Paediatric Forensic Medicine- A Review : Controversies Regarding The 'Shaken Baby Syndrome'

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Abstract

The controversy surrounding the so called "shaken baby syndrome" (SBS) has been around for a while now. This syndrome is characterized by intracranial and retinal haemorrhages and acute encephalopathy not explained by history. Other injuries to the neck, spinal cord, skeleton and soft tissues may also be seen. Infants are the most common victims of SBS.

INTRODUCTION

In 1946, Caffey was the first to offer a description of multiple long bone fractures associated with chronic subdural haematoma in infants where a history of long bone fractures and of the head was lacking [1]. There were no radiological or clinical conditions predisposing to pathological fractures. In the 1960's through his biomechanical research Ommaya and others reported that high speed rotational displacements of the head on the neck, without significant direct head impact, could produce cerebral concussion and haemorrhages over the surface of the brain and spinal cord in primates. In the 1970's, Guthkelch reported infants with subdural haemorrhage (SDH) ,without external marks of injury on the head, and extrapolating from Omamaya suggested that in some cases repeated acceleration/deceleration rather than direct impact was the cause of the haemorrhage (i.e the infant being shaken). This was supported by the high frequency of SDH in 'battered children'. In the 1970 Caffey further described 27 infants thought to have been shaken and sustained SDH and metaphyseal avulsions. He postulated that these were due to indirect acceleration/deceleration stresses on the periosteum and articular capsules, rather than direct impact [23].

BIOMECHANICS IN INFLICTED HEAD INJURY

The term "whiplash shaken-baby syndrome" was coined by Caffey to explain this constellation of infantile subdural and subarachnoid haemorrhages, traction-type metaphyseal fractures, and retinal haemorrhages and was based on evidence that angular (rotational) deceleration is associated with cerebral concussion and subdural haematoma.

Because the type but not the magnitude of deceleration was addressed in early reports of the syndrome, it was postulated that injuries could be inflicted unintentionally by caretakers through generally acceptable child care practices. Recent biomechanical studies of these injuries show that the magnitude of angular deceleration is 50 times as great when the head of an infant model held by the trunk forcefully strikes a surface as when shaking alone occurs, and it only reaches injury thresholds calculated for infants at the moment of impact. When the surface is soft, the force of the impact is widely dissipated and may not be associated with visible signs of surface trauma, even though the brain itself decelerates rapidly. It is the sudden angular deceleration experienced by the brain and cerebral vessels, not the specific contact forces applied to the surface of the head that result in the intracranial injury. It is thought that this angular force, is distinct from the forces generated in most cases of accidental trauma in infants.

The majority of abused infants have clinical, radiological, and autopsy evidence of blunt impact to the head [4]. Following a publication by Duhaime et al of children most of whom had retinal haemorrhages, subdural or subarachnoid haemorrhage and history of abuse, he conducted experiments on dolls and concluded that shaking alone did not generate sufficient force to cause the type of injuries seen [5]. He suggested that the impact must be a factor and suggested an alternative 'shaken-impact syndrome'. Since then the diagnoses of shaken baby syndrome has generated high profile cases and has generated a great deal of controversy both in the medical and legal fields. Some authors' view was that the use of the term

"shaking–impact syndrome" rather than "shaken-baby syndrome", reflect more accurately the mechanism responsible for these injuries [6] Whether shaking alone can cause the constellation of findings associated with the syndrome is still debated, but most investigators agree that trivial forces, such as those involving routine play, infant swings, or falls from a low height are insufficient to cause the syndrome. Instead, these injuries appear to result from major rotational forces, which clearly exceed those encountered in normal child-care activities.

