Characteristic Differences in Neuroimaging and Physical Findings Between Non-Accidental and Accidental Traumatic Brain Injury in Young Children. A Local Experience in General Hospital of Kuala Lumpur

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SUMMARY

Objective: The objective of this study is to identify the characteristic neuroimaging (namely brain CT) as well as physical findings found in young children with non-accidental traumatic brain injury (TBI) and to compare them with accident cases of the similar age group, in order to study the specific features of the former group more precisely. Materials and methods: A cross sectional study was done involving 92 children aged 3 years old and below who were admitted to the Kuala Lumpur Hospital with diagnosis of moderate to severe traumatic brain injury from period of June 2007 to September 2009. These children were categorized into non-accidental and accidental TBI and their physical examination data, brain computed tomography and skeletal surveys were done within one week from the date of admission were compared. Results: There was a male predominance in both non-accidental and accidental TBI groups with male-to-female ratio of 2:1 and 3:1 respectively. The majority of the non-accidental TBI cases presented with no definite history of trauma (52.2%) while most of the accidental TBI cases were caused by motor vehicle accidents (69.9%). Subdural haematomas appeared to be significantly the most common brain haematomas among the non-accidental TBI as compared to the accidental group while extradural haematomas were only present in the accidental TBI group. Cerebral edema was also significantly more common in the non-accidental group. Signs of pre-existing brain injury, including cerebral atrophy and subdural hygroma/effusion were present in 23.9% and 19.6% respectively among children with non-accidental TBI and in none of the children with accidental TBI. None of the children in the non-accidental group diagnosed to have shear injury while 6 (13.0%) of the children in the accidental group was diagnosed with diffuse axonal injury. In our series, retinal haemorrhage was significantly more common in the non-accidental TBI group (93.5%) as opposed to only 4(8.7%) children noted to have retinal haemorrhage in the accidental group. Seizures also occurred significantly more often in children with non-accidental TBI. Depressed skull fractures were only found in the accidental TBI group (19.6%), while other types of skull fractures occur more or less similar in both groups. Bodily fractures were also more predominant among the accidental group of TBI. Bodily lacerations/abrasions were only found in the accidental group while findings of bodily bruises were quite equal in both groups.

KEY WORDS:
Traumatic brain injury, Non-accidental head injury (NAI), Young children

INTRODUCTION

Head injuries remain the leading cause of mortality & morbidity in both adult & paediatric population worldwide. According to Division of Injury Control, CDC, in United States, TBI accounts for about 40% of fatalities in children from 1 to 4 years of age and about 70% of fatalities in children from 5 to 19 years of age [1]. More importantly, with changing in the socioeconomic background and the increase in number of nurseries as well as hired babysitters, non-accidental injury (NAI) has now become a recognizable cause of serious brain damage especially among children less than 2 years of age. In Krauss and Hemyari[2] epidemiologic study of paediatric TBI, the reported rates of non-accidental TBI among infants and preschoolers with TBI ranging from 4% to 24%, while Duhaime *et al*[3] noted that 24% of consecutively admitted children 0 to 24 months of age with head trauma were presumed to have non-accidental injuries. Epidemiologic data of non-accidental TBI in Malaysia is yet to be published, but unofficial information obtained from the General Hospital of Kuala Lumpur (GHKL) SCAN (Suspected Child abuse and Neglect) team estimates 30 to 40 cases per year. NAI may be misdiagnosed in its most subtle form and under diagnosed in its most serious form[4].

The aim of this study is to determine the characteristic patterns of radiological findings by means of conventional neuroimaging (CT scan and skeletal survey) as well as physical and neurological examination findings noted in children with suspected non-accidental TBI as opposed to the accidental group, which may be used as an adjunct parameters to the thorough history taking in differentiating these two groups more precisely in the future.

