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Fatal Head Injury an Autopsy study

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SUMMARY

Craniocerebral trauma is a condition encountered all too often in Pakistan, leading to death that may be avoidable. We report an autopsy study of 67 fatal head injuries with autopsies performed to reveal the cause of death. The type and distribution of the craniocerebral trauma, its aetiology and associated injuries are discussed. Most of the deaths in our series occurred due to large extradural haematomas, very few having associated severe primary brain injury. This suggests that there is a need to improve the referral and neurosurgical services to reduce the fatality due to craniocerebral injury.

Key Words: Craniocerebral trauma, extradural haematoma, head injury, intracerebral haematoma, intracranial haematoma, cerebral contusions, multiple trauma, subdural haematoma.

INTRODUCTION

Head injury is a common cause of death and disability leading to deaths. In nearly every country the young are affected in the most productive years of their lives. In our country as well, this is one of the most common causes of death in the earlier years of life. We conducted a retrospective study to analyse the pathological correlations of death due to head injury. Autopsy is the final investigation that may be carried out for finding out the exact cause of death and also any other co existing pathologies. This is the method used in advanced countries to increase the body of knowledge about disease processes for the last century or so. To our knowledge this is the first reported autopsy study on head injury or other pathological processes attempted in our country.

Brain injury during head trauma may be due to the initial blow, the primary injury which is preventable by health education and the after effects which are to secondary injury and preventable by prompt treatment. The primary injury may cause brain contusions, intracranial haematomas, including extradural and subdural haematomas. Brain shifts caused by haematomas as well as raised intracranial pressure causing brain hypoxia are a major cause of death. An attempt is made to identify the various factors having a bearing on the outcome and to pinpoint the avoidable causes of death.

MATERIALS AND METHODS

The records of all the subjects having a postmortem at the Forensic Medicine Department, King Edward Medical College between 1985 and 1987 were examined with a significant head injury having an autopsy performed by one of the authors (S.M.Y), were included in the study. This was done so as to eliminate any variation in autopsy technique.

All the subjects were either victims of suspected homicidal attacks, or accidental deaths from road traffic accidents, railway accidents or falls from variable heights. The reported cause of death and the deduced cause of death following autopsy were recorded.

The autopsies were done with the following technique. Injuries to the scalp, skull including fractures and the face were recorded before the calvarium was opened. The dura was examined before and after the removal of the brain to check for dural tears, traumatic or surgical. The brain was then sectioned after removal from the calvarium in three, an axial fashion so as not to miss any

intracerebral contusions and haematomas. The anatomical location of the haematomas and the relationship to any overlying calvarial fractures and superficial injuries was also noted. The haematoma volume was noted immediately as it was removed so as to prevent error due to dessication. It was noted whether the subject had any brainstem injury or pituitary injury. Any associated injuries were also recorded as well as a conclusion as to the exact cause of death and its aetiology. Other facts noted were the reported cause of death and the actual deduced cause and aetiology after autopsy. A correlation of the accuracy of the reported cause by the police was made.

RESULTS

The total number of patients in our study was 67 with 11 females and 56 males with males being 83.1% of the total. The ages varied from a minimum of 6 months to a maximum of 85 years with a mean age of 34.04 years and an SD od +/- 18.07. Only 25 of the patients (37.3%) were hospitalized prior to death.

Cause of death

Most of the deaths were due to accidents (67.2%) road traffic accidents constituted 57.2% of the total and other accidental blunt trauma including falls and Railway Accidents being 10.5% of the total. Homicide including Blunt (18 cases) and sharp trauma (4 cases) made up the rest (32.9%). These figures were obtained after autopsy conclusions without regard to the reported cause of death given in Table 1. The reported cause of death was slightly different possibly due to wording of documentation. However major discrepancies were not noted between the deduced and the reported causes of death. Especially no differences were noted in the homicidal versus the accidental categories.

Aetiology

The deduced aetiologies leading to death are given in Table 2. Intracranial haematoma was seen in fully 98.5% with only one patient not having an intracranial haematoma. Crush injury to the head and brain was seen in 14 subjects (20.9%). Extradural haematoma was present alone in 11 cases (16.4%), subdural haematoma in 6 cases (9%) with extradural haematoma/subdural haematoma combined in 7 (10.4%). Thus 35.8% of patients had an extracerebral haematoma without evidence of massive brain damage. Intracerebral haematoma was seen in 25 patients (37.3%).

