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The correlation between skull fractures and intracranial lesions due to traffic accidents

[Trafik kazalarına bağlı kafatası kırıkları ve intrakranyal lezyonlar arasındaki ilişki]

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Abstract

In this study, it was aimed to investigate the relationship between skull fractures and intracranial lesions following head injury. For this purpose, 500 cases, which were referred to the Third Committee of Council of Forensic Medicine in Istanbul due to traffic accidents by the courts of laws between 1998 and 2000, were examined retrospectively. They were categorized in three groups based on findings of their cranium X-rays and brain tomographies. 1- The cases who have fractures in skull bones with brain lesions 2- The cases who have fractures in skull bones with no brain lesions 3- The cases who have brain lesions with no skull fractures. They were examined in detail according to age, sex, localization of skull fractures and brain lesions, and if surgery was applied or not. Of the cases, 152 (30.4%) had only linear fractures, 69 (13.8%) had depressed fractures, 92 (18.4%) had linear fractures plus intracranial lesions, 49 (9.8%) had depressed fractures plus intracranial lesions and 138 (27.6%) had only intracranial lesions. The rate of intracranial lesion among the cases with the skull fracture was 38.9% (141/362), while the rate of skull fracture among the cases with the intracranial lesion was 50.3% (141/279) ($p<0.001$). Male to female ratios were 2.4/1 for linear fractures, 5.2/1 for depressed fractures, and 3.5/1 for intracranial lesions. Linear fractures were more frequent among females whereas depressed fractures were often among males (χ^2: 9.68, df: 4, p: 0.046). The mean age was 26.3. The rate of depressed fractures was higher the age groups of 0-30 years. (χ^2: 16.28, df: 4, p: 0.003). Depressed fracture in the regions of frontal and parietal and, linear fracture in the regions of temporal and occipital were found at higher rates ($p<0.001$).

Özet

Çalışmamızda, travma sonrası kafatası kırıkları ile intrakranial lezyonların ilişkisinin araştırılması amaçlanmıştır. Bu amaç için, 1998-2000 yılları arasında Adli Tıp Kurumu 3. İhtisas Kurulu'na trafik kazası sonucu kafa travması nedeniyle adli rapor düzenlenmesi için müracaat ettirilen 500 olgu retrospektif olarak değerlendirilmiştir. Onlar Kranial x-ray ve/ veya Cranial Tomography ' sinin radyolojik incelemeleri sonucunda 1-kafa kemiklerinde kırık ve beraberinde beyin lezyonu saptanan olgular, 2-kafa kemiklerinde kırık mevcut olmasına rağmen beyin lezyonu bulunmayan olgular , 3-kafa kemiklerinde kırık olmaksızın beyin lezyonu saptanmış olgular olmak üzere üç gruba ayrılmış ve yaş, cinsiyet, kafatası kırıkları ve beyin lezyonlarının lokalizasyonları, cerrahi tedavi uygulanıp-uygulanmaması yönünden ayrıntılı olarak değerlendirilmiştir. Olguların 152 (%30,4) sinde yalnız lineer fraktür, 69 (%13,8) unda çökme fraktürü, 92 (%18,4) sinde lineer fraktür + intrakranial lezyon, 49 (%9,8) unda çökme fraktürü + intrakranial lezyon, 138 (%27,6) inde ise intrakranial lezyon bulunmakta idi. Kafatası kırığı olan olgular arasında lineer kırık oranı 38.9% (141/362), intrakranial lezyonu olan olgularda kafatası kırığı oranı 50.3% (141/279) idi ($p<0.001$). Erkek/kadın oranları ise tüm lineer kırıklar için 2.4/1, tüm çökme kırıkları için 5.2/1, tüm intrakranial lezyonlar için 3.5/1 olarak hesaplandı. Lineer kırık oluşumu kadınlarda, çökme kırığı oluşumu ise erkeklerde daha fazla görülmekteydi (χ^2: 9.68, df: 4, p: 0.046). Ortalama yaş 26.3 olup, çökme kırıkları en fazla 0-30 yaş grubundaydı (χ^2: 16.28, df: 4, p: 0.003). Çökme kırıkları frontal ve parietal bölgelerde, lineer kırıklar temporal ve occipital bölgelerde daha yüksek orana sahipti ($p<0.001$).

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In conclusion, 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 male, the skull architecture is thicker and stronger than females and young males. We can state that presence of skull fractures lowers the incidence of intracranial lesions by lowering the intracranial pressure.