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MATERIALS AND METHODS

Research procedure :

This was a cross sectional study whereby a total of 92 children aged three years and below who suffered from moderate to severe traumatic brain injury of any cause (e.g: accidental or non-accidental) and presented to the General Hospital of Kuala Lumpur from a period of June 2007 to September 2009 were enrolled. A total of 51 admissions were identified for the non-accidental group but only 46 patients were selected during the study recruitment period. The other five patients did not fit the criteria (1 had previous history of preterm delivery with perinatal brain hypoxia, 2 patients had history of congenital hydrocephalus and the other 2 were excluded by the SCAN team following repeat history taking and clinical examination). For the accidental group, a total of 102 admissions were noted over the recruitment period who met the study criteria. However, since the number of patients for this group was earlier anticipated to far exceed the number of patients in the non-accidental group, patients were selected by including every alternate admission till the number of subjects reached 46 after which recruitment was stopped. All children were examined by either a qualified paediatrician / paediatric neurologist / neurosurgeon for verification of the clinical histories & physical signs within 72 hours of injury. For the non-accidental TBI group, fundoscopic examination of both eyes were examined by a qualified ophthalmologist in order not to miss subtle but significant retinal haemorrhages. For the accidental group, funduscopic examinations were only done by the attending paediatrician or neurosurgeon since the SCAN team (which includes the ophthalmologist) was not involved in the management of this group of patients. All neuroimaging films particularly the brain CT were reviewed by either a consultant neurosurgeon and/or qualified radiologists. Only scans that were done on the day / within 1 week of admission to GHKL were included in the study. The above data was analysed separately before the differences in the distribution of findings between the non-accidental and accidental groups assessed using Chi square or Fisher exact test statistical analyses wherever applicable (SPSS version16.0.1)

Inclusion criteria

For both groups the children needed to have:

1. moderate to severe TBI, with no known previous history of TBI, no history of neurologic disorder, no metabolic disorder or any congenital brain anomalies. Severity of TBI was based on i) The standard modified Glasgow Coma Scale (GCS) for children; (GCS of < 12) ii) Positive CT brain findings of extraaxial haematoma, intraparenchymal haematoma or cerebral edema regardless of GCS score.
2. been born at least after 37 weeks of gestation with no previous history of perinatal / postnatal insult

For children with suspected non-accidental TBI, other criteria included:-

i) Inconsistency in history, clinical findings & neuroimaging findings
ii) Head injury in which there had been confession by the perpetrator
iii) Cases in which non-accidental TBI (NAI) had been established by the committee/SCAN team GHKL or as a result of conviction in the criminal court

Exclusion criteria

Children aged above three years or those three years and below with history of :

i) Ex-premature delivery
ii) Prenatal / postnatal insult
iii) Neurologic or metabolic disorders, or any congenital brain anomalies
iv) Those cases excluded as non-accidental TBI case by the SCAN team

Sample size calculation and sampling methods

Calculated N value was done based on the statistically significant parameters (*) observed in the previous study by Linda Ewing- Cobbs *et al* Am J Paed. 1998.

Formula used : $N = \dfrac{P0\,(1 - P0) + P1(1 - P1)}{(P0 - P1)^2} \times (Z\text{æ} + Z\text{ß})^2 = 62$

P0 = Control (Noninflicted)
P1 = Cases (Inflicted)
Zæ(0.05) = 1.96 } constant
Zß = 0.84 } "

- Level of study significance (α) = 0.05
- Power of study (1- β) = 0.8 since throughout the limited study period, we only manage to recruit 46 patients out of the true sample size of 62 patients for each group:-

Statistical analysis

Data from the admission notes and films were studied and collected using a standardized questionnaire. Analysis of the data was done using SPSS version 16.0.1 with the guide of our statisticians. Most of the data were categorical except for age. Descriptive analysis was done for all the variables data. Multivariate analysis was done using Chi-square test or Fisher exact test wherever applicable to identify significant differences in the variables distribution among the two studied groups.

