine mutation affecting the *COL1A1* and *COL1A2* gene on chromosome 17q21.33 and 17q22.1 respectively, disrupting the integrity of collagen formation. Effects include skeletal vulnerability, connective soft tissue laxity as well as scleral and dental discolouration. The inheritance pattern is usually autosomal dominant, affecting up to 1 in 10,000 live births. This is a heterogeneous group of conditions, with up to 8 distinct phenotypes identified.

Intracranial haemorrhage and specifically, subdural haemorrhage, has been reported in a small number of OI Type III, or the progressive deforming subtype, following trivial trauma.[52,53] OI is also associated with wormian bones, which is a finding that would need to be actively sought in a child presenting with intracranial haemorrhage as they may be mistaken for skull fractures. Skeletal survey may also reveal other features that may point towards this diagnosis, such as platyspondyly, decreased bone mineralization, and gracile bones.

Another skeletal dysplasia that is associated with wormian bones is pycnodysostosis. This is an autosomal recessive skeletal dysplasia caused by mutation in active cathepsin K (CTSK) gene located on chromosome 1q21 which results in a defect in osteoclasts implicated in the resorption of bone mineralization leading to reduced bone turnover and consequent increased bone fragility.[54] Again, due to increased bone fragility, patients may have multiple bone fractures suspicious for inflicted trauma.

There are case reports of skull fractures in the context of relatively minor trauma which can be associated with intracranial haemorrhage. As a part of workup, skeletal survey should demonstrate the more typical findings of pycnodysostosis which includes wormian bones, increased bone density or mineralization, spinal curvature and acroosteolysis.[55]

There are a spectrum of connective tissue diseases associated with intracranial aneurysms, dissection, arteriovenous malformations and shunts which have a propensity to bleed.

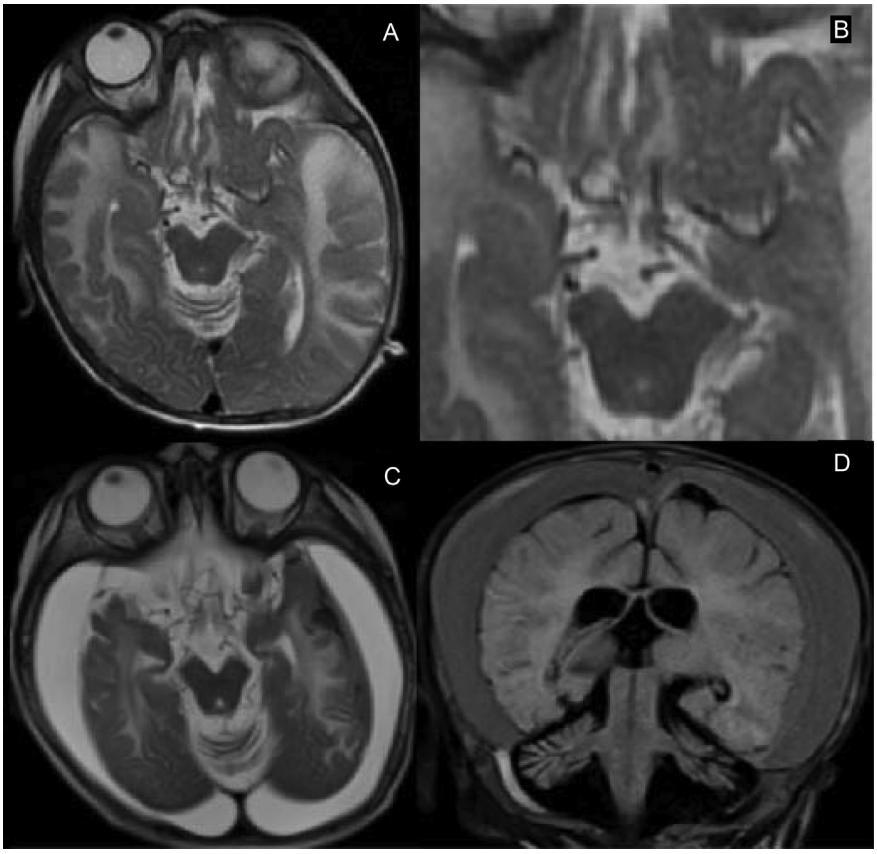

Fig. 6 – Menkes disease. Initial T2-weighted axial images (A and B) demonstrate characteristic features of Menkes disease. There is parenchymal volume loss, white matter signal abnormality within the frontal and temporal lobes. Arterial tortuosity is noted around the circle of Willis. Follow up scanning (C and D) for macrocrania demonstrates bilateral subdural collections. Clinically there were no features to suggest AHT.

