

## POST-TRAUMATIC EPILEPSY AND EEG ABNORMALITY IN DEPRESSED FRACTURES

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### SUMMARY :

*Post-traumatic epilepsy (PTE) is the most important problem in patients who are able to return to a normal life after head injuries. Depressed fractures are commonly accepted as risk factors increasing development of PTE. In this study PTE was found in 9 % of 122 patients with depressed fractures and 3 % in 200 patients with closed head injury. PTE was found to significantly increased in patients with complicated depressed fractures ( $p < 0.05$ ). There was a weak relationship between anti-convulsant therapy and PTE development. EEG abnormalities were more common in cases with depressed fractures even in non-convulsive patients ( $p < 0.05$ ).*

### KEY WORDS :

*Depressed fractures, post-traumatic epilepsy, EEG abnormality.*

### INTRODUCTION

It is well known that epilepsies can develop after head injuries. In various reports, post-traumatic epilepsy (PTE) has been reported in a range of 2.3-60 % (2,3,4,5,6,8,12,13,14). PTE occurs within two years after trauma. In general, epilepsies that develop within a week after trauma are called early PTE, whereas others which develop afterwards as late (4,8). Early PTE is commonly accepted as having no clinical significance, but on the other hand, Desai et al. reported that early PTE should be accepted as a sign of poor prognosis (3,4,6,8). It has been also reported that late PTE could develop in 15-25 % of cases with early PTE (7,12).

Age (particularly younger than 5), severity of injury, linear or depressed fracture of skull and intracranial hemorrhage increase the risk of PTE (3).

It is claimed that, particularly in depressed fractures associated with cortical damage or dural tear, to remove depressed fracture early may decrease the incidence of PTE by removing the necrotic cerebral tissue and foreign body which would prevent the production of scar tissue (14).

In general, patients with PTE are followed up with clinical findings and EEG.

In this report, we tried to investigate EEG abnormalities and development of PTE in depressed fractures following head injuries.

### MATERIAL AND METHOD

One hundred and twenty-two cases of depressed fracture and 200 cases of closed head injury (GCS higher than 10) admitted to the Neurosurgery Depart-

ment of Mediterranean University, Medical School, between July 1981 and November 1986 and who were followed for at least two years were studied. EEG was performed in both groups and all depressed fracture cases had been operated. The depressed fractures had been elevated by burr hole or craniectomy. Depressed fractures in association with dural tear and/or cerebral laceration in varying degrees were called complicated, and others were considered simple. In the complicated group, lacerated cerebral tissue was debrided and the dural tear was repaired.

Epilepsies which occurred within the first week of trauma were considered as early and others developing afterwards as late.

Fifty-three patients with depressed fractures were selected randomly for anti-convulsant therapy. Phenobarbital was used in patients younger than 6. The others were administered diphenylhydantoin. Anti-convulsants were used only in case of epilepsy in the closed head injury group.

The first EEG was performed within 3 months following trauma and the last was obtained 2 years later in both the depressed fracture and closed head injury groups.

Of 122 patients with depressed fractures, 94 (77 %) were male and 28 (23 %) female. The age of the patients ranged from 1 to 57 years with a mean of 19.2 years. 24 (19.6 %) patients were younger than 5 years. 54 (44.3 %) patients had complicated depressed fractures and 68 (55.7 %) simple. Depressed fractures were mostly located in parieto-temporal area with a ratio of 65.5 % (80 cases) and there were 3 (24 %) cases who had depressed fracture in the occipital area. (Table I)

107 (87.7 %) patients with depressed fracture were operated within the first 24 hours, and other 15 by the end of the second day after trauma.

PTE was observed in 11 (9 %) patients with depressed fracture. 3 (4.4 %) of them were simple and 8 (14.8 %) complicated. Epilepsies occurred early in 7 and late in 4. They were generalized in 6 and focal in 5. PTE in the complicated group, was found to be significantly increased when compared with the closed head injuries (pr 0.05). But no significant difference was found in PTE development between simple depressed fractures and closed head injuries. Distribution of PTE with respect to location is shown in Table II.

Anti-convulsants were administered to 53 patients selected randomly, with depressed fracture (Table III). In the complicated depressed fracture group, no significant difference was found between patients with and without anti-convulsants.

