



The Acute Reversal Sign: Comparison of Medical and Non-accidental Injury Patients

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OBJECTIVES: (1) To compare the intracranial computed tomography (CT) appearances of patients admitted with various causes of hypoxic ischaemic encephalopathy. Children with known documented accidental trauma were excluded. (2) To compare our results with those in the published literature.

MATERIALS AND METHODS: Seventy-three patients aged between 1 day and 15 years were admitted with clinical features of brain injury and underwent cranial CT. A retrospective review of their medical records and radiology was undertaken. Clinical and radiological data were collected.

RESULTS: On the basis of the combination of the presenting clinical history, progress, outcome, long term follow up and radiology, two groups of patients were identified. Forty-seven children had been the victims of non-accidental injury (NAI). No child in this group had any associated medical condition or any other medical cause for brain injury. All 47 children demonstrated hypoxic ischaemic encephalopathy and had CT signs of cerebral oedema and 'Reversal Sign'. Intracranial haemorrhage was a highly associated feature. Subdural blood was demonstrated in all 47, acute interhemispheric fissure subdural in 42, intracerebral blood in 16 and intraventricular blood in nine. The remaining 26 children were found to have an identifiable 'medical' cause for brain injury. Acute reversal was demonstrated in 21 of this group. Intracranial haemorrhage was uncommon, found in only five and all five had an underlying predisposition to bleeding. No patient in this group demonstrated subdural blood.

CONCLUSION: Non-accidental injury is strongly associated with the finding on CT of intracranial blood, particularly subdural haematoma and interhemispheric fissure bleeding in the presence of hypoxic ischaemic brain injury. The outlook is extremely poor whatever the underlying cause for hypoxic ischaemic brain injury. Rao, P. et al., (1999) *Clinical Radiology* 54, 495–501.

Key words: non-accidental injury, hypoxic ischaemic brain injury, Acute Reversal sign, subdural haematoma.

Non-accidental injury (NAI) is a major cause of childhood mortality and morbidity. The spectrum of injuries encompassed by the term 'child abuse' or 'non-accidental injury' has been well documented in the literature. Originally described by Tardieu in 1860 [1], much insight into the manifestations of this syndrome was contributed by John Caffey [2] when he described the association of multiple fractures in the long bones of infants suffering from chronic subdural haematoma. Until the 1930s, subdural haemorrhages were thought to be infective in origin. Since then the various skeletal, visceral, soft tissue and cerebral manifestations have been extensively reported.

NAI is the leading cause of serious head injury in young infants. During the last three decades, much attention has been focused on the craniocerebral manifestations of NAI. A wide variety of descriptive terms are used, some of which have aimed to describe the likely mechanism and pathogenesis of the injuries.

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In 1968, Ommaya *et al.* [3], in an experimental study on whiplash injury in monkeys, demonstrated that experimental cerebral concussion, as well as contusions and haemorrhages over the surface of the brain and upper cervical cord, can be produced by rotational displacement of the head on the neck in the absence of significant direct head impact. Guthkelch 1971 [4] described the mechanism of production of subdural haematoma during shaking in Battered Child Syndrome (BCS) as a tearing of the bridging veins in the subdural space by rotational forces. In 1972, Caffey [5] stated that shaking was a major cause of non-accidental intracranial morbidity in infants and coined the term 'whiplash – shaken baby syndrome' to describe a constellation of clinical findings in infants which consisted of subdural and/or subarachnoid haemorrhage, retinal haemorrhage, massive cerebral oedema in the absence of external signs of cranial trauma, fractured ribs and metaphyseal injury.

The initial brain injury in head trauma is known as the primary injury. This may be further complicated by cerebral oedema, vasospasm of the cerebral arteries with a fall in cerebral blood flow, shock, fits or compression by blood

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clots, all of which may contribute to the secondary injury, hypoxic ischaemic damage. Han *et al.* 1990 [6], reported a distinctive computed tomography (CT) appearance which they termed 'The Reversal Sign' in a subgroup of children with hypoxic-ischaemic cerebral injury. The CT features of the reversal sign are diffusely decreased density of the cerebral cortical grey and white matter with a decreased or lost grey-white matter interface or reversal of the grey-white matter densities and relative increased density of the thalamus, brain-stem and cerebellum. This may result from a variety of brain insults which ultimately lead to hypoxic-ischaemic encephalopathy, such as accidental and non-accidental trauma, birth asphyxia, drowning, status epilepticus or prolonged fits from any cause, status asthmaticus, cardiac arrest, encephalitis and meningitis. They indicated that the reversal sign carried a poor prognosis with irreversible brain damage and was highly associated with child abuse.