Table 1: Reported aetiology

	No.	*%*	*Cummulative %*
Accidents	40	59.7	59.7%
Blunt traumas	16	23.9	83.6
Fa¹	4	6.0	89.6
Sharp trauma	6	9.0	98.5
Electric shock	1	1.5	100.0

Table 2: Causative Pathology

	No.	*%*	*Cummulative %*
Brain lacerations	3	4.5	4.5
Contusions	1	1.5	6.0
Crush injury	14	20.9	26.9
Extradural Haematoma	11	16.4	43.3
Extradural Haematoma + Subdural Haematoma	7	10.4	53.7
Intracerebral Haematoma	25	37.3	91.0
Subdural haematoma	6	9.0	100.0

Injuries

Scalp contusions were seen in 54 subjects (80.9%) out of whom 38 subjects also had scalp lacerations. A single scalp contusion was seen in 50.9% of these whereas multiple (5 or more contusions) were seen in 19.6% of the autopsies. The distribution of the Scalp injuries is given in Figure 1. Skull fractures were seen in 57 (85.1%) of subjects. The type of the fractures is given in Table 3. The distribution of the fractures is given in Fig.2.

Intracranial Haematomas and Associations

Nearly three quarters of the subjects (74.9%) had haematomas with a size greater than 75 ml and nearly 50% greater than 100 ml. It was seen that 59 of the subjects (88.1%) had an anatomical

association and contiguity between the underlying extradural haematoma (EDH)/subdural haematoma (SDH) intracerebral haematoma (ICH) and the overlying injury whether a skull fracture or scalp contusion/laceration.

Table 3: Skull fracture type

	No.	*%*	*Cummulative %*
Basal compound	2	3.0	3.0
Comminuted compounds	2	3.0	6.0
Depressed	7	10.4	16.4
Diastases	2	3.0	19.4
Egg shell	16	23.9	43.3
Linear	38	56.7	100.0

Table 4: Locations of Scalp injuries, extradural haematomas, and subdural haematomas (n=52)

	Scalp Injury		*Extradural Haematoma*		*Subdural Haematoma*	
	No.	*%*	*No.*	*%*	*No.*	*%*
LFl	13	25	10	19.2	11	21.1
RF	14	26.9	14	26.9	13	25
LT	8	15.3	16	30.6	15	28.8
RT	11	21.1	18	34.6	9	17.3
LP	7	13.5	10	19.2	7	13.5
RP	7	13.5	10	19.2	4	7.69
LO	13	25	12	23.1	10	19.2
RO	13	25	8	154.4	9	17.3
Vertex	4	7.69	5	9.6	5	9.6

LF, Left frontal; RF Right Frontal, LT Left temporal; RT, Right temporal; LP, Left parietal; RP, Right parietal; LO, Left Occipital; RO, Right Occipital

As fourteen of the subjects had crush injury these were excluded from an analysis of the haematoma locations and associations between the extradural and subdural haematoma locations. The distributions of the haematomas is shown in Table 4. Meningeal tears were seen in 25 (37.3%). Brainstem injury was noted in 43 subjects (64.2%). It was not noted whether these were primary injuries or secondary ones. Pituitary injury was seen in 18 (26.9%) of the subjects.

Associated injuries

Facial injuries, including maxillary and mandibular injuries and other associated injuries such as spinal injuries, chest injuries and limb injuries were also noted. Facial injury was seen in 41 autopsies (61.2%) with a maxillary fracture in 13 and a mandibular fracture in 15 subjects. There was no spinal injury in the whole study whereas fractured ribs were seen in 14.9%. Limb fractures were seen in 31.3% (21 subjects).

DISCUSSION

Cranio-cerebral trauma is one of the largest killers in all countries. Western studies put the incidence of Head injury an annual incidence rate of 17.3 per 100,000 population with a mortality rate of 5.2 per 100,000 per year[1]. In South Africa where the patient population is similar to ours the violent deaths even in the paediatric population were mainly attributable to head injury with 54.4% of an autopsy population having died due to head injury[2]. Japanese figures also put one of the highest causes of death as being road traffic accidents with about 10% of all autopsies being for patients with RTAs.[3] In our study also of an autopsy population the deaths due to Road traffic accident form a major component of the violent deaths being 57.2% of the study group with male predominance. Homicidal injury is also a large component being 32.9% of the subjects autopsied. It is of course possible that our figures underestimate the total number of road accident deaths as many go unreported and in others relatives do not give permission for autopsy. This is the experience in other countries as autopsies are required only in about 6% cases of RTAs in Japan and this is only to find out whether some negligence is involved[3].