Keywords:

Skull fractures, intracranial lesions, correlation.

Sonuç olarak, trafik kazasına bağlı olarak meydana gelen kafatası kırıkları ve/veya intrakranial lezyonlar incelendiğinde; çökme kırıklarının erkeklerde, lineer kırıkların kadınlarda ve genç erkeklerde daha fazla görüldüğünü belirledik. Erkeklerde, kafatası kadınlar ve gençlere göre, daha kalın ve sağlamdır. Kafatasında kırık varlığının, kafa içi basıncı azalmasına sebep olarak intrakranial lezyon oluşumunu engellediği düşünülmüştür.

Anahtar Kelimeler:

Kafatası kırıkları, intrakranial lezyonlar, ilişki.

1.Introduction

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. They are the most serious traumas in terms of morbidity and mortality (1). 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 more forceful strikes cause secondary fractures in shape of asterisks and if effecting a limited area, it results in depressed fractures (2).

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 for fragmented in shape. They may also be in form of open or closed fractures (3-5).

In some regions of skull, such as supraorbital ridges in front, temporal apexes 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, great 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 (6,7). 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 to cases with no skull fractures (8). 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 (3).

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 (2-9).

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 was studied.

2. Material and method

In this study, the reports belonging to surviving 500 cases that had head injuries in traffic accidents between 01.01.1998 and 31.12.2000 were examined retrospectively. Although, the cases included pedestrians, automobile, bus, trunk, motorcycle and bicycle drivers and passengers, we avoided from classification about this subject, because most of the cases (n=347) 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 age, sex, and localization of lesions. Presence of any relationship between skull fractures and brain lesions were investigated.

Chi square test was performed for the statistical analysis.

3. Results

Totally, 500 cases that were categorized as having skull fractures plus brain lesions, skull fractures with no brain lesions, brain lesions with no skull fractures according to their radiographic findings which were referred to the council of forensic medicine by the

courts of laws between years 01.01.1998-31.12.2000. All cases had a head injury in traffic accidents.

Of the 500 cases, 152 (30.4%) had only linear fractures, 69 (13.8%) had depressed fractures, 92 (18.4%) had linear fractures plus intracranial lesions, 49 (9.8%) had depressed fractures plus intracranial lesions and 138 (27.6%) had only intracranial lesions. The rate of intracranial lesion among the cases with the skull fracture was 39.0% (141/362), while the rate of skull fracture among the cases with the intracranial lesion was 50.3% (141/279) ($p<0.001$) (Table-1).

Table 1. *Sex and age groups for cranial injuries*

LESION	Only Linear Fracture			Only Depressed Fracture			Linear Fracture & Intracranial Lesion			Depressed Fracture & Intracranial Lesion			Only Intracranial Lesion			TOTAL		
Sex / Age	M	F	T	M	F	T	M	F	T	M	F	T	M	F	T	M	F	T
0-5	11	13	24	5	2	7	5	2	7	2	2	4	3	2	5	26	21	47
6-10	12	8	20	5	2	7	5	4	9	4	0	4	4	4	8	30	18	48
11-15	14	7	21	8	0	8	6	1	7	3	2	5	10	2	12	41	12	53
16-20	16	6	22	6	1	7	11	1	12	6	2	8	20	4	24	59	14	73
21-25	10	4	14	4	2	6	3	3	6	8	0	8	19	0	19	44	9	53
26-30	11	2	13	7	0	7	11	0	11	5	1	6	6	2	8	40	5	45
31-35	5	1	6	3	1	4	9	4	13	3	0	3	8	5	13	28	11	39
36-40	6	2	8	5	0	5	6	2	8	2	0	2	8	2	10	27	6	33
41-45	8	2	10	4	1	5	3	0	3	4	0	4	11	4	15	30	7	37
46-50	4	2	6	5	1	6	5	3	8	2	0	2	5	1	6	21	7	28
51-55	0	1	1	2	1	3	2	1	3	1	0	1	4	2	6	9	5	14
56-60	4	0	4	2	0	2	1	0	1	1	0	1	2	2	4	10	2	12
61-65	1	0	1	0	1	1	1	1	2	0	0	0	2	1	3	4	3	7
65-70	1	1	2	1	0	1	0	0	0	1	0	1	0	1	1	3	2	5
71-71<	0	0	0	0	0	0	1	1	2	0	0	0	4	0	4	5	1	6
TOTAL	103	49	152	57	12	69	69	23	92	42	7	49	106	32	138	377	123	500

(M: male, F: female, T: total).