EEG abnormality was found in 46 patients with depressed fracture. 9 of them had PTE and 37 were nonconvulsive. At the end of the 2 - year follow up period, EEG abnormality persisted in 1 patient with PTE and in 9 nonconvulsive patients. Distribution of EEG abnormality in non-convulsive patients with respect to location is shown in Table IV.

PTE occurred in 6 (3 %) patients with closed head injury. It was generalised in 4 and focal in 2. Late PTE was observed only in one of the patients. EEG abnormality was originally found in 41 (21.1 %) non-convulsive patients and disappeared 2 years later, in all patients. EEG abnormality was found to be statistically significant in patients with depressed fractures, when depressed fractures and closed head injuries were compared in non-convulsive patients (pr 0.05).

## DISCUSSION

PTE development following head injuries has been commonly reported by various authors (5,8,14). But there are only a few reports about the factors increasing risk of PTE. Ransohoff et al. suggested that in depressed fracture associated with cerebral laceration, PTE might develop in 90 % and this is a very high rate (10). Caveness reported PTE as 60 % (2).

Jennet and Levin reported PTE as 12.5 % in depressed fractures and found no significant difference between intact or penetrated dural cases (8), in complicated depressed fractures (5). In our in- and non-operated simple depressed fractures (12). Hahn et al. reported PTE 3 % in simple and 14.3 % in complicated depressed fractures (4). In our investigation PTE occurred in 3 % of closed head injuries, 4.4 % of simple depressed fractures and 14.8 % of complicated cases.

Location of depressed fracture	Simple depressed fracture	Complicated depressed fracture	Total	%
Parieto-temporal	46	34	80	65.5
Frontal	21	18	39	31.9
Occipital	1	2	3	2.4

Table I

Location of depressed fracture	Early PTE	%	Late PTE	%	Total	%
Parieto-temporal	5	7.9	2	3.1	7	11.1
Frontal	2	5.1	2	5.1	4	10.2
Occipital	—	—	—	—	—	—

Table II

Simple depressed fracture	Anti-convulsant	Case	Late PTE	%
	No anti-convulsant	21	1	2.1
Complicated depressed fracture	Anti-convulsant	32	1	3.1
	No anti-convulsant	22	2	9.09

Table III

Location of depressed fracture	Non-convulsive EEG abnormal	%	2 years later	%
	Parieto-temporal	22	30.1	4
Frontal	15	42.8	5	14.2
Occipital	—	—	—	—

Table IV

Depressed fractures were found in 65.5 % in parieto-temporal and occipital had the least depressed fracture, with 2.4 %. In other reports, also, depressed fractures were mostly parietal or parieto-temporal (4,8,13).

In our study, patients with PTE had depressed fractures in frontal and parietal bone. Jennet and Levin found 12.5 % PTE with frontal and 14.3 % with parieto-temporal depressed fractures and reported that they had never seen PTE with occipital depressed fracture(8).

Epilepsies were generalised in 54.5 % and focal in 44.5 % of our cases. Any other form of epilepsy was not observed though atypical and other forms were reported in some articles (5,8,14).

The relationship between improvement of epilepsy and prophylactic anti-convulsant therapy, seems

unconvincing and natural prognosis and abolishment of traumatic effect appears to be much more important (14,15). Hahn et al. advised anticonvulsant therapy for prophylaxis in every case with cerebral edema, acute subdural hematoma, depressed fracture or severe head injury(5). Jennet reported that there was no significant difference with respect to development of late PTE between the groups with and without anti-convulsant drugs(8). Late PTE was observed in 3.1 % of cases with anti-convulsant and 9.09 % without in this study. For this reason, we believe administration of anti-convulsant in penetrated dural injury would be helpful.

EEG abnormality persisted in only one case of PTE after 2 years and was found in 20.5 % of closed head injury and 33.3 % of depressed fractures, even though they were both non-convulsive. Yoshii et al. reported that no significant difference in EEG abnormality was observed between the epileptic and non-epileptic group (15).

Klonof et al. found a positive correlation between en clinical findings and EEG improvement (9). It is assumed that EEG abnormality improves in a period of 7 months to 6 years (5), but also, it is claimed that it might persist in 25 % of cases (14).

In conclusion, anticonvulsant therapy would be helpful in depressed fractures, particularly with penetrated dural injury, because of the increased risk of PTE development. EEG abnormality was found to have no clinical importance even though it was found more often in patients with depressed fractures.

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