RESULTS

Table I: Summary of variables observed between the two groups of TBI

A) Neuroimaging Findings From Acute CT Brain

	Group	
	Non-Accidental TBI (n = 46)	Accidental TBI (n = 46)
Extraaxial collection		
Haematoma		
Subdural	39	21
Extradural	0	14
Subarachnoid	28	12
Hygroma/effusion		
Subdural	9	0
Parenchymal involvement		
Intracerebral haematoma	5	9
Cerebral edema	22	13
Shear injury	0	6
Atrophy	11	0
Skull fracture		
Linear	8	15
Comminuted	4	6
Diastatic	0	4
Depressed	0	9
Basilar	2	3

B) Bodily Injuries in Non-accidental and Accidental TBI Groups

	Group	
	Non-accidental TBI (n = 46)	Accidental TBI (n = 46)
Retinal haemorrhage		
Bilateral	42	4
Unilateral	2	0
Bodily fractures	1	11
Bodily bruises	11	18
Bodily laceration/abrasions	0	10

C) Neurological Findings in Non-Accidental and Accidental TBI Groups

	Group	
	Non-Accidental TBI (n = 46)	Accidental TBI (n = 46)
Seizures		
Present	36	6
Absent	10	40
Hemiparesis		
Present	12	18
Absent	34	28

Statistical analysis

Table II: Differences in distribution of pattern of neuroimaging and physical findings between non-accidental and accidental groups of TBI

	Traumatic brain injury		X^2stat (df)	P value
	Non-accidental n(%)	Accidental n(%)		
Subdural haematoma				
Yes	39 (84.8)	21(45.7)	15.525 (1)	<0.001[a]
No	7 (15.2)	25 (54.3)		
Extradural haematoma				
Yes	0 (0)	14 (30.4)	16.513 (1)	<0.001[a]
No	46 (100)	32 (69.6)		
Subarachnoid haemorrhage				
Yes	28 (60.9)	12 (26.1)	1.658 (1)	0.198[a]
No	18 (39.1)	34 (73.9)		
Subdural effusion/hygroma				
Yes	9 (19.6)	0 (0)		0.003 [b]
No	37 (80.4)	46 (100)		
Intracerebral haemorrhage				
Yes	5 (10.9)	9 (19.6)	1.348(1)	0.246[a]
No	41 (89.1)	37 (80.4)		
Cerebral edema				
Yes	22 (47.8)	13 (28.3)	3.735(1)	0.053[a]
No	24 (52.2)	33 (71.7)		
Cerebral atrophy				
Yes	11 (23.9)	0 (0)	12.494(1)	<0.001[a]
No	35 (76.1)	46 (100)		
Shear injury				
Yes	0 (0)	6 (13.0)		0.026[b]
No	46 (100)	40 (87.0)		
Linear skull fracture				
Yes	8 (17.4)	15 (32.6)	2.841(1)	0.092[a]
No	38 (82.6)	31 (67.4)		
Comminuted skull fracture				
Yes	4 (8.7)	6 (13.0)	0.449(1)	0.503[a]
No	42 (91.3)	40 (87.0)		
Diastatic skull fracture				
Yes	0 (0)	4 (8.7)		0.117[b]
No	46 (100)	42 (91.3)		
Depressed skull fracture				
Yes	0 (0)	9 (19.6)		0.003[b]
No	46 (100)	37 (80.4)		
Basal skull fracture				
Yes	2 (4.3)	3 (6.5)		0.242[b]
No	44 (95.7)	43 (93.5)		