When we examine the distribution of cases according to sex, 75.4 % (n=377) of cases were male and 24.6% (n=123) were female. Male to female ratio was 3.1/1.

Rates of cases are as follows: For males: 27.3 % for only linear fractures, 15.1% for just depressed fractures, 18.3% for linear fractures plus intracranial lesion, 11.1% for depressed fractures and intracranial lesion, 28.1% for only intracranial lesion. In female group: 39.8% for only linear fractures, 9.8% for only depressed fractures, 18.7% for linear fractures and intracranial lesion, 5.7% 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 3.5/1 for entire intracranial lesions. Linear fractures were more frequent among females whereas depressed fractures were often among males (χ^2: 9.68, df: 4, p: 0.046) (Table-1).

The mean age was 26.3. Of entire cases, 44.2% (n=221) were within age group 0-20 and 47.0% of cases (n=235) 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 the age group of 0-30 years. (χ^2: 16.28, df: 4, p: 0.003) (Table-1).

Of the cases, 152 had only linear fractures. Among them, there were 45 (29.6%) in frontal, 28 (18.4%) occipital, 23 (15.1%) temporal and 22 (14.5%) cases in parietal bone. In the other cases (n=34), the fractures were related to more than one bone. Location of fractures among 92 cases having linear fractures and intracranial lesion are as follows: 22 (23.9%) frontal, 22 (23.9%) temporal, 18 (19.6) parietal and 11 (12.0%) occipital bones and in 19 cases (20.6 %), fractures were related to more than one bone (Table-2).

Among the whole cases, 69 had only depressed fractures. Location of depressed fractures was distributed in to: 29 (42.0%) frontal, 19 (27.5%) parietal, 7 (10.1%) temporal, 3 (4.3%) occipital bones

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and in 11 cases were related to more than one bone. Among 49 cases having depressed fractures and intracranial lesions, locations of depressed fractures are as follows: 16 (32.6%) parietal, 12 (24.5%) at frontal, and 3 (6.1%) temporal, 3 (6.1%) occipital, and 15 (30.6%) more than one bone (Table-3).

Table 2. *Localization of linear fracture(s) with or without intracranial lesion.*

Localization of Linear Fractures	Intracranial Lesion (-)	Intracranial Lesion (+)	TOTAL
Frontal	45	22	**67**
Parietal	22	18	**40**
Temporal	23	22	**45**
Occipital	28	11	**39**
Frontoparietal	10	6	**16**
Frontotemporal	0	2	**2**
Parietotemporal	15	7	**22**
Parietooccipital	6	0	**6**
Temporooccipital	1	3	**4**
Sphenoid	2	1	**3**
TOTAL	**152 (%62.3)**	**92 (%37.7)**	**244 (%100.0)**

χ^2: 5.56, df: 4, p: 0.24 * Sphenoid fractures and the cases related to more than one bone were evaluated as one group.*

Table 3. *Localization of depressed fracture(s) with or without intracranial lesion.*

Localization of Depressed Fractures	Intracranial Lesion (-)	Intracranial Lesion (+)	TOTAL
Frontal	29	12	**41**
Parietal	19	16	**35**
Temporal	7	3	**10**
Occipital	3	3	**6**
Frontoparietal	5	3	**8**
Frontotemporal	1	1	**2**
Parietotemporal	1	8	**9**
Parietooccipital	2	2	**4**
Temporooccipital	2	1	**3**
TOTAL	**69 (%59)**	**49 (%41)**	**118 (%100.0)**

χ^2: 6.31, df: 4, p: 0.18 * The cases related to more than one bone were evaluated as one group.*

Location of linear and depressed fractures are distributed to: 29.8% frontal, 20.7% parietal, 15.2% temporal, 12.4% occipital, 8.6% parietotemporal, 6.7% frontoparietal, 2.8% parietooccipital, 1.9% temporooccipital, 1.1% frontotemporal, and 0.8% sphenoid bones (Table 4).