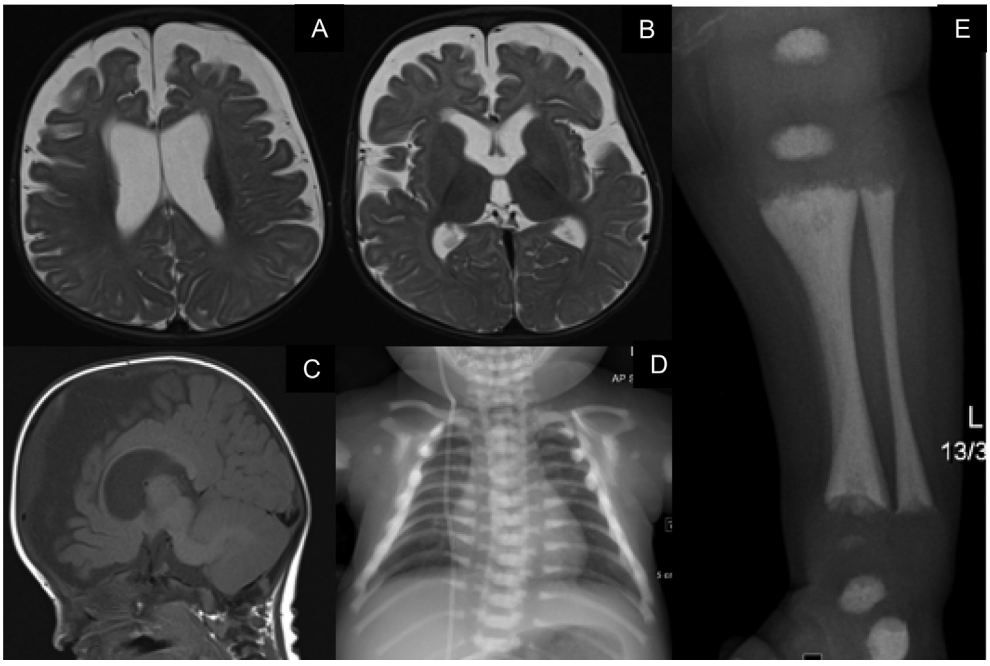

Fig. 7 – Rickets. A 4 month old male was scanned for suspected hydrocephalus. No prior history of trauma of features of abusive injury were identified on clinical examination. Axial T2-weighted (A and B) and sagittal T1-weighted (C) images demonstrate bilateral subdural collections. Radiographs of the chest (D) and left lower limb (E) demonstrate typical features of rickets including metaphyseal cupping and widening of the physes.

These include Ehler-Danlos, Marfan, Neurofibromatosis and Loeys-Dietz disease.[56]

Ehler-Danlos syndrome type IV is an autosomal dominant condition which predisposes the individuals to vessel wall weakness and rupture. This is a result of mutation in the *COL3A1* gene which encodes type III procollagen. Known complications include aneurysms with secondary haemorrhage, carotico-cavernous fistula, as well as arterial dissection.[57]

Marfan syndrome is an autosomal-dominant disorder with musculoskeletal and cardiovascular manifestations due to mutation in the gene encoding fibrillin on chromosome 15, resulting in disruption of the elastin layer. Patients with Marfan syndrome have a predisposition to intracranial aneurysms as well as dissection. Haemorrhagic intracranial events have been reported in the paediatric population, manifesting as subdural hematoma although the underlying mechanism is not clear.[58]