Previous reports in the literature suggest that acute interhemispheric fissure subdural haemorrhages may be specific for NAI [7]. The presence of both hypoxic ischaemic encephalopathy with interhemispheric fissure subdural in our experience is highly specific for NAI.

The objective of this study was to retrospectively review and compare the clinical features, aetiology, events precipitating admission, outcome and skeletal radiology with the intracranial appearances on CT in patients admitted with head injury to determine whether there were any significant differences in the CT appearances depending on the underlying cause.

METHODS

A retrospective analysis of the medical records and radiology of 73 patients who demonstrated acute reversal on cranial CT performed at Alder Hey Children's Hospital over a 10-year period from April 1987 to April 1997 was undertaken. The patients were identified from CT records but does not necessarily include all the patients with this CT sign in this period. The study population consisted of 43 boys aged between 14 days and 15 years (mean age 12 months), and 30 girls aged between 1 day and 10 years 3 months (mean age 18 months). The group consisted of children with primary presentation to this hospital and also those whose CT examination had been referred for a second opinion or for medicolegal purposes. Data was collected on underlying medical conditions, clinical presentation, progress, outcome and presence of any other injuries including soft tissue and skeletal injuries and other investigations performed. Clinical information on some of the patients was incomplete as full medical records were not always available or the information was obtained from the referral letter.

All patients primarily presenting to this hospital underwent an initial CT of the brain. Follow-up CT was performed as clinically indicated. All CT examinations, both initial and follow-up, both from this hospital and outside referral, were analysed on hard copy and assessed for the following:

- (i) presence and extent of oedema and acute reversal;
- (ii) presence and type of infarction;
- (iii) late changes of cystic encephalomalacia, hydrocephalus, cerebral atrophy, venous infarction and leptomeningeal cysts;

- (iv) presence or absence of intracranial haemorrhage including site, size and appearance;
- (v) presence of subdural and falx haemorrhage;
- (vi) the age of subdural at presentation;
- (vii) whether the subdural was unilateral or bilateral or of differing ages;
- (viii) the change in size, number and appearance of lesions between CT examinations.

Falx haemorrhage, or acute interhemispheric fissure subdural, was defined as a bright and irregularly thickened interhemispheric fissure.

The study was performed on unenhanced CT examinations. Contrast enhanced CT was obtained in 14 patients. The reasons for performing them were not always clear, some having been performed at other referring hospitals.

RESULTS

On the basis of the results of the clinical findings, laboratory investigations and radiology the patient population was divided into two groups. The first was termed the 'medical group' and consisted of 26 patients all of whom had an identifiable medical cause leading to the acute cerebral presentation. Eleven patients had sepsis. Three patients were identified as sudden infant death syndrome/near miss cot deaths. One child was admitted post smoke inhalation and two handicapped children were admitted after a seizure. Nine children suffered cardiac arrests: five were post-surgical, two post-asthmatic attack, one following electrocution and one was after an epileptic fit. The skeletal radiology available for review in this group was normal. No child had a skull fracture. Clinical examination did not reveal any soft tissue injury such as a rash, bruising, retinal haemorrhages or scalp haematoma.

The second group consisted of the remaining 47 children who had no identifiable medical reason for admission. Analysis of the skeletal radiology in these patients demonstrated fractures in 30 of the 47 children (Table 1). Skull fractures were present in 16, usually single and most commonly in the parietal region. Rib fractures were detected in 14 patients and in the majority were multiple. Long bone fractures were present in 19 patients. Equal numbers had diaphyseal and metaphyseal fractures. When present metaphyseal fractures were multiple in 75% of those in whom they were detected. In this group of 47, prior to the admitting episode, 10 children had previously presented unwell either to their general practitioner or to the Accident and Emergency department and sent home without further investigations or follow-up. In eight this occurred within 1 month of the final admitting episode and in the remaining two, within the previous 4 months. Of the 10, seven survived and three died.

