

EPILEPSY AFTER SEVERE CRANIO-CEREBRAL TRAUMA

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Posttraumatic epilepsy may be an early or a late consequence of the cranio-cerebral trauma (CCT). About 4 per cent of the patients manifest initial epileptic convulsions already during the first week after CCT in the form of focal, Jackson's or secondarily generalized tonic-clonic fits. Early posttraumatic epilepsy can be complicated by an epi-status [1,2,5-7].

Late posttraumatic epilepsy develops more commonly in cases with severe CCT with long-lasting amnesia and cranial fractures. Most often, its beginning is between one and two years after injury. One observes focal and/or secondarily generalized convulsions [2,4,6,7,9]. Penetrating CCTs cause 10 times more frequently epileptic fits than blunt CCTs [2,3,5,6]. According to Gillingham [8], breach of dura mater integrity enhances the risk of appearance of a late epilepsy in up to 80 percent of the cases. Besides focal neurological and psychic symptomatics focal and secondarily generalized epileptic convulsions are typical of posttraumatic epilepsy [2,5,9]. CCT severity, long-lasting amnesia and coma, presence of impression or depression cranial fractures, intracranial haematoma, alien bodies, cerebral contusion, posttraumatic infection and local ischemia are considered risk factors of posttraumatic epilepsy. Anamnestic data about CCT, neurological symptomatics as well as roentgenological, CT and EEG investigations indicate the etiological mechanism and confirm the diagnosis of posttraumatic epilepsy.

The purpose of the present work is to study the relation between CCT severity and the peculiarities of the manifestation of the epileptic syndrome as well as the data from instrumental examinations and the results from the surgical treatment.

MATERIAL AND METHODS

A total of 32 patients hospitalized in the Clinic of Neurosurgery of the Department of Neurology and Neurosurgery, Higher Institute of Medicine, Varna, on the occasion of severe CCT were covered in this study. Of them, there were 23 males aged between 9 and 64 years and 9 females aged between 16 and 62 years. Patients were divided into two groups: Ist - with a clinical picture of early posttraumatic epilepsy, and IInd - with a picture of late one. The first group consisted of 12 patients but the second one - of 20 patients. An open intracranial haematoma was established in 8 patients while there was an open or blunt cranial fracture in the rest 24 cases. A surgical treatment was carried out in all the patients. Up to 5 years after operation, 20 patients were rehospitalized for a second and even for a third time on the occasion of a late posttraumatic epilepsy.

RESULTS AND DISCUSSION

The group of early posttraumatic epilepsy included 8 male and 4 female patients. There was an open CCT in 7 cases and a blunt one - in 5 cases. First epileptic convulsion was registered immediately after the injury when patients with open CCT were concerned. There was a Jackson's fit in 5 cases and a generalized one - in the rest two cases. First epileptic convulsion was registered in patients with blunt CCT between the 1st and 14th day after injury. Jackson's fit was observed

in two patients and a generalized one - in 3 cases. Epileptic fit frequency prior to operation varied between one and 4 fits. Neurologically, there was a contralateral hemiparesis varying between latent and severely expressed one and accompanied by unilateral mydriasis in 7 cases. By means of instrumental examinations an epidural haematoma was established in 2 cases, an acute subdural haematoma was found out in 5 cases, and impression cranial fractures were seen in 5 cases. CT revealed cerebral contusion foci in 7 patients which were proved by EEG, too. All the patients underwent operation. However, an epileptic symptomatics was registered during the postoperative period. Irritative EEG changes were of diffuse character and both local and focal changes determined by cerebral contusion dominated in the cerebral bioelectric activity when early epileptic convulsions were concerned.

The group of late posttraumatic epilepsy included 15 males and 5 females. There was an open CCT in 13 cases and a blunt one in the rest patients. First epileptic fit was registered between the first week and the third year after injury in patients with open CCT and between the first month and the third year - in patients with blunt CCT. Generalized epileptic fits were observed in 10 patients but Jackson's ones - in three patients only. In all the patients with blunt CCT there were generalized epileptic convulsions. In one patient a chronic subdural haematoma was detected while in the rest 19 cases there was cranial fracture. Of these 19 patients, 15 did not demonstrate any focal neurological symptoms. Only 4 patients had a neurological deficit. A surgical intervention was carried out in all the patients with late posttraumatic epilepsy. During the postoperative period, 13 patients manifested generalized fits. The frequency of fits varied, e.g. it was 1-2 times in a year in 8 patients, 1-2 times in a month in 2 patients, and 2 times weekly in the rest 3 cases. In operated patients with blunt CCT the following symptomatics was registered: in 4 patients there were generalized epileptic fits and in 3 ones - Jackson's fits. In one female patient, 26 years old with a severe blunt CCT in 1973, postoperatively, grand mal of a frequency of 6-7 convulsions in a year was established. CT revealed cortical atrophy and two posttraumatic cysts in the frontal lobe of the brain while pneumoencephalographically, a severely expressed adhesive arachnoiditis could be observed.

EEG demonstrated the presence of specific epileptic changes in all 32 patients. We established on the background of irregular alpha-rhythmus, dysrhythmia, diffuse and localized slow wave changes pathognomonic to an organic cerebral lesion, graphoelements arguing for an increased excitability of cortical epileptic activity in 20 patients. It was manifested by irregular or grouped sharp waves, peaks, complexes of peaks and slow waves coinciding with the topics of contusion focus. In the half of these cases as well as in 8 patients without any focal changes we established a secondary and primary, respectively, bilateral synchronization of pathological graphoelements. EEG demonstrated diffuse irritative changes only in 4 patients.

Our results demonstrate convincingly that open CCT plays a greater role in the etiopathogenesis of posttraumatic epilepsy than blunt CCT does. Initial convulsion symptomatics in patients with early posttraumatic epilepsy occurs between the first and fourth day after injury while it appears between the first month and the third year in cases with late epilepsy. Jackson's convulsions are more common in the first group while generalized fits prevail in the second one. Our results correspond with these reported by other investigators, too [2,5,6,8].

EEG reflects the complex pathomorphological and pathophysiological cerebral reactions to CCT of various severity and in different time intervals. EEG is of special significance in the early and distant periods after CCT as it presents an objective criterion for cerebral status. EEG indicates the degree of restitution and compensation of the brain after injury, it allows to follow-up dynamically focal changes and to detect the predisposition for development of convulsion symptomatics.

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ЭПИЛЕПСИЯ ПОСЛЕ ТЯЖЕЛОЙ ЧЕРЕПНО-МОЗГОВОЙ ТРАВМЫ

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РЕЗЮМЕ

Анализируются 32 случая эпилепсии, наступившей после тяжелой черепно-мозговой травмы - закрытой или открытой. Наблюдаемые пациенты были разделены на две группы. К первой группе были отнесены пациенты с ранней посттравматической эпилепсией, а к второй - пациенты с поздней посттравматической эпилепсией. У восьми пациентов был обнаружен гематом, а в остальных 24 случаях была установлена фрактура черепа без нарушения или с нарушением целости дуры. Все пациенты подверглись операции. Непосредственной зависимости между тяжести травмы и появлением первой эпилептической судороги не было установлено. При всех пациентах были проведены рентгенографическое, электроэнцефалографическое и компьютертомографическое исследования.

В работе обсуждаются корреляции между особенностями клинического проявления эпилептического синдрома после черепно-мозговой травмы, а также данные, полученные в результате инструментального исследования и оперативного лечения.