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TRAFFIC ACCIDENTS;
CORRELATION BETWEEN SKULL FRACTURES AND INTRACRANIAL LESIONS

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ABSTRACT... Objective: To find out the correlation between different types of skull fractures and intracranial lesions in traffic accidents. **Setting:** Patients examined in surgical emergency Allied Hospital, Punjab Medical College, Faisalabad. **Duration:** Two years from 1st January 1996 to 31 December 1997. **Materials and Methods:** In this study, the reports belonging to surviving 250 cases that had head injuries in traffic accidents were examined retrospectively. Data was collected from surgical emergency record office. Collected information was analyzed with descriptive and inferential statistics. **Results:** The rate of intracranial lesions among the cases with the skull fracture was 39.0%, while the rate of skull fracture among the cases with the intracranial lesion was 50.3% (P < 0.001). Depressed fractures were more in males, while linear fractures were more in females as well as in young males as whole intracranial lesions were less in patients with skull fracture due to lower intra cranial pressure.

Key words: Skull, fracture, intracranial lesion, accident, hemorrhage.

INTRODUCTION

Head injury is a frequent cause of emergency department attendance, accounting for approximately 3.4% of all presentations, with an incidence of around 450 cases per 100,000 population per year. Head injury associated with traumatic brain injury (TBI) occurs with an incidence of 20-40 cases per 100,000 population per year. It is the most common cause of death in young adults (age 15-24yrs) and is more common in males than the females. Road traffic accidents (RTAs) are most common cause of TBI in UK, followed by falls and accidents[1].

Blunt head injuries are most frequently caused by traffic accidents, assaults, falling or jumping from high altitudes, home accidents, industrial accidents or sport accidents, birth traumas, incidents of terror and wars. These are the most serious traumas in terms of morbidity and mortality[2]. After the trauma, blunt traumatic lesions of scalp and even more serious lesions such as skull fractures and intracranial lesions may develop. A strike to head with enough force ends with linear fractures. Faster and more forceful strike cause secondary fractures in

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shape of asterisks and if effecting a limited area, it results in depressed fractures[3].

The occurrence, degree of deformation and extent of fracture is not only related to amount of strike power (i.e. energy level) and the ratio of strike power to area of strike, but also to the physical properties of the skull at the point of contact including thickness of the scalp, amount of hair and also the thickness and elasticity of the individual skull. Skull fractures may be localized to the dome or base of the skull and may be linear, diastatic, depressed or fragmented in shape. They may also be in form of open or closed fractures[4,6].

In some regions of skull, such as supraorbital ridges in front, temporal bones at the sides, occipital curves at back, there are vertical bone formations called Rathke's columns which increase strength of skull. The petrous portion of temporal bone, greater wing of sphenoids, occipital protuberance, and glabellae are well supported. However, side parts of parietotemporal, frontal, and occipital bones are relatively weaker regions where linear fractures are more likely to develop[7,8]. Linear fractures may lead to cerebrocortical contusion or intracranial hematoma.

Skull fractures were shown in 80% of cases that died due to head injury and frequency of intracranial hematoma is higher in cases with skull fractures compared with cases with no skull fractures[9]. Although a high strike power is required to develop a skull fracture, no brain lesion may exist in a case with skull fracture. Similarly, no skull fracture may be present in a case with large brain damage. Although developing skull fractures require a high degree of applied energy, there is a weak correlation between skull fractures and brain lesions[4].

Depressed fractures are more serious due to high risk of injury to the underlying dural sinuses and brain. Intracranial lesions like cerebral contusions and lacerations, diffuse axonal injury, concussions and brain edema may develop following head injuries and may be localized to epidural, subdural, subarachnoidal, and intracerebral regions[3–10].

The patients with head injury may present fully conscious, semiconscious or unconscious depending upon the nature extent & duration of brain trauma. More than two million patients receive head injuries in USA per year & 25% of these are hospitalized[11].

In this study, the relationship between skull fractures following head injury and intracranial lesions was investigated, and the factors playing a role in the etiopathogenesis of this relationship were studied.

MATERIALS AND METHODS

The materials and methods, which are used in the study, are explained through various techniques and tools. These techniques and tools are used for data collection, analysis and interpretation.

In this study, the reports belonging to surviving 250 cases of either sex and irrespective to age group that had head injuries in traffic accidents between 01.01.1996 and 31.12.1997 were examined retrospectively from surgical emergency department of Allied Hospital/PMC, Faisalabad. Although, the cases included pedestrians, automobile, bus, truck, motorcycle and bicycle drivers and passengers. We avoided classification about this subject, because most of the cases (n=174) did not have the classification in their accident reports which were referred to the forensic medical society by the courts of laws.