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	Traumatic brain injury		X^2stat (df)	P value
	Non-accidental n(%)	Accidental n(%)		
Retinal haemorrhage				
Yes	43 (93.5)	4 (8.7)	66.162(1)	<0.001[a]
No	3 (6.5)	42 (91.3)		
Bodily fractures				
Yes	1 (2.2)	11 (23.9)	9.583(1)	0.002[a]
No	45 (97.8)	35 (76.1)		
Bodily bruises				
Yes	11 (23.9)	18 (39.1)	2.467(1)	0.116[a]
No	35 (76.1	28 (60.9)		
Laceration/abrasion wound				
Yes	0 (0)	10 (21.7)	11.220(1)	<0.001[a]
No	46 (100)	36 (78.3)		
Seizures				
Yes	37 (80.4)	6 (13.0)	41.961(1)	<0.001[a]
No	9 (19.6)	40 (87.0)		
Hemiparesis				
Yes	15 (32.6)	11 (23.9)	0.858(1)	0.354[a]
No	31 (67.4)	35 (76.1)		

a Pearson χ_2 test applied.
b Fisher exact test applied.

DISCUSSION

In our study, the comparison between the two groups was done among children below three years of age for several reasons :- i) In most epidemiologic data worldwide, non-accidental TBI mainly affect children below 2 years of age [5] while for the accidental cases it is quite rare among children aged less than one year [6, 7]. Too wide an age difference between subjects may complicate data comparison, as body physiology of young children and older children is quite different particularly in response to trauma. ii) Previous data from our hospital has shown that most of the NAI or physical child abuse cases with moderate to severe traumatic brain injury occurred among children less than 2 years old in the form of shaken baby syndrome.

The mean age group for the non-accidental group of TBI was 10.5 months while for the accidental group was 19 months. The mean age of the former group was comparable with those of Linda Ewing *et al* [8]. However for the gender distribution, males were more commonly involved than females (2:1 ratio) in the non-accidental group as compared to the above author's group whereby the findings was reversed (male to female ratio was 1:5.6). For the ethnic group involved, non-accidental traumatic brain injury was found to be most common among Malays followed by others (no specific subgrouping of the ethnicity of 'others' was done in this particular study). The distribution among Chinese and Indians seems to be equal. However, these ethnicity distributions reflect not just the ethnic distribution of the country in general but also that of the paediatric admissions to HKL. Further study needs to be conducted to determine the association between the incidence of non-accidental TBI and ethnicity in terms of their socioeconomic background, cultural belief or practice etc.

As for the suspected cause of injuries, 52.2% of the non-accidental TBI presented with no definite history while the second most common cause was either a fall from a swing or being dropped by caregivers. The real cause in the former might be postulated as repeated shaking either in a swing or on the caregiver's lap since most of them will give the reason that they did not know that the act might lead to serious brain injury. This is a form of maltreatment that commonly happens as a result of frustration felt by the caregivers following repeated or unconsolable cry of the child. For the accidental group of TBI, motor vehicle accidents were the leading cause of serious brain injury in the children which accounts for 69.9%. This finding was also comparable with the adult population.

The occurrence of subdural haematoma was found to be more common in the non-accidental group of TBI as compared to the accidental group. This findings together with the findings of a lack of external signs of trauma (skull or skeletal fractures, soft tissue injuries) suggest that the pattern of inflicted brain injury in our series was actually caused by repeated shaking. This findings is comparable with those reported in UK, Hong Kong and Canada [9,10,11]. However, in Japan majority of the abused children with subdural haematoma had suffered direct blows to the face and head, resulting in external signs of trauma [12]. About one third of children in the non-accidental group with subdural haematoma had either subacute or chronic subdural haematoma while the remaining had acute subdural haematoma, whereas for the accidental group all of the patients had acute subdural haematoma. These findings together with the findings of preexisting brain injury such as subdural effusion/hygroma or cerebral atrophy which were exclusively found only in the non-accidental group of TBI suggest the different mechanism and pathophysiology of brain injury in both groups. The etiololgy of subdural haematoma in the former was most likely due to the repeated shaking (rotational or acceleration deceleration forces) while in the latter group mostly due to the severe direct impact to the head. Another possibility for the signs of preexisting brain injury could be previous assault and cumulative brain injury. As noted by Alexander R *et al* [13],

non-accidental injuries in young children are frequently preceded by other forms of maltreatment.