TERMINOLOGY

Some people have argued that SBS perhaps can be more appropriately referred to as inflicted (or abusive) head trauma (IHT). There has been a tendency to move away from the terms "shaken baby syndrome" and "shaken infant syndrome". Although shaking may cause an acute ecepaholopathy, SDH and retinal haemorrhage, the diagnosis shaking as a mechanism of injury, or attributing any mechanism of injury to a particular child who presents with these clinical findings is not possible, because these are unwitnessed injuries that may be incurred by a whole variety of mechanisms solely or in combination. The brain may be injured by impact acceleration, impact deceleration, compression, penetration, rotational injury or rotation with impact. More and more authorities in the medico-legal field now believe that the use of IHT refocuses the analysis of the findings in any particular case with head injuries to the determination of whether they are inflicted or non-inflicted (i.e. accidental vs non-accidental). The use of IHT also has the advantage of minimizing the controversy during expert testimony of whether shaking alone (i.e., rotational acceleration and deceleration) is sufficient to produce the spectrum of findings associated with IHT or whether head impact is also necessary.

The names applied to the syndromes of inflicted head injury in infancy reflect the evolving and sometimes controversial understanding of the actions necessary to cause the types of injuries seen, such as shaking an infant held by the arms or trunk or forcefully striking an infant's head against a surface. Although there is considerable controversy, the available evidence suggests that it is the sudden deceleration associated with the forceful striking of the head against a surface that is responsible for most, if not all severe inflicted brain injuries. Because the histories given when infants with such injuries present for medical attention are often vague or unreliable, the events must be inferred from knowledge of the causative forces in witnessed cases of accidental trauma and experimental models of injury. Studies of the biomechanics of brain injury have established that forces applied to the head that result in a rotation of the brain about its centre of gravity cause diffuse brain injuries. It is this type of movement that is responsible for the diffuse axonal injury and subdural haematoma seen, (i.e. in cases of motor vehicle accidents that result in severe disability or death). In contrast, forces that result in a translation, or straight-line, movement of the centre of gravity are generally less injurious to the brain, with the effects largely determined by the specific focal contact forces [7]. The type and severity of the injury are determined both by the type of deceleration and by its magnitude. In infants and young children, household falls causing head injuries mainly involve low velocity translational forces and rotational (or angular) deceleration is thought to be uncommon.

GEDDES' HYPOTHESIS

The other controversies surrounding IHT centre on the specificity of retinal haemorrhages and whether there is a lucid interval immediately following fatal injury in cases of IHT. In IHT, the intracranial injuries typically consist of severe cerebral oedema, thin subdural and subarachnoid haemorrhages and axonal injury depending on duration of survival. Recent studies by Geddes suggested that cerebral oedema may be hypoxic-ischemic in origin and secondary to apnoea caused by axonal injury within the brainstem and upper cervical spinal cord that has been attributed to shearing forces [8910]. The controversy erupted when she suggested that it may not be necessary to shake an infant very violently to produce stretch injury to the neuroaxis . Another study by Geddes identified microscopic intradural haemorrhages in the dura taken from fetuses which died in utero and newborns who died in the perinatal period. It was proposed that all of the findings, including the subdural and retinal haemorrhages that are characteristic of IHT/SBS, could be a consequence of hypoxic injury [11]. This has led opponents to the Geddes theory to state that if gentle shaking were capable of causing fatal injury, such events would be an everyday occurrence [1213]. Other investigators however allege that domestic minor head trauma is common, but it is only very rarely associated with severe intracranial injury.

RETINAL HAEMORRHAGES

Retinal haemorrhage is one of the important elements in the diagnosis of IHT/SBS , especially when other causes of inflicted head trauma are not apparent from the history or clinical evaluation. However, the presence of retinal haemorrhages is not diagnostic of IHT, and their absence