Depending on their cranial x-ray and brain tomography findings, the cases having skull fractures and brain lesions, cases having skull fractures with no brain lesion and cases having brain lesion without any skull fractures were selected. They were examined in detail according to their sex, age, and localization of lesions. Presence of any relationship between skull fractures and brain lesions were investigated.

Collected information was analyzed using chi-square test. (Statistical analysis was done through SPSS).

RESULTS

Total 250 cases were studied retrospectively that were categorized as having skull fractures with brain lesions,

skull fractures with no brain lesions, brain lesions with no skull fractures according to their radiographic findings.

Out of 250 cases, it was found that 76(30.4%) had only linear fractures, 35(14.0%) had depressed fractures, 46(18.4%) had linear fractures with intracranial lesions, 24(9.6%) had depressed fractures with intracranial lesions and 69(27.6%) had only intracranial lesions. The rate of intracranial lesion among the cases with the skull fracture was 39.0%, while the rate of skull fracture among the cases with the intracranial lesion was 50.3% ($P < 0.001$).

When we examine the distribution of cases according to sex, 75.6% (n=189) of cases were male and 24.4% (n=61) were female. Male to female ratio was 3.2/1. Occurrence of different types of fractures was as follows For males: 27.3% for only linear fractures, about 15% for just depressed fractures, 18.4% for linear fractures with intracranial lesion, 11.2% for depressed fractures and intracranial lesion, 28.0% for only intracranial lesion. In females: 39.6% for only linear fractures, 9.6% for only depressed fractures, 18.8% for linear fractures and intracranial lesion, 5.6% for depressed fractures and intracranial lesion, and 26.0% for pure intracranial lesion. Male to female ratios were 2.4/1 for entire linear fractures, 5.2/1 for entire depressed fractures, and 3.6/1 for entire intracranial lesions. Linear fractures were more frequent among females whereas depressed fractures were often among males ($[chi]^2$: 9.68, df: 4, p: 0.046).

It was concluded that the mean age was 26.3. Of entire cases, 44.0% (n=110) were within age group 0–20 and 47.2% of cases (n=118) were within range of 21–50 years. 8.8% of cases were in 51 years and above. Most of cases having only linear fractures accumulated in the age group of 0–20. With the advance of age, the rate of cases having only linear fractures decreased both in general population and among age groups. The rate of depressed fractures was higher in the age group of 0–30 years. ($[chi]^2$: 16.28, df: 4, p: 0.003).

Of the 250 cases, 76 had only linear fractures. Among them, 23 cases (30.3%) had frontal, 14 (18.4%) occipital, 11 (14.5%) fracture of temporal and 11 (14.5%) cases had fracture of parietal bone. In the other cases (n=17), the fractures were related to more than one bone. Location of fractures among 46 cases having linear fractures and intracranial lesion were as follows: 11 (23.9%) frontal, 11(23.9%) temporal, 9 (19.6%) parietal and 6 (12.0%) occipital bones and in 10 cases (20.6%), fractures were related to more than one bone.

Among the total cases, 35 had only depressed fractures. Location of depressed fractures was distributed in to: 14 (42.8%) frontal, 9 (25.7%) parietal, 4 (11.4%) temporal, 3 (8.6%) occipital bones and in 5 cases were related to more than one bone. Among 25 cases having depressed fractures and intracranial lesions, locations of depressed fractures were as follows: 8 (32.0%) parietal, 6 (24.0%) frontal, and 1 (4.0%) temporal, 2 (8.0%) occipital, and 8 (32.0%) more than one bone.

Location of linear and depressed fractures was distributed to: 30.0% frontal, 20.8% parietal, 15.2% temporal, 12.4% occipital, 8.4% parietotemporal, 6.8% frontoparietal, 2.8% parietooccipital, 1.6% temporooccipital, 1.2% frontotemporal, and 0.8% sphenoid bones.

Among 69 cases having only intracranial lesions, most frequent lesions were contusion with 33.3% (n=23), intracerebral hemorrhage with 31.8% (n=22) and subdural hematoma with 18.8% (n=13). Most frequent lesions among 46 cases having both linear fractures and intracranial lesions were epidural hematoma with 39.1% (n=18), subdural hematoma with 26.1% (n=12) and intracerebral hemorrhage with 19.5% (n=9). Most frequent lesions among 25 cases having both depressed fractures and intracranial lesions consisted of epidural hematoma with 28.0% (n=7), contusion with 24.0% (n=6), and laceration of dura mater with 16.0% (n=4) respectively. In the review of all cases, having intracranial lesions, of the 140 cases, 15 (10.7%) had multiple lesions. Intracranial lesions included contusion (25.7%), epidural hematoma (25.0%), intracerebral hemorrhage (24.0%), subdural hemorrhage (21.4%), subarachnoidal hemorrhage (7.8%), lacerations (3.6%), hygroma (2.9%), and intraventricular hemorrhage (1.4%).