Among 138 cases having only intracranial lesions, most frequent lesions were contusion with 33.3% (n=46), intracerebral hemorrhage with 31.2% (n=43) and subdural hematoma with 19.6% (n=27). Most frequent lesions among 92 cases having both linear fractures and intracranial lesions were epidural hematoma with 40.2% (n=37), subdural hematoma with 26.1% (n=24) and intracerebral hemorrhage with 18.5% (n=17). Most frequent lesions among 49 cases having both depressed fractures and intracranial lesions consisted of epidural hematoma with 28.6% (n=14), contusion with 22.2% (n=12), and laceration of dura mater with 18.5% (n=10) respectively. In the review of all cases, having intracranial lesions, of the 279 cases, 30 (10.7%) had multiple lesions. Intracranial lesions included contusion (25.8%), epidural hematoma (24.7%), intracerebral hemorrhage (24.0%), subdural hemorrhage (21.2%), subarachnoidal hemorrhage (7.9%), lacerations (3.8%), hygroma (2.9%), and intraventricular hemorrhage (1.8%) (Table-5)

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Table 4. *Localization of linear and depressed fractures.*

Localization of fractures	Linear Fractures	Depressed Fractures	TOTAL
Frontal	67	41	**108**
Parietal	40	35	**75**
Temporal	45	10	**55**
Occipital	39	6	**45**
Frontoparietal	16	8	**24**
Frontotemporal	2	2	**4**
Parietotemporal	22	9	**31**
Parietooccipital	6	4	**10**
Temporooccipital	4	3	**7**
Sphenoid	3	0	**3**
TOTAL	**244**	**118**	**362**

χ^2: 20.98, df: 4, p< 0.001 * Sphenoid fractures and the cases related to more than one bone were evaluated as one group.*

Table 5. *Type of Intracranial lesion and fracture*

Type of Intracranial Lesion	Only Intracranial Lesion	Linear fracture + Intracranial Lesion	Depressed fracture + Intracranial Lesion	TOTAL
EDH	16	31	10	**57 (%20.4)**
SDH	20	17	4	**41 (%14.8)**
SAH	11	4	0	**15 (%5.4)**
ICH	33	15	5	**53 (%19.0)**
Laceration	0	1	10	**11 (%3.8)**
Contusion	39	12	12	**63 (%22.6)**
Hygroma	2	2	3	**7 (%2.5)**
EDH + SDH	1	4	3	**8 (%2.9)**
SAH + ICH	1	0	0	**1 (%0. 4)**
IVH	1	1	0	**2 (%0. 7)**
SDH+ Contusion	2	1	0	**3 (%1.1)**
Contusion+ Hygroma	1	0	0	**1 (%0.4)**
SDH + SAH +ICH	1	0	0	**1 (%0. 4)**
SDH + ICH	2	0	1	**3 (%1.1)**
SAH + Contusion	2	1	0	**3 (%1.1)**
IVH + ICH	3	0	0	**3 (%1.1)**
SDH + ICH +Contusion	1	0	0	**1 (%0. 3)**
Contusion + ICH	1	0	0	**1 (%0. 3)**
EDH + ICH	1	1	1	**3 (%1.1)**
SDH + SAH	0	1	0	**1 (%0.3)**
EDH + SDH +SAH+ICH	0	1	0	**1 (%0. 3)**
TOTAL	**138 (%49.5)**	**92 (%33.0)**	**49 (%17.5)**	**279 (%100)**

(EDH: epidural hematoma, SDH: subdural hematoma, SAH: subarachnoidal hemorrhage, ICH: intracerebral hemorrhage, IVH: Intraventricular hemorrhage).

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Table- 6. *Correlation of lesion and surgical operation*

Surgical Operation Lesion	Operation (+)	Operation (-)	TOTAL
Linear fracture	0 (% 0.0)	152 (%100.0)	**152 (%100.0)**
Depressed fracture	25 (%36.2)	44 (% 63.8)	**69 (%100.0)**
Linear fracture + Intracranial lesion	23 (%25.0)	69 (% 75.0)	**92 (%100.0)**
Depressed fracture+ Intracranial lesion	35 (%71.4)	14 (% 28.6)	**49 (%100.0)**
Intracranial lesion	20 (%14.5)	118 (% 85.5)	**138 (%100.0)**
TOTAL	**103 (%20.6)**	**397 (%79.4)**	**500 (%100.0)**

χ^2: 51.15, df: 2, p< 0.001* Only fractures, fractures with intracranial lesion, and only intracranial lesion were grouped for test.*