Information from the case sheets on associated soft tissue injuries on the children in this group primarily presenting to Alder Hey is complete but data is incomplete from those children initially referred to other hospitals. However, when recorded, retinal haemorrhages were stated to be present in 21 of the 47 (12 survived and seven died) and skin bruises in 15 (eight survived and seven died). A petechial rash was seen in six and necklace calcification in one [8]. Scalp haematoma was detected in eight – on CT six were of high density and fresh and

Table 1 – Fractures : NAI group

Area	Total Number	Single	Multiple
Skull	16		
Parietal		9	3
Temporal		3	
Occipital		1	
Ribs	14	2	12
Long Bones	19		
Metaphyseal	8	2	6
Diaphyseal	8	3	4
Others	3	–	3

two were of low density and old. Eighteen children had a tense fontanelle at presentation. Of these, 13 survived and four died.

The diagnosis of NAI in this group was made based on the combination of clinical and radiological findings and the exclusion of identifiable cause for the hypoxic ischemic injury and bleeding.

CT Findings

All patients had an initial diagnostic CT. Follow-up CT examinations were performed in a further 12 of the medical group and in a further 37 of the NAI group. Most CT examinations were performed on or within 24 h of admission (Table 2). The patient in the NAI group who had a diagnostic CT examination 6 days after admission was initially admitted with suspicious soft tissue injury and had a provisional diagnosis of NAI following confirmation of multiple fractures on skeletal survey. The patient was initially neurologically stable but became irritable, though remaining stable, and CT was performed to complete the radiological imaging.

Patients were categorized into three groups on the basis of the CT features (Table 3):

Table 2 – Time of performing first CT examination relative to admission

Time from admission	Non-NAI	NAI
< 24 hours	19	42
1–2 days	1	4
3–6 days	3	1
7–10 days	1	0
11–14 days	2	0

Table 3 – Acute reversal

	Non-NAI n = 26		NAI n = 47	
	Number	% of Total	Number	% of Total
Oedema	11	42	21	45
Early acute reversal	7	27	5	11
Acute reversal	21	81	47	100

- (1) oedema with preservation of grey-white matter differentiation;
- (2) early acute reversal;
- (3) full acute reversal.

Oedema was defined as a swollen brain. Oedema was present in 11 of the medical group, seven of whom survived and four died. In 10 of the 11 it was seen on the first CT and in the remaining one it appeared on the second. In the NAI group, oedema was present in 21 patients, 15 survived and six died. In 15 it was seen on the initial CT and in the remaining six on the second CT examination. Nineteen patients who demonstrated oedema went on to develop full acute reversal changes.

Early acute reversal was defined as loss of grey-white matter differentiation (Fig. 1). Patients with early acute reversal also had oedema but, in addition, a definite loss of grey-white matter differentiation was evident. It was seen in seven of the medical patients and all demonstrated it on the initial CT. Six of the seven survived. Early change was seen in five NAI patients, all on the initial CT. All patients in the NAI group developed full acute reversal changes.

Established acute reversal was defined as a featureless brain with increased density of the thalamus and cerebellum (Fig. 2). It was seen in 21 of the medical group, nine survived and 12 died. Eighteen of the 21 demonstrated full acute reversal on the initial CT. Acute reversal was