DISCUSSION

In this study, we have reviewed 250 cases, each of which had a head injury in a traffic accident. The cases consisted of 30.4% only linear fractures, 13.8% depressed fractures, 18.4% linear fractures plus intracranial lesions, 9.8% depressed fractures plus intracranial lesions and 27.6% only intracranial lesions. As a result, of the 122 linear fractures and 59 depressed fractures, 37.7% and 41.5% respectively were accompanied by intracranial lesions.

In this study, there were 75.4% men and 24.6% women. Male to female ratio was 3.1/1. This ratio is similar to findings of Azmak and his colleagues[12] and Akin O[13] that are 3.7/1 and 3.36/1 respectively. Those studies were performed in Turkey. However, it is obviously higher than findings of Ingebrigstein and his colleagues[14] and Tiret and his colleagues[15] that are 1.7/1 and 2.07/1 respectively. Those studies were performed outside Turkey. In the classic literature, male to female ratio varies between 2/1 and 2.8/1[16]. In other studies performed in Turkey, male to female ratio is higher since males are more active in daily life[12,13]. In this study, the males injured as either a pedestrian or a driver could explain the higher ratio of males. Male to female ratio for whole linear fractures and whole depressed fractures are 2.4/1 and 5.2/1 respectively. When we consider that all cases are randomly selected and were exposed to similar traffic-related impacts, the higher ratio of linear fractures in females could be explained by the female skull being weaker and more flexible with evenly distributed impact energy. Similarly, a higher ratio of depressed fractures in males could be explained by the male skull being thicker and stronger and absorbing impact energy.

Of all the cases 44.2% fall into age group 0–20, and 19.6% fall into age group 21–30 (mean age was 26.3). These findings were compared with findings of other studies related to blunt head injuries and were found to be similar to Azmak and associates[12] whose mean age 28.4% and 22.7% of cases within age group 20–30, to Haug and associates[17] 54% within age group 16–30 and to classic literature being concentrated between ages 15 and 24. It differs from report of Akin O[13] that is 35.4% for age group 0–10. This study has shown that just linear fractures concentrate between ages 0 and 20, and decrease by advance in age. Linear fractures being more frequent among the young could only be explained by the flexibility of their skull bones. The relative increase of intracranial lesions obviously occurs with the advance in age. This situation could be explained by atherosclerotic changes of intracranial vessels being a risk factor for intracranial lesions.

It was concluded that the linear fractures were located to frontal, temporal and parietal bones in decreasing order. The depressed fractures were located to frontal, parietal, and temporal bones in decreasing order. There were no statistical difference between the cranial lesion and the localization of linear or depressed fractures. When considering all cases, linear and depressed fractures were located in the following list of bones in decreasing frequency: frontal (29.8%), parietal (20.7%), temporal (15.2%), occipital (12.4%), parietotemporal (8.6%), frontoparietal (6.7%), parietooccipital (2.8%), temporooccipital (1.9%), frontotemporal (1.1%), and sphenoidal (0.9%) regions. Depressed fractures in the regions of frontal and parietal and, linear fracture in the regions of temporal and occipital were found at higher rates ($P < 0.001$). These findings show similarity to reports of Colak and associates[18]. They have reported in a study, about radiologic examination of head injuries, that the lesions were located in the frontal (26.3%), parietal (21.1%), temporal (21.1%), frontoparietal (10.5%), parietotemporal (5.3%), and occipital (5.3%) regions. Nicol and associates[19] reported rate of multiple fractures in children due to traffic accidents, was 58.8%. Miura and associates[20] reported the incidence of depressed fractures in parietal bone due to head injuries to be 55.8% in a study performed on children aging between 2 and 7.

Of the 140 cases having intracranial lesions, 70 (50.0%) accompanied a skull fracture and 15 (10.7%) had multiple lesions. Comparisons between the existence or type of fractures and one or multiple intracranial lesions were not statistically significant ($P > 0.05$).