There did not seem to be any difference in the distribution of intracerebral haematoma and subarachnoid haemorrhage between the two groups which is comparable to what is reported in the international literature. Extradural haematoma in our series was exclusively found in the accidental group. This is not surprising as this type of haematoma can only occur as a result of direct impact or contact forces to the head which leads to skull fracture haematoma or haematoma from tearing of the meningeal vessels. On the other hand, most of the patients in the non-accidental group in our series are likely victims of the Shaken Baby Syndrome (SBS) rather than direct blow to the face or head by the perpetrator. However the fact that about 10 to 20% of our patients in the non-accidental group also had either linear or comminuted skull fractures provides some support for the "shaking-impact" mechanism of injury proposed by Bruce DA and Zimmerman [14].

Shear brain injury or more commonly known as diffuse axonal injury (DAI) was diagnosed in none of the children with non-accidental TBI in our study. DAI is a diagnosis of exclusion whereby patient who came with definite history of trauma has a low GCS at presentation but yet a 'normal' brain CT or just multiple small punctate haemorrhages. Magnetic resonance imaging or MRI is a more reliable modality to detect this type of axonal damage. Six (13%) of our patient in the accidental group was diagnosed to have shear brain injury and all of them presented with high impact motor vehicle accidents as the cause of the injury. Eventhough the above findings were not made with the benefit of MRI, it was actually comparable with the work of Geddes JF *et al*[15], which is a neuropathological study of 53 cases of children with NAI that concluded that diffuse axonal injury is an uncommon sequel of non-accidental head injury in children.

Retinal haemorrhage is the most constant finding among children diagnosed with non-accidental TBI worldwide. Most of the international studies quote an incidence of either unilateral or bilateral retinal haemorrhage to range between 70% to 80% [5, 8, 10, 11, 15]. The exact pathophysiology is still not well understood but some investigators observed that there was a significant association between subdural bleeding and the presence of retinal haemorrhages. In all cases with intraocular bleeding there were subdural haemorrhages [15]. This finding might imply severity of impact to the head (either by contact forces or acceleration/deceleration) as the cause of retinal haemorrhage. Some would also suggest repeated episodes of seizures (which is the common occurrence among children with NAI) as the cause of the retinal bleed. However, Tyagi *et al*[16] and Sandramouli *et al*[17] in their series of children presented with convulsions episodes (not related to NAI) and had their fundus examined by ophthalmologist within 48 hours of admission, found none of them had retinal haemorrhage. Others claim retinal haemorrhage can also be associated with cardiopulmonary resuscitation [18]. However in our opinion, the fact that none of our patient in the non-accidental TBI group required CPR at presentation, this seems to be less likely to cause retinal haemorrhage. In our series the incidence of retinal haemorrhage among non-accidental TBI was slightly higher (93.5%) compared to other authors as mentioned above. Out of 46 patients with accidental TBI, we found 4 (13%) to have retinal haemorrhage all of which were bilateral. This finding was slightly different from the Ewing-Cobbs *et al*[8] whereby none of the children with accidental traumatic brain injury in their series had retinal haemorrhage. However the work of Kivlin JD *et al*[19] gives some support to our finding in which out of 10 children less than 3 years of age who involved in a fatal motor vehicle crashes, 8 had retinal haemorrhages (from autopsy findings). Hence they made a conclusion that any form of severe accidental traumatic brain injury can also lead to development of retinal haemorrhage in young children.