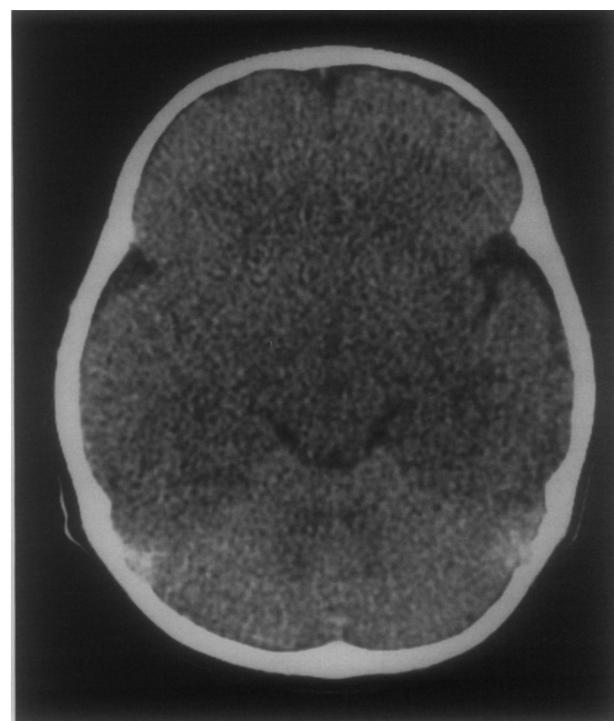


Fig. 1 – Axial non-enhanced CT through the brain in an abused child demonstrating an oedematous brain with generalized swelling and compression of the ventricles and sulci and a loss of grey-white matter differentiation.

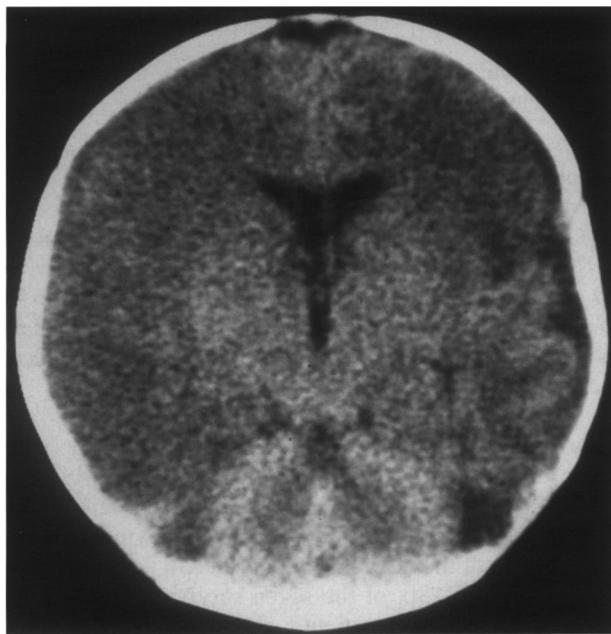


Fig. 2 – Axial non-enhanced CT through the brain of an abused child above the level of the fourth ventricle demonstrating the Acute Reversal Sign. There is diffusely decreased density of the grey and white matter with loss of grey–white matter differentiation and a relative increase in density of the cerebellum.

seen in all 47 of the NAI group, 34 of whom survived and 13 died.

Intracranial haemorrhage

Intracranial haemorrhage was an uncommon finding in the medical group, demonstrated in a total of only five out of 26 (Table 4). In all five there was an underlying predisposition to bleed. Three patients developed a disseminated intravascular coagulation following meningitis or meningococcal septicaemia. In one the blood was intraventricular, in one intracerebral and in the third small petechial haemorrhages occurred. One patient on warfarin for congenital heart disease bled spontaneously and developed an intracerebral haematoma in the temporoparietal region. The last patient developed multi-organ failure and clotting abnormalities post cardiac arrest. No

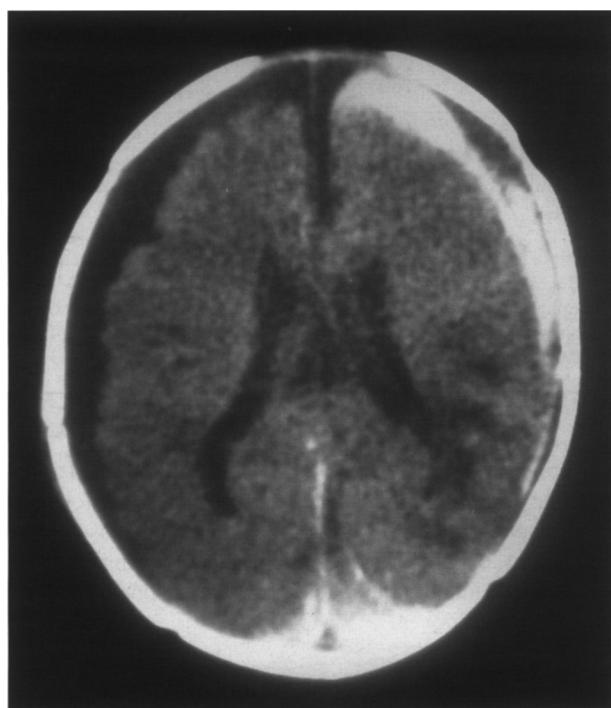


Fig. 3 – Axial non-enhanced CT through the brain in a child who suffered non-accidental injury demonstrating acute on chronic subdural haematoma on the right with chronic subdural on the left. The acute haemorrhage is seen as high density against the low density chronic blood.

patient in this group had subdural haemorrhage or acute interhemispheric fissure subdural haemorrhage (falx haemorrhage).