Head injuries due to traffic accidents are most common in the young, and the rate of linear fractures is higher

than that of depressed fractures in them. Likewise, of the 122 cases having linear fractures, 59 cases having depressed fractures, 46 (37.7%), and 25 (42.3%) were accompanied by an intracranial lesion respectively. Of the 181 cases having a skull fracture, 70 (38.6%) were accompanied by an intracranial lesion. Considering these findings, we can state that presence of skull fractures lowers the incidence of intracranial lesions by lowering the intracranial pressure. This hypothesis needs to be supported by experimental studies.

Traffic accidents often cause epidural hematomas and 65 to 90% of them are accompanied by a skull fracture. Epidural hematomas with no fracture are usually seen in children since they have very elastic bones, which permit dura to separate from bones. Epidural hematomas with no skull fracture are very rare over 60s and in first 2 years of life since dura is firmly attached to inner layer of skull. About 10% of epidural hematomas are accompanied by subdural hemorrhage[3,5,7,21,22]. In this study, of 69 cases having epidural hematomas, 73.9% had accompanying skull fractures. Among cases, having epidural hematomas, the rate of accompanying subdural hematoma was found to be 13.0% which is close to reports in the literature[22].

Of subdural hematomas, 24% are due to traffic accidents, which usually present together with other brain lesions such as contusion and lacerations[4,5,10]. In this study, intracranial lesion accompanies only 30.5% of subdural hematomas and 69.5% are alone. This finding is different from reports in the literature.

Subarachnoidal hemorrhage may be only a finding of serious head injuries, however other intracranial lesions such as intracerebral hemorrhage and skull fractures may also be present[7,22]. In this study, 68.2% of subarachnoidal hemorrhages were alone, and other intracranial lesions accompanied the remaining cases.

Intracerebral hematomas are often demonstrated in serious head injuries and may be developed by coup and countercoup mechanisms in any regions of cerebral hemispheres such hematomas may also open into the ventricular system[5,23,24]. In this study, 24.0% of cases having intracranial lesions were accompanied by intracerebral hemorrhages and of 33 intracerebral hemorrhages, 2 were accompanied by intraventricular hemorrhages. One case had intraventricular hemorrhages without any intracerebral hemorrhage.

Contusions are most common traumatic intracranial lesions and are always caused by trauma[4,7,10,25]. Similarly, in this study, of 140 cases, 36 had contusions and found to be the most common intracranial lesions by 25.7%.

Lacerations either develops due to open skull fractures or bone fragments in closed skull fractures. Other intracranial lesions may accompany them. Lower surfaces of temporal lobes and orbital faces of frontal lobes are most commonly affected[4,5,10,25]. In this study, of the 140 cases, 6 (4.3%) had lacerations and all lacerations were accompanied by skull fractures.

Other intracranial lesions are brain edema and axonal injury. Diffuse axonal injury has been demonstrated in 35–50% of serious head injuries. They are mostly due to head injuries caused by traffic accidents[4,10,26-28]. In this study, no case of diffuse axonal injury has been demonstrated. Hygroma was found to be present at a rate of 2.9% (8/279).

Surgery rates for all cases, depressed fractures plus intracranial lesions, just depressed fractures, linear fractures plus intracranial lesions, only intracranial lesions and only linear fractures are 20.6%, 71.4%, 36.2%, 25.0%, 14.5% and 0.0% respectively. In this study, this rate is 50.8% in all cases with depressed fractures (60/118). The rate of surgical operation for cases with intracranial lesions associated by linear or depressed fractures is higher than cases with only fractures or only intracranial lesions ([chi]2: 51.15, $P < 0.001$).

CONCLUSIONS

We reviewed skull fractures and/or intracranial lesions due to traffic accidents, and found depressed fractures to be more common among males whereas linear fractures to be more common among females and young males. In the elderly males, the skull architecture is thicker and

stronger than females and young males. This, in turn results in inability to distribute strike energy and to be absorbed at the place of strike.

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PREVIOUS RELATED STUDIES

- M. Hussain Khan, Iftikhar Ahmed, Niamatullah Zia, *Tariq Sohail Babar, Khalid Shakeel Babar,* Road Traffic Accidents; Study of risk factors. Professional Med J 2007; Vol: 14, No. 2, 323-327

CORRECTION

The amendment of the Professional Vol:16, No.01 (Jan, Feb, Mar 2009) Prof-1435 page 29 are as under;

INCORRECT
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MANAGEMENT OF FISTULA IN ANO;
FIRM CUTTING SETON VS LOOSE SETON / FISTULOTOMY FOR SUPRASPHINCTERIC AND HIGH TRANS-SPHINCTERIC

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CORRECT

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