The findings of skull fractures were almost equal across the two groups of TBI except for depressed skull fractures which was only found in the accidental group of TBI. Depressed skull fractures among young children especially those less than 1 year of age appear as 'ping-pong ball' type of fracture (referring to the continuous depression of the skull bone cortex due to the soft texture of the immature bone). This type of fracture is commonly seen following a direct impact or blow to the skull by hard object. The fact that none of the children in the non-accidental group had depressed skull fracture as opposed to 9(19.6%) children who had it in the accidental group, supports our theory that the mechanism of head injury among the non-accidental group in our series is due to the shaken baby syndrome. Bodily fractures (e.g: limbs, ribs, spine and pelvis) were also more common among the accidental TBI group, whom the majority as we know, are children involved in motor vehicle accidents. As for the non-accidental group, only one had multiple ribs fractures. It was claimed by the mother that the child was 'assaulted' by his own elder brother while playing. Laceration and abrasion wounds were also not seen in the non-accidental group but present in 10 children in the accidental group while bodily bruises were almost equally common in these two groups with no special pattern of bruises (e.g: bite or canning marks etc.) in the non-accidental group of children.

Seizures are far more commonly present among children in the non-accidental group (80.4%) in our study. This is also a common finding in most of the international studies with the frequency ranging from 40%-70% [5, 8, 10, 20]. In fact, this is included in the clinical triad together with subdural haematoma and retinal haemorrhage for the diagnosis of NAI. Seizures in children may be subtle or subclinical, hence vigilant clinical assessment is important to identify this sign as proper management of seizures may determine the future outcome of the children. Prolonged intractable seizures or status epilepticus is usually associated with high morbidity and mortality in these children [21]. The exact pathophysiology of frequent seizures in this group of patient is still not well understood. However it is well known that seizures following head trauma is most likely due to the irritation of the brain cortex by the blood degradation product such as haemosiderin as well as some tissue scarring all of which can only be found in a case of chronic or repeated brain injury as in the NAI cases. Another interesting point to note is that some of the children in the non-accidental group, particularly those below one year old, did present with prolonged intractable seizures or status epilepticus which later led to the

development of global cerebral infarction found on repeat CT (hypoxic-ischaemic injury). However, making a diagnosis of diffuse cerebral ischaemia/infarction in young children based on CT alone can sometimes be tricky as the appearance may mimic diffuse cerebral edema. The more accurate way to establish the diagnosis is via diffusion weighted image (DWI) MR. However some authors postulate that the hypoxic-ischaemic insult precede and cause the seizures to occur. This has come about from the observation of infants less than 4 months old with SBS who present with recurrent episodes of apnoea and tend to have hypoxic-ischaemic brain injury at initial admission. The pathophysiology for the apnoea is thought to be due to injury of the cervicomedullary junction as a result of repeated shaking since the ligaments at the craniocervial junction in the very young infants are still lax. In Geddes *et al* [15], a series of autopsies, 11 out of 53 infants of 2-3 months old with inflicted TBI showed axonal damage at corticospinal tracts of the craniocervical junction. However further details on the frequency and pathophysiology of apnoea among infants with non-accidental brain injury is beyond the scope of our study.

Finally hemiparesis was almost equally distributed among the two groups of TBI. In the non-accidental group of children hemiparesis was most likely due to the focal unilateral cerebral ischaemia/infarction as a result of chronic venous compression by the subdural haematoma or even secondary to repeated episodes of seizures. As for the accidental group of TBI, hemiparesis was commonly a result of either damage to the corticospinal tract following brain contusion or mass effect caused by the acute extradural/subdural haematomas which in the latter also frequently accompanied by hemispheric edema.

CONCLUSION

From our study, the characteristic features of the non-accidental TBI among young children in terms of neuroimaging (acute computed tomography) findings were subdural haematomas and signs of preexisting brain injury (cerebral atrophy and subdural effusion/hygroma) while retinal haemorrhages and seizures were the most common physical signs related to the non-accidental as compared to the accidental TBI. SBS appeared to be the most likely variant of the non-accidental TBI among children less than 3 years old presented to our hospital. Further study involving different centres might help to establish a form of standard scoring system or diagnostic criterias (as adjunct to the thorough history taking) in making the diagnosis of non-accidental traumatic brain injury or NAI more convenient and precise in the future.

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