Of the cases, 152 having only linear fractures had no surgical operation. Of the 69 cases having only depressed fractures, 44 had no surgical operation, whereas 25 underwent surgery. Of the cases that have both linear fractures and intracranial lesions, 69 had a surgical operation whereas 23 underwent surgery. Of the 49 cases having depressed fractures and intracranial lesions, 14 had no surgical operation whereas 35 underwent a surgical intervention. Of the 138 cases having only intracranial lesions, 118 had no surgical operation whereas 20 had surgery (Table-6)

4. Discussion

In this study, we have reviewed 500 cases, each of which had a head injury in a traffic accident. The cases were referred to the council of forensic medicine by the courts of laws for medical report to be prepared between 1998 and 2000. In their radiological examinations, skull fractures and/or brain lesions were examined. The cases consisted of 152 (30.4%) only linear fractures, 69 (13.8%) depressed fractures, 92 (18.4%) linear fractures plus intracranial lesions, 49 (9.8%) depressed fractures plus intracranial lesions and 138 (27.6%) only intracranial lesions. As a result, of the 244 linear fractures and 118 depressed fractures, 92 (37.7%) and 49 (41.5%) respectively were accompanied by intracranial lesions (Table-1)

In this study, there were 377 (75.4%) men and 123 (24.6%) women. Male to female ratio was 3.1/1(Table-1). This ratio is similar to findings of Azmak and his colleagues (10) and Akin O (11) 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 (12) and Tiret and his colleagues (13) 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 (14). In other studies performed in Turkey, male to female ratio is higher since males are more active in daily life (10,11). 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 (Table-1). When we consider that all cases are randomly selected and were exposed to similar to 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 male skull being thicker and stronger and absorbing impact energy.

Mean age was 26.3. Of all the cases, 44.2% (n=221) fall into age group 0-20, and 19.6% (n=98) fall into age group 21-30 (Table-1). These findings were compared to findings of other studies related to blunt head injuries and were found to be similar to Azmak and associates (10) whose mean age 28.4 and 22.7% of cases within age group 20-30, to Haug and associates (15) 54 % within age group 16-30 and to classic literature being concentrated between ages 15 and 24. It differs from report of Akin O (11) 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 (figure-1). 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.

The linear fractures were located to frontal, temporal and parietal bones in decreasing order (Table-2). The depressed fractures are located to frontal, parietal, and temporal bones in decreasing order (Table-3). 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 are 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 fracture 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) (Table 4). These findings show similarity to reports of Colak and associates (16). They have reported in a study,

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about radiological 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 (17) reported rate of multiple fractures in children due to traffic accident was 20/34 (58.8%). Miura and associates (17) 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.