Intracranial haemorrhage was a highly associated feature in NAI patients (Table 4). Intracerebral haemorrhage was evident in 16 patients, 12 on the first CT and four on the second CT. Intraventricular haemorrhages were seen in nine patients. In six patients, the blood was evident on the initial CT. Two patients had petechial bleeds and one contusion.

Subdural haematoma was demonstrated in all 47 patients in this group (100%). Sixteen (34%) of the 47 patients had subdurals of different ages (Fig. 3). In 11 this was evident on the first CT. In the remaining five, due to early gross oedema, on the presenting CT only a small rim subdural was initially seen. As the swelling and oedema subsided subdurals of differing ages later became apparent. Thirteen patients (28%) had a unilateral subdural on presentation and in 24 (51%) the subdurals were bilateral. In 10 patients the subdural was not initially apparent only becoming so as the oedema resolved. Of these 10 the subdural was unilateral in three patients and bilateral in seven (Table 4).

Forty-two patients (89%) demonstrated acute interhemispheric fissure subdural blood. Three patients had acute interhemispheric subdural associated with a chronic/older subdural in another site on presentation. Seven NAI patients demonstrated acute reversal and subdural blood with falx haemorrhage as the main manifestation of abuse (Fig. 4).

Infarction

Infarction was demonstrated in 28 (59.6%) NAI patients and in 12 (46.1%) medical patients. Infarction was classified as

Table 4 – Intracranial haemorrhage

Site of haemorrhage	Non-NAI		NAI	
	Number	% of Total	Number	% of Total
Intraventricular	1	3.8	9	19.0
Contusion	0	0	1	2.1
Petechial	1	3.8	2	4.2
Laceration / Shearing	0	0	0	0
Intracerebral	2	7.6	16	34.0
Other (unspecified)	0	0	1	2.1
Subdural (any age or type)	0	0	47	100.0
Interhemispheric fissure subdural	0	0	42	89.0

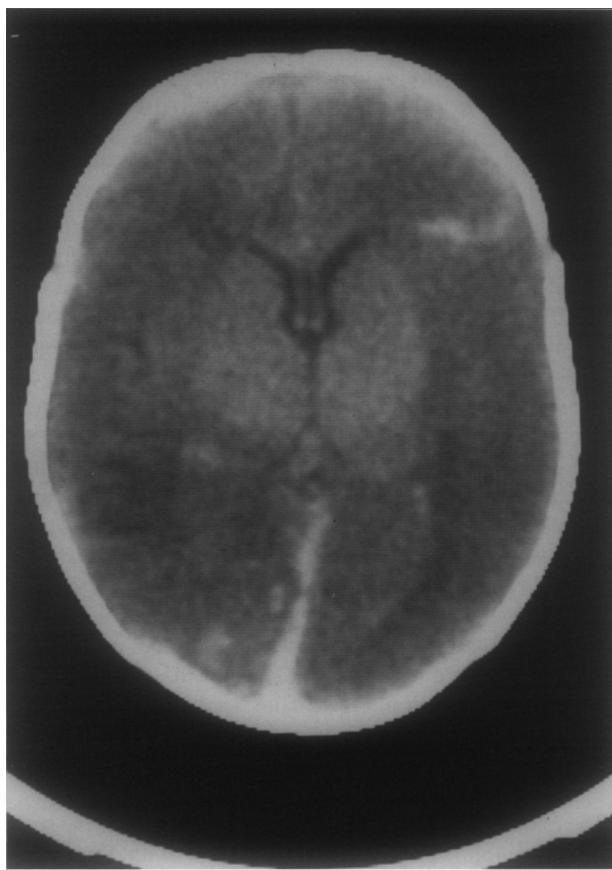


Fig. 4 – Axial non-enhanced CT through the brain of a shaken baby in non-accidental injury demonstrating both the Acute Reversal Sign of hypoxic-ischaemic encephalopathy and acute interhemispheric fissure subdural posteriorly along the falx. There are also several haemorrhagic lesions in the brain.

focal, generalized or haemorrhagic. Some patients had more than one type of infarction. In the 11 medical patients all but one had infarction at presentation. In the 28 NAI patients five patients demonstrated it on their presentation CT.