does not exclude its diagnosis. Retinal haemorrhages have been observed in children in association with numerous pre-existing conditions, including meningitis, elevated intracranial pressure, vitamin K or C deficiency and anoxia. However, these are rare complications and when they do occur they are few and confined to the posterior pole of the retina. Superficial flame or splinter shaped retinal haemorrhages are said to follow normal birth but usually resolve within three weeks while deeper layer haemorrhages resolve in two to four weeks. Retinal haemorrhages in accidental head injuries are also said to be rare, but may occur after high velocity side impact road traffic accidents. In contrast retinal haemorrhages of non-accidental head injury (NAHI) have been observed to be severe and extensive throughout the retina and involve the sub-retinal, intra-retinal and pre-retinal layers and extend to the ora serata. The most favoured explanation for the retinal haemorrhage in shaking is that of vitreous traction. A study by Schoff et al did not identify vitreous haemorrhage in 53 of 55 children with intracranial haemorrhage not caused by abuse [14]. Retinal haemorrhages have been documented in children who were resuscitated after head injuries that were sustained during motor vehicle accidents or following accidental, non-inflicted household trauma. However, the retinal haemorrhages that have been described or illustrated in these conditions have been few in number, occasionally superficial, small in size, and do not extend to the ora serrata, even when the prothrombin and partial thromboplastin times and platelet counts are abnormal. In contrast to these conditions, retinal haemorrhages associated with inflicted head trauma are characteristically bilateral, involve the full thickness of the retina, and extend to the ora serrata . Several mechanisms including shearing forces, sustained elevated intracranial or elevated intrathoracic pressures, direct tracking of blood from the intracranial space, or direct impact trauma have been proposed for the development of retinal haemorrhages. There seems to be an increasing consensus among workers in the field that shearing forces may be the most important etiological factor. One study examined that orbital tissue injury is more common in SBS than accidental head trauma without orbital fracture [15]. In addition, optic nerve sheath and optic nerve intradural haemorrhage are also significantly more common in SBS. The researchers concluded that these findings are due to unique acceleration-deceleration forces in this type of abusive head injury.

TIMING OF THE INJURY

The other controversy has been, whether a lucid interval exists in cases of inflicted head trauma. If a lucid interval does not occur, or is very brief, it is thought then it is much easier to establish the timing of a lethal head injury and thus establish possible perpetrators. Neutral witnesses have reported the immediate loss of consciousness of infants being severely shaken. A literature review [16], provided no evidence of lucid intervals following inflicted head trauma leading to death. In contrast to inflicted head injuries that cause death, lucid intervals may follow accidental craniocerebral trauma that ultimately ends in the death of the child. Lucid intervals are commonly reported in childhood victims of lethal accidental head trauma following the formation of epidural haemorrhages. In another study of 18 cases of children older than 1 year of age who sustained lethal accidental head injuries showed that 67% experienced a lucid interval, however, in some cases, the interval was less than 1 hour [17]. It appears that the vast majority and the most reliable evidence to date indicates that infants who sustain lethal inflicted as opposed to accidental head trauma experience neurological deterioration and loss of consciousness very rapidly, if not immediately following, craniocerebral trauma. In a study by Biousse et al all patients with confirmed SBS showed an abnormal diffusion-weighted magnetic resonance imaging and suggested diffuse or posterior cerebral ischemia, in addition to subdural haematomas, in the pathogenesis of this disorder [18]. The mechanisms responsible for the cerebral injury remain debatable. Subdural haematoma, which was present in all cases, was thought to be likely caused by the shearing forces provoked by the acceleration-deceleration which disrupt the small bridging veins over the surface of the brain. Some authors states that cerebral hypoxia/ ischemia in SBS may be explained by numerous factors such as reactive vasospasm adjacent to hemorrhagic lesions, strangulation, cervicomedullary injuries, and persistent crying and apnea. These is some suggestion that cerebral ischemia in SBS is one of the mechanisms leading to irreversible neuronal damage in SBS.

Since the history is often unreliable in cases of the inflicted head trauma, information about the timing of the injury is extrapolated from data on accidental trauma. Acute subdural hematoma associated with severe neurologic compromise, brain swelling, or death occuring in the setting of a clear injury involving a major mechanical force is followed by the immediate or rapid onset of neurologic symptoms. In a series of 95 children who died from accidental head injuries, all but 1 had an immediate decrease in the level of consciousness [19]. This type of injury, generated by contact forces to the