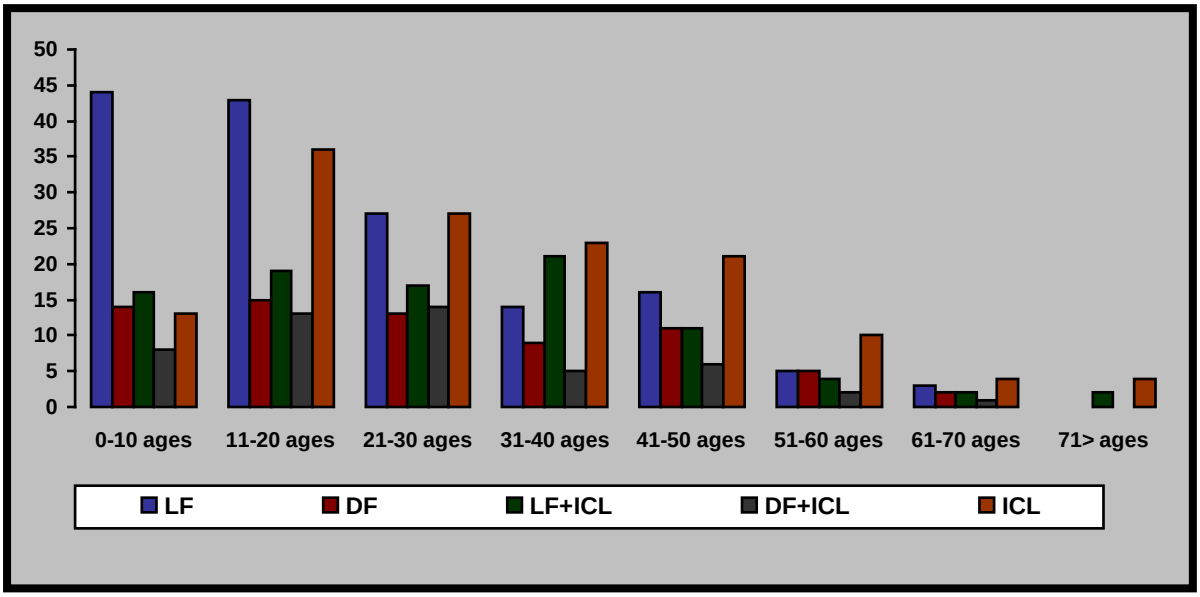

Figure-1. *Percentile of lesions according to age groups. (LF: Linear fracture, DF: Depressed fracture, ICL: Intracranial lesion)*

Of the 279 cases having intracranial lesions, 141 (50.5%) accompanied a skull fracture and 30 (10.7%) had multiple lesions. Comparisons between the existence or type of fractures and one or multiple intracranial lesions were not statistically significant ($p>0.05$) (Table 5).

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 two years of life since dura is firmly attached to inner layer of skull. About 10% of epidural hematomas are accompanied by subdural hemorrhage (2,4,6,19,20). In this study, of 69 cases having epidural hematomas, 51 (73.9%) had accompanying skull fractures. Among cases, having epidural hematomas, the rate of accompanying subdural hematoma was found to be 13.0% (9/69) which is close to reports in the literature (20) (Table-5).

Of subdural hematomas, 24% are due to traffic accidents, which usually present together with other brain lesions such as contusion and lacerations (3,4,9). In this study, intracranial lesion accompanies only 30.5% (18/59) of subdural hematomas and 69.5% (41/59) are alone. This finding is different from reports in the literature (Table-5).

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 (6,20). In this study, 68.2% (15/22) of subarachnoidal hemorrhages were alone, and other intracranial lesions accompanied the remaining cases (Table-5).

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 (4,21,22). In this study, 24.0% (67/279) of cases having intracranial lesions were accompanied by intracerebral hemorrhages and of 67 intracerebral hemorrhages, 3 were accompanied by intraventricular hemorrhages. 2 cases had intraventricular hemorrhages without any intracerebral hemorrhage (Table-5).

Contusions are most common traumatic intracranial lesions and are always caused by trauma (3,6,9,23). Similarly, in this study, of the 279 cases, 72 had contusions and found to be the most common intracranial lesions by 25.8%.

Lacerations either develop 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 (3,4,9,23). In this study, of the 279 cases, 11 (3.8%) had lacerations and all lacerations were accompanied by skull fractures (Table-5).

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 (3,8,24,26).

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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) (Table-5).

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. Miura and associates (18) found surgery rate for depressed fractures to be 69.8%. 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 (χ^2: 51.15, p< 0.001) (Table 6).

In conclusion, 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 male, 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.

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. Likewise, of the 244 cases having linear fractures, 118 cases having depressed fractures, 92 (37.7%), and 49 (41.5%) were accompanied by an intracranial lesion respectively. Of the 362 cases having a skull fracture, 141 (38.9%) 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.