Focal infarction occurred more frequently than either generalized or haemorrhagic infarction. In the medical group single or multiple foci of infarction were equal in number. Most focal infarcts were in the cerebral hemispheres but three patients had internal capsule and basal ganglia infarcts. In the NAI group, 20 patients had focal infarction and multiple focal bilateral infarction was common. A medical patient developed haemorrhagic infarction evident on the presentation CT. Five NAI patients had haemorrhagic infarction. The location was varied. In one it was present on the initial CT; the remaining four developed it on later CT examinations.

A feature that was observed, not uncommonly in the medical group but not seen in the NAI group was frontal periventricular low density. This feature has been well documented to occur in association with ischaemic changes in the elderly.

Late changes

A proportion of patients in each group had one or more follow-up CT examinations and in these patients long-term consequences could be assessed (Table 5).

Table 5 – Outcome

Outcome	Non-NAI Number	Non-NAI % of survivors	NAI Number	NAI % of survivors
Deaths	13		13	
Survivors	13		34	
mild impairment	4	31	7	20.5
severe impairment	9	69	25	73.5
No information	–	–	2	6

Cerebral atrophy, seen in 15 NAI and four medical patients was much more often generalized than focal. Hydrocephalus developed in five of the medical group and in 11 of the NAI group. A small proportion of patients in each group developed an appearance known as 'bright gyri' or liquefaction, which is due to venous infarction. Progression to cystic encephalomalacia was observed in seven NAI patients and one medical patient. No patient developed leptomeningeal cysts.

Outcome

In each group, the number of deaths and the number of survivors was calculated. Survivors were categorized according to the degree of neurological impairment they suffered (Table 5). Mild to moderate neurological impairment encompassed such features as mild developmental delay, swallowing difficulties and visual problems such as ptosis. Severe impairment described patients who were severely mentally and/or physically retarded with considerably reduced quality of life. In the medical group, 13 patients died and 13 survived. Of the survivors, four showed a mild-moderate degree of neurological impairment, and nine were severely impaired. In the NAI group, 13 patients died and 34 survived. Apart from two survivors on whom clinical follow-up is lacking, the remaining all show some degree of neurological impairment; seven patients show a mild-moderate degree and 25 are severely impaired.

DISCUSSION

All the patients in our two study populations had severe intracranial brain insult at presentation. When a child is admitted obtunded in a state of shock, the cause is often unknown. The immediate concern is to resuscitate the child. During resuscitation the cause may become apparent as the clinical history becomes clearer or examination reveals a meningococcal rash or, in NAI, other bruising. A diagnosis of NAI is relatively straightforward if there are the typical soft tissue or skeletal injuries. When these features are absent the diagnosis is difficult and the cause of a child's collapse is not immediately apparent.

In some abused children, brain injury due to hypoxic-ischaemic encephalopathy arising from the shake is the only manifestation of physical abuse and it can occur in the absence of external markers of injury [5,9]. This occurred in seven children in this study. Less severe brain injury in abuse may

lead to the child presenting repeatedly to the general practitioner or Accident and Emergency Department with vague non-specific symptoms, such as feeding difficulties, irritability and vomiting. These symptoms are non-specific and unless a high index of suspicion is exercised, the diagnosis in these children may be missed. They are sent home, only to return later either dead on admission or with more severe injury leading to death. In our group of NAI patients, 10 had previously been seen in casualty and sent home and allowing for incomplete clinical data, may in fact have occurred in even more. Eight of the 10 were found to have either fractures and/or soft tissue injuries on the episode which eventually resulted in admission. Seven of the 10 survived and three died.

The mean age of presentation for patients in the medical group was 32.2 months but for the NAI group was significantly lower at 5.3 months. This age range is consistent with previous studies in which abuse was found to be most commonly associated with children under 12 months of age and often under 6 months [5,9]. At this age the young infant has a large head relative to the body and weak neck muscles resulting in poor support and control of the head and neck. The baby's brain is small in relation to the size of the cranium and the meninges are loose. The resulting whiplash movement causes the head to rock to and fro and rotate in different directions. Shearing forces are generated within the skull and brain which are important in producing the intracranial injury.