3.7. *Benign enlargement of the subarachnoid space (BESS) (Figs. 2 and 3)*

This is an entity that is also known by a number of names, including but not limited to benign subdural effusions of infancy, benign extra-axial collections of infancy, external hydrocephalus or extraventricular obstructive hydrocephalus. BESS of infancy, as the name suggests, is a self-limiting cause of macrocephaly, with no known genetic predisposition.[59]

The clinical presentation is typically that of macrocephaly with normal development and normal findings on neurological examination. The causative mechanism is not proven; proposed theories include a mismatch between the production and resorption of cerebrospinal fluid, altered venous sinus pressures and restriction of arterial pulsation.[60]

In the context of rapidly increasing head circumference the typical imaging findings are those of enlargement of the subarachnoid space over the frontal lobes, frontoparietal areas with a prominent interhemispheric fissure and normal or mildly enlarged ventricles.[61]

Incidental small SDH are seen in a proportion of these cases scanned purely for the indication of increasing head circumference. A proposed theory for this is increased vulnerability of stretched bridging veins as they traverse the enlarged subarachnoid spaces to pierce into the dura. A number of case series have reported SDH or subdural collections in conjunction with BESS, either spontaneously or following non-abusive trauma indicating the need for further evaluation of these children.[62,63]

3.8. *Cerebral oedema and brain swelling secondary to non-traumatic causes*

Cerebral oedema associated with trauma may occur as a primary phenomenon from parenchymal contusion or infarction associated with traumatic vascular injury. This is usually focal and typically always associated with parenchymal haemorrhage.[64] There is a spectrum of imaging findings which develop over time based on the severity of the insult and development of secondary brain injury.

Patterns of hypoxic ischemic injury (HII) have been described in association with AHT. In a series of 33 infants and young children with non-accidental head trauma, Zimmerman et al. described five patterns of injury demonstrated on DWI.

These include diffuse supratentorial infarction of the both the cortex and white matter of both cerebral hemispheres (39%), watershed infarction of both the supra (36%) and infratentorial brain (6%) and venous infarction secondary to disruption of the bridging veins (12%). Diffuse axonal injury (DAI) and focal parenchymal contusion was seen in only 6% of cases respectively.[26]

Focal and multifocal cerebral oedema can also be seen in the context of encephalitis, demyelination, infarction and seizure related oedema. The differential diagnosis based on imaging findings therefore needs to be kept wide.

3.9. *Neurometabolic conditions*

Glutaric aciduria type 1 (GA1) is a rare autosomal recessive neurometabolic disorder with an estimated prevalence of 1 in 110,000 newborns.[65] Deficiency of glutaryl-CoA dehydrogenase leads to impaired degradation of amino-acids lysine, hydroxylysine and tryptophan resulting in elevated glutaric acid, 3-hydroxyglutaric acid, glutaconic acid and glutarylcarnitine.[66] There are case reports in the literature where GA1 has been misdiagnosed as AHT.[67]

Clinically these children typically have macrocephaly. The characteristic neuroimaging features are widening of the Sylvian fissures with poor opercularization and expansion of the cerebrospinal fluid (CSF) space anterior to the temporal lobes, widening of the basal cisterns, diffuse white matter signal abnormality, and variable basal ganglia changes on MRI.[68]

SDH and subdural collections are associated with GA1 with a reported incidence between 4% and 30%.[69] The proposed mechanism for this is stretching and mechanical stress of the vulnerable bridging veins in the subarachnoid space secondary to widened extra-axial spaces.[70] The underlying brain parenchymal abnormality helps to establish the diagnosis of GA1 and differentiate this entity from AHT (Fig. 4).

Menkes disease, also known as trichopoliodystrophy, is a multisystem genetic disorder which occurs due to a mutation in the *ATP7A* gene. *ATP7A* is a membrane-bound, copper-specific ATPase which transfers copper across the membrane into the lumen of the trans-Golgi network. A mutation in *ATP7A* results in low levels of copper in the central nervous system due to inadequate distribution of the element in the body.[71] There are case reports in the literature of Menkes disease being misdiagnosed as AHT.[72]

The neuroimaging features of Menkes disease are heterogeneous but include vascular abnormalities which manifest as tortuous arteries, white matter lesions which may be tumefactive or non-tumefactive, focal parenchymal DWI hyperintensity, basal ganglia abnormalities and parenchymal atrophy. A summary of the findings associated with Menkes disease has recently been published by Manara et al. (Fig. 6).