References

1. *Yucel F, Asirdizer M, Cansunar N, Batuk G, Ildiz E. The Deaths Caused Intracranial Complications After Blunt Head Injury. Journal of Forensic Medicine Istanbul. 1996; 12(1): 49-57.*
2. *LaHaye PA, Gade GF, Becker DP. Injury to the Cranium. In: Moore EE, Mattox KL, Feliciano DV, eds. Trauma 2nd edition. Norwalk: Appleton Lange, 1996: 247-8.*
3. *Di Maio DJ, Maio VJM. Forensic Pathology. New York: Elsevier, 1993: 139-69.*
4. *Geisler FH, Skull Fractures. In: Wilkins RH, Rengachary SS, eds. Neurosurgery. USA, Mc Graw Hill, 1996: 2741- 55.*
5. *Tedeschi CG. The Wound: Assessment by Organ Systems Head and Spine. In: Tedeschi CG, Eckert WG, Tedeschi LG, eds. Forensic Medicine. Philadelphia: W.B. Sounders Company, 1977: 29-75.*
6. *Knight B. Forensic Pathology. New York: Oxford University Press, 1997: 171-216.*
7. *Kolusayin RO, Gok S, Soysal Z. Craniocerebral Trauma I: General Principles of Trauma and Anatomical Structure of the Skull and Brain. Journal of Forensic Medicine Istanbul. 1985: 1; 62-73.*
8. *Graham DI. Neuropathology of Head Injury, In: Narayan RK, Wilberger JE, Povlishock JT, eds. Neurotrauma. USA: Mc Graw Hill, 1996, 43-59.*
9. *Mc Cormick WF. Pathology of Closed Head Injury. In Wilkins RH, Rengachary SS, eds. Neurosurgery, USA: Mc Graw Hill, 1996, pp. 2639-66.*
10. *Azmak D, Imer M, Cobanoglu S, Yilmaz A, Ozakbas S, Kulali A, Memis M, Head Injury: The Epidemiological Study of 705 Cases. Journal of Forensic Medicine Istanbul. 1994; 10: 3-10.*
11. *Akin O, The Evaluation of Cranial X-Ray and CT for Head Trauma. Thesis for Specialization at the Section of Forensic Medicine of Medical Faculty of Ataturk University in Turkey. Erzurum, 2000, 49-111.*
12. *Ingebrigsten T, Mortensen K, Romner B. The Epidemiology of Hospital-Referred Head Injury in Northern Norway. Neuroepidemiology. 1998;17:139-46.*
13. *Tiret L, Hausherr E, Thicoipe M, Garros B, Maurette P, Castel JP, Hatton F. The Epidemiology of Head Trauma in Aquitaine (France) 1986: A Community-Based Study of Hospital Admissions and Deaths. Int J Epidemiol. 1990; 19: 133-40.*
14. *Kraus JF, McArthur DL, Silverman TA, Raman MJ, Epidemiologist of Brain Injury, In Narayan RK, Wilberger JE, Povlishock JT, eds. Neurotrauma, USA: Mc Graw Hill, 1996: 13-30.*
15. *Haug RH, Adams JM, Conforti PJ, Likavec MJ. Cranial Fractures Associated with Facial Fractures: A Review of Mechanisms, Type, And Severity of Injury. J Oral Maxillofac Surg. 1994; 52(7): 729-33.*
16. *Colak B, Bicer U, Aydin B, Ildiz E, Kolusayin RO, Altinkok M, Examination of X-ray Grafies in Cranial Trauma Cases From the Forensic Medicine Aspect. Journal of Forensic Medicine Istanbul. 2000; 14: 15-28.*
17. *Nicol JW, Johnstone AJ. Temporal Bone Fractures in Children: A Review 34 Cases. J Accid Emerg Med. 1994;11(4):218-22.*
18. *Miura FK, Plese JP, Ciquini Jr O, Martinez JA, Matushita H, Depressed Skull Fractures in Children Under 2 Years of Age. Retrospective Study of 43 Cases. Arq Neuropsiquatr. 1995; 53(3-B): 644-8 (Abstract).*
19. *Greenberg MS. Handbook of Neurosurgery. Florida: Greenberg Graphics, 1994: 521-69.*
20. *Gordon I, Shapiro HA. Forensic Medicine A Guide to Principles. New York: Churchill Livingstone, 1975: 218-52.*
21. *Samutrala S, Couper PR. Traumatic Intracranial Hematomas. In Wilkins RH, Rengachary SS, eds. Neurosurgery. USA: Mc Graw Hill, 1996: 2797-807.*
22. *Gennarelli TA, Meaney DF, Mechanisms of Primary Head Injury. In Wilkins RH, Rengachary SS, eds. Neurosurgery. USA: Mc Graw Hill, 1996: 2611-21.*
23. *Kolusayin RO, Gok S, Soysal Z. Craniocerebral Trauma III: Lesions of the Brain. Journal of Forensic Medicine Istanbul. 1986;2: 77-92.*
24. *Imajo T. Diffuse Axonal Injury: Its Mechanisms in an Assault Case. Am J Forensic Med. 1996;17: 324-6.*
25. *Yamaki T, Murakami N, Iwamoto Y, Nakagawa Y, Ueda S, Irizawa Y, Komura S, Matsura T. Pathological Study of Diffuse Axonal Injury Patients Who Died Shortly After Impact. Acta Neurochir (Wien). 1992;119: 153-8.*
26. *Kubo S, Kitamura O, Orihara Y, Ogata M, Tokunaga I, Nakasona I. Immunohistochemical Diagnosis and Significance of Forensic Neuropathological Changes. J Med Invest. 1998; 44: 109-19.*

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