The CT manifestations of hypoxic ischaemic injury have been well documented in the past and, in particular, there has been much literature written on hypoxic ischaemic encephalopathy in premature neonates and long-term findings [10]. Han *et al.* [6] introduced the term 'Reversal Sign' to describe the appearance of the brain after hypoxic ischaemic insult in which, in children, there is diffusely decreased attenuation of the cerebral cortex with relative presentation of density in the thalamus, brainstem and cerebellum and loss of grey-white matter differentiation. All the patients in our two study groups demonstrated the reversal sign of varying severity or degree. The overall outlook was extremely poor for both groups.

A major and significant difference between the two groups was the very high percentage of patients in the NAI group who demonstrated intracranial blood (100%). Once other underlying reasons for intracranial bleeding had been excluded, for example, disseminated intravascular coagulation, anticoagulant therapy or haemorrhagic infection, this feature was unique to the NAI patients.

Three further features that were highly associated with the NAI patients and which deserve particular attention are, firstly, the high proportion of bilateral as opposed to unilateral subdurals in this group of patients. This is in contrast to accidental injury where the majority of subdurals are unilateral and occur under the injury site. The acceleration-deceleration forces generated during shaking in abuse affect both sides of the brain which results in bilateral subdurals [4,5,11,12,13].

Secondly, though the external signs of head injury such as fractures and scalp haematoma were present in 16 children with NAI, 31 children had brain injury with subdural collections, without external markers of head injury. It is this absence of external markers which leads to confusion and delays in the diagnosis. No patient in the medical group had either a skull fracture or scalp haematoma.

The third feature is the high percentage of patients having acute interhemispheric fissure subdural, another feature which appears to be highly associated with the presence of NAI. Zimmerman *et al.* [14], found this feature in 61% of their study group of patients. In this study it was present in 89%. On CT this is recognized as increased density and thickening of the falx which may also have irregularity of the posterior parieto-occipital portion of the interhemispheric fissure. Decrease in density of the falx between CT examinations will confirm that there is subdural blood. There is often accompanying cerebral parenchymal injury. Cerebral swelling may mask the presence of a small rim subdural under the skull bones. Magnetic resonance imaging (MRI) in children with head injury in child abuse is valuable in patients in whom the clinical symptoms are disproportionate to the CT findings, in identifying small subdurals especially if they are orientated transversely, in identifying subdurals of different ages, in distinguishing a chronic subdural associated with ventricular dilatation from cerebral atrophy and in the detection of subtle intracranial shearing injuries. In the acute setting however, CT remains used in far greater frequency because of its speed and accessibility and its ability to identify acute subarachnoid blood and identify surgically treatable lesions [14]. Thus, at the present time, in the emergency situation, CT remains superior [14].

The vast majority of our NAI patients (43 of 47 or 91%) were under 1 year of age and 27 (57%) were between 1 and 4 months of age.

The explanation offered by the carer as a cause for the severe intracranial injuries is often inconsistent with the injuries sustained. All cases must be reviewed in the light of the clinical history and a judgement made as to the compatibility of the injuries and history. Major injuries are sustained by major trauma. In the presence of extensive skull or brain injury a history of a minor injury or short fall is highly questionable [15].

Our findings are in agreement with those of Han *et al.* [6] in that the reversal sign of severe anoxic ischaemic brain injury carries a poor prognosis. When found in association with subdural haematoma, especially interhemispheric fissure subdural, it is highly associated with child abuse. Once a coagulopathy has been excluded, the demonstration of intracranial blood in the presence of the acute reversal sign should be considered an indicator of non-accidental trauma.

In the absence of a history of significant head injury subdural haematomas are highly associated with NAI. In this study population they occurred exclusively in the NAI group. Features which are further suspicious of NAI as the cause are the presence of acute interhemispheric fissure subdural haemorrhage, bilateral subdurals and subdural collections of different ages. In the initial stages the investigation of choice is CT. Intravenous enhancement does not appear to contribute to the CT diagnosis. MRI should be performed when the child is stable.

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