Pathophysiology

Head injuries may be due to direct blows to the head which may cause local deformations and mechanical effects due to the force of the blow. Other effects are due to the acceleration and deceleration forces set into motion due to the application of mechanical force. A third mechanism of injury is due to the rapid acceleration and

deceleration injuries that occur in high speed traffic accidents. The initial blow produces scalp injuries and deformation of underlying bone leading to skull fractures. This may be fatal at the site of accident or may cause the formation of related extradural haematomas and subdural collections of blood. If there is enough force to deform the inner table of the skull there may be associated intracerebral contusion damage. Death is produced due to the space occupying effects of these haematomas which lead to initially raised intracranial pressure and consequently to decreased cerebral perfusion (Cerebral Perfusion Pressure = Mean Arterial Pressure - Intracranial Pressure) once compensatory mechanisms have been overcome[4].

In our study the main injuries leading to death were those due to direct contact effects and focal injuries. Fourteen subjects had a severe crush injury and this was probably a primarily fatal injury. This was borne out by the fact that none of the crush injury subjects was admitted to hospital. There were skull fractures in 57 patients and nearly all of these had underlying extradural haematomas or associated subdural haematomas. In experimental studies it has been noted that fractures were associated with shallow extradural and subdural haematomas[5]. In our study the size of the haematomas was substantial (25-750 mls) mean 128.0 mls (SD 123.10) thus showing that these were clinically significant. Post traumatic extradural haematomas are due to bleeding from the dural arteries and veins associated with overlying fractures[6] while studies have shown that acute subdural haematomas occur due to rupture of bridging veins associated with high strain acceleration injuries due to falls and blows where 72% are due to high-strain falls and assaults and 24% are due to lower strain-rate vehicular injuries[7]. This bears well with the pattern of injuries in our series where most of the subjects were pedestrians struck by vehicles or were assaulted by blunt weapons. One may independently conclude from the analysis of the cases that in low velocity and impact injury is mainly responsible for the fatal head injuries coming to autopsy. Diffuse axonal injury unassociated with haematomas and associated with diffuse brain swelling was not seen in our study which is also consistent with the predominant aetiology causing death and not allowing changes to develop in the brain over time.

Coup injuries are described as those underlying the impact site while the countercoup injuries are described as those opposite the site of impact most often in a straight line from the point of impact. However as Gurdjian has pointed out these are most often found in the temporal and frontal regions no matter what the point of impact6. Although the countercoup injuries are common in the West along with diffuse cerebral oedema we did not find these in our study. The reason may be that these contusional haematomas develop over a period of time after the head injury often taking as much as 48-72 hours to develop[7].

Those subjects in our study who were not crushed at the site of accident died due to massive intracranial haematomas and resultant brain herniation within hours.

Management

Only 25 of our autopsy subjects (37.2%) were ever hospitalised. Initial management of head injury or for that matter any trauma involves the early transport of the patients to a medical facility able to deal with the emergency surgical situation. It has been shown quite clearly for example that surgical treatment of acute subdural haematoma within the first four hours of the injury causes great improvements in prognosis[8]. This is usually done in a major trauma centre or a neurosurgical unit. Time is of the essence and good ambulance services are essential for accident victim transportation[9] General surgeons must be trained to recognise intracranial haematomas and deal with them including performing surgery for evacuation[10].

If crush injuries are excluded extradural haematomas are responsible for most of the deaths in our series and these are associated in some (10.4% of the total study-7 subjects) with subdural haematomas and intracerebral haematomas (16.4%). Prognostically diffuse axonal injury and subdural haematomas tend to have the worst outcome. In our study brainstem injury was seen in most of our patients 64.2%. Only 26.9% had pituitary injury. It is assumed that most of the brainstem injury was secondary to brain herniation. However in better organised societies it has been determined that most of the patients dying early after head injury have either a diffuse homogenising necrosis of the brain or there is irreversible brainstem injury[11]. These patients, however, have concomitant surgically important haematomas only rarely. There is little or no therapy for these pathological conditions while surgical therapy for extradural haematoma is quick,

simple and effective. We may deduce from our study especially if the management and treatment of head injury under ideal circumstances is considered, that better management of trauma and specifically neurotrauma in our society would lead to a better prognosis of head injury than possibly in the West.

CONCLUSION

Further hospital based studies with established protocols need to be done to determine our needs so that the most cost effective method of providing head injury management is evolved. Our autopsy study possibly serves to point us in a correct direction

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