skull and dura, is usually not associated with a serious primary brain injury and is rarely associated with child abuse. Other reports of delayed deterioration after paediatric head injury have primarily involved the onset of seizures, followed by recovery. On the basis of these data, it can be discerned that there is no evidence of a prolonged interval of lucidity between the injury and the onset of symptoms in children with acute subdural hematoma and brain swelling. A separate issue concerns the possibility of a subclinical injury that is later exacerbated by a relatively minor second mechanical trauma. Such rare events have been reported in older children and adults, usually in the setting of acute subarachnoid and subdural hemorrhage and brain swelling related to recurrent impact to the head involving well-documented concussive forces during sports activities [2021]. This pattern of injury, with a clear time line and rapid, well-described acute deterioration, stands in sharp contrast to the vague histories of previous episodes of trivial trauma that are sometimes suggested as possibly causative in the shaking–impact syndrome. There is no evidence that traumatic acute subdural hematoma, particularly that leading to death, occurs in otherwise healthy infants in an occult or subclinical manner.[2223]

SUBDURAL HEMATOMAS

Acute subdural hematoma in infants is distinct from that occurring in older children or adults because of differences in mechanism, injury thresholds, and the frequency with which the question of nonaccidental injury is encountered. In a study to analyze the clinical characteristics of acute subdural haematoma in infancy, Loh et al retrospectively reviewed 21 cases of infantile acute subdural haematoma and concluded that the most common cause of injury was shaken baby syndrome [24]. The most common clinical presentations were seizures, retinal haemorrhages, and consciousness disturbance. Eight patients with large subdural haematomas underwent craniotomy and evacuation of the blood clot. None of these patients developed chronic subdural haematoma. Thirteen patients with smaller subdural haematomas were treated conservatively. Among these patients, 11 developed chronic subdural haematomas after the acute subdural haematomas. The author concluded infantile chronic subdural hematoma can result if infantile acute subdural haematoma is treated conservatively or neglected. Rebleeding of chronic subdural haematomas has been documented radiographically [25]. The controversy centres on whether spontaneous rebleeding of chronic subdural haematomas can cause catastrophic clinical deterioration and death. It thought that rebleeding into chronic subdural haematomas is capillary in origin, under low pressure, and of insufficient volume to become a space-occupying lesion or cause cerebral edema . In contrast, acute subdural haematomas result from tearing of bridging veins, leading to more rapid haemorrhage and the accumulation of larger volumes of blood before the bleeding stops. In the study by Loh et al those who developed chronic subdural haematomas presented with seizures and disturbances of consciousness, but did not died suddenly or unexpectedly . Krous and Byard state that sudden onset of catastrophic (as opposed to gradual) clinical deterioration and sudden death in an infant whose chronic subdural haematoma was caused by inflicted injuries and then complicated by rebleeding is very rare if it ever occurs.[2223]

ACCIDENTAL FALLS

Short falls are often cited as an explanation by the defence in SBS cases. They are regularly reported as falls from bed, from the changing table or the parents arm. Although falls are the leading cause of injury in children who are brought to the emergency department and require hospital admission, they are an uncommon cause of death. A summary of more than 4500 children from 25 studies includes 1732 who generally fell from less than 5 feet (short-fall group) and 1902 cases who fell from heights of greater than one storey [26]. Among the short-fall group, several sustained linear skull factures, but none experienced injury to the central nervous system. Another recent paper described 18 children who died after short falls [27]. Responses to this controversial paper noted the series of cases to confirm that head injury causing death in young children falling short distances is extremely rare, but possible, and that despite the author's claim that biomechanical thresholds for adult brain injury were reached, an appropriate analysis was not undertaken in any of the cases [28]. The discussion of medical literature on retinal haemorrhages was considered inadequate and misleading, and the discussion of one of the cited references compared the fatal cases with cases in which the child did not die. Some authours believe that with the exception of extraordinarily unusual circumstances, short falls rarely cause the death of infants and young children.

CONCLUSION

The diagnosis of IHT/SBS requires careful evaluation of the character and extent of all components of the injury and should not rest merely on the presence or absence of one or more of the constituent lesions. The basic triad should have all the necessary features for confident diagnosis with the

reasonable conclusion that undue force has been applied. Damage to the neck or spinal cord provides further useful confirmation and the presence of gripping injuries, while often absent, can add further weight to the diagnosis. Other inflicted extracranial injuries provide evidence of inflicted injury even if they are not contemporaneous with the head injury.

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