Of note, subdural collections may be seen in approximately 25% of cases in association with spontaneous fractures secondary to osteoporosis and wormian bones which may be confused as skull fractures.[73–75]

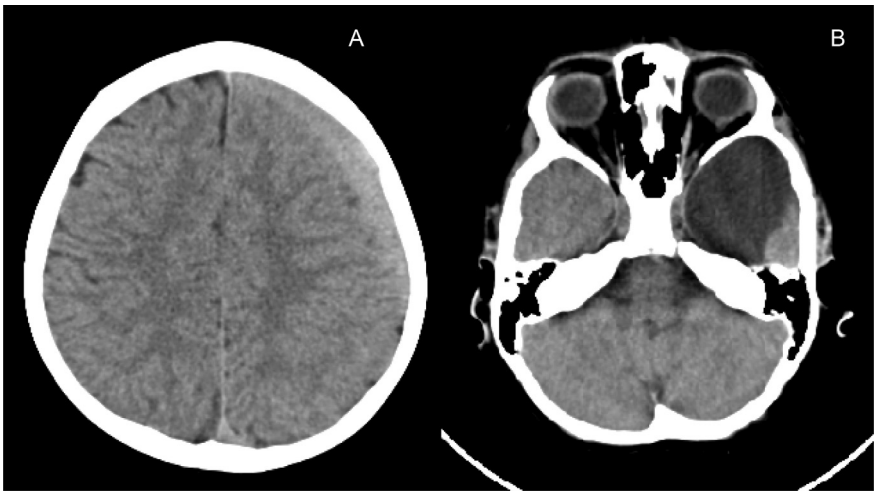

Fig. 8 – Subdural haemorrhage associated with an arachnoid cyst. CT head in a 5 year old male patient demonstrates hyperdense left convexity subdural haemorrhage secondary to a left middle cranial fossa arachnoid cyst. There was no history of trauma.

3.10. *Haemorrhage secondary to venous sinus thrombosis, intracranial vascular malformations and lesions*

Venous sinus thrombosis (VST) is an uncommon cause of extra-axial haemorrahge. In a series of 160 patients, deVeber et al. identified 66 patients with cerebral parenchymal infarcts in the context of VST of which 45 were haemorrhagic (68%). Of these, extra-axial haemorrhage was present in 14 cases (9%), although the exact site of haemorrhage was not recorded.[76]

It is unlikely for venous sinus thrombosis by itself to cause extra-axial haemorrhage, and other associated causes should be considered when it is present along with subdural or epidural haemorrahge.

Spontaneous intracranial haemorrhage may also occur as a consequence of bleeding from a vascular abnormality or an underlying intracranial lesion, such as an arteriovenous malformation (AVM) or arachnoid cyst, respectively.[77,78] In these cases the underlying lesion should be apparent on imaging, either at the time of presentation or interval imaging that is performed following resolution of the intracranial haemorrhage (Fig. 8).

4. Conclusion

AHT is an entity which is likely to be underestimated in terms of prevalence. It has considerable implications not just in terms of morbidity and mortality but also from a social and medicolegal perspective. The pathophysiology of AHT remains poorly understood and the spectrum of presentation of AHT is broad and non-specific.

Whilst thorough clinical evaluation still plays a key role in the work up of AHT, intracranial injury is often occult and therefore neuroimaging has become a standard component in the work up of these patients.

From both a clinical and imaging perspective there are a number of entities which can mimic AHT. In the absence of well-defined diagnostic criteria for differentiating accidental and AHT based on imaging features it is important for the radiologist to be aware of the mimics. The role of the radiologist is to provide a logical and reasoned interpretation of images, stating what conclusions can and cannot be drawn from the imaging findings. Failure to recognise key imaging findings, or misinterpretation of imaging can have severe consequences.

It should be borne in mind that imaging in isolation is not enough to reach the correct diagnosis. The importance of unambiguous and timely communication with the clinical team cannot be emphasized enough in these cases.

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Conflict of interest

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Appendix A. Supplementary data

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