World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-35, June 2024
ISSN: 2749-3644

DEVELOPING A DIAGNOSTIC COMPLEX FOR THE POST-TRAUMATIC EPILEPSY

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Article history:
Received: March 30th 2024
Accepted: April 26th 2024

Abstract:
The purpose of our study was to study clinical features and improve instrumental diagnostics in patients with post-traumatic epilepsy.

Keywords: with post-traumatic epilepsy, method, treatment, diagnosis, patient.

INTRODUCTION

Post-traumatic epilepsy (PTE) is one of the most common (up to 70% of cases) etiopathogenetic variants of locally caused symptomatic epilepsy. The incidence of PTE exceeds 3 million people annually. Moreover, the incidence of epilepsy in cases of previous TBI is, according to various authors, from 10 to 50%, and the course of PTE is often progressive. About half of patients with PTE have more than 12 seizures per year, and remission of seizures can be achieved only in 5-10% of patients, which causes a high frequency of personality changes. Moreover, the mortality rate of patients with post-traumatic epilepsy exceeds the population rate by 2-3 times.

MATERIALS AND METHODS

Comprehensive clinical-neurological, electroencephalographic (clinical EEG and video-EEG monitoring with a mandatory sleep study), neuroimaging (MRI of the brain according to a special epileptic program with a targeted study of the hippocampus, PET, 1NMRS, MR tractography), A psychological examination was carried out in 341 patients with PTE. In addition, the group also included 15 pairs of mono- and heterozygous twins. All patients suffered a TBI of varying severity before the onset of epilepsy, and the presence of trauma and seizures was confirmed by appropriate medical documentation.

RESULTS AND DISCUSSION

A post-traumatic epileptic seizure (epileptic syndrome) is usually designated as a single epileptic paroxysm that occurs after a head injury in the first 24 hours and does not recur subsequently. The occurrence of an attack in different time periods after injury is not uniform. The frequency of early post-traumatic seizures is 3-5%, late post-traumatic seizures are characterized by a higher incidence - up to 8-9%. For late post-traumatic seizures, they are more likely to recur in the future. Thus, post-traumatic epilepsy is a chronic brain disease that occurs as a result of a TBI and is characterized by repeated unprovoked epileptic seizures. A diagnosis of PTE requires the presence of at least two unprovoked seizures.

The occurrence of an epileptic seizure after TBI does not always imply the formation of a subsequent PTE in this patient. For example, factors that influence the risk of developing post-traumatic seizures may also have a similar influence on the development of PTE. The very fact of having an attack that occurred during the first week after TBI increases the risk of developing PTE by more than 25%.

Until now, the question of the concept of epileptic seizures that occur at the time of injury or in the early period after it, and of PTE as an independent nosological form, remains controversial. Most researchers believe that the development of PTE is possible over the subsequent months and years after TBI, depending on the process of formation of the epileptic focus. The time frame for the formation of PTE can be from 7 days to 2 years after a TBI. The "critical" time of onset of the first epileptic seizure, which determines the development of PTE or the epileptic reaction to acute brain injury, is the seven-day period after TBI.

Clinical and neurological examination of patients with PTE included assessment of predisposition factors (congenital, hereditary and acquired), determination of the type of epileptic seizures and assessment of psychoneurological status.

Epilepsy in patients with a history of mild TBI is an etiologically heterogeneous disease, in which mild trauma is usually only a provoking factor in its occurrence, and the disease itself develops in the vast majority of patients based on an already existing predisposition to the disease. PTE in patients with a history of severe TBI has a clearer cause-and-effect relationship with the presence of injury and may be the

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Volume-35, June 2024
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only predisposition factor in the development of the disease.

The clinical phenomenology of epileptic seizures in patients with PTE with mild TBI in the anamnesis was represented mainly by simple (vegetative-visceral, with impaired mental functions) or complex (automatic) partial seizures. Only in 6.2% of observations the attacks occurred as simple somatomotor and somatosensory attacks. In cases of severe or moderate-severe TBI in the anamnesis of patients with PTE, complex partial ones with secondary generalization and a tendency to a serial course predominated. As for the timing of the onset of the first epileptic seizures after injury, significant variability was observed in patients with a history of mild TBI (from 3 months to 5 years or more). Whereas in cases of more severe brain injuries, the first epileptic seizures developed, as a rule, in the shortest period after the injury or 3-18 months later, which does not contradict the literature data [2]

As for the history of birth trauma, the clinical phenomenology of epileptic seizures and the onset of the disease in the examined twin pairs revealed high heterogeneity. At the same time, a distinctive feature of heterozygous twins is the more frequent occurrence of epilepsy in the younger of the pair only as a result of birth trauma. At the same time, in the eldest of a pair of heterozygous twins, the disease usually debuts after suffering another TBI. The established facts require a deeper and more detailed examination using modern genetic methods. As for monozygotic twins, in our study the incidence of epilepsy in older and younger twins was approximately the same, which is consistent with the literature data.

According to the results of video-EEG monitoring in the group of patients with PTE we examined with a history of moderate and severe TBI, in the vast majority of cases (70.2% of observations) moderate and/or pronounced local changes in the production of epileptiform activity in the form of spike waves and "sharp-slow wave" complexes, while in mild TBI the anamnesis revealed less pronounced local changes. MRI performed according to a special epileptic program in patients with PTE with a history of mild TBI revealed various types of MR changes: expansion of the temporal horn of one of the lateral ventricles, expansion of the fissures of the subarachnoid space, gliotic, atrophic and cystic changes, and also sclerosis of one of the hippocampi [3]

MRI changes in PTE with a history of severe TBI were more pronounced. One typical example is illustrated.

It should be noted that in cases where focal hypometabolism of radionuclide glucose was detected by positron emission tomography (PET), it most often corresponded to the area of registration of the epileptic focus, according to video-EEG monitoring (79.4% of observations). In 1NMRS, metabolic changes were most often detected in the mediobasal temporal localization, corresponding to the etiological variant of the lesion, according to MRI and PET studies (see Fig. 4). MR tractography revealed a significant decrease in fractional anisotropy coefficients in the projection of one of the hippocampi in 47% of cases, which may indicate a violation of the integrity and demyelination in the pathways [4]

CONCLUSION

Thus, patients with PTE with a history of moderate to severe TBI are characterized by a number of features in the indicators of clinical and instrumental studies. Performing video-EEG monitoring, MRI according to a special epileptic program and metabolic neuroimaging using 1NMRS and PET for PTE with a history of mild TBI can significantly increase the information content of diagnostic measures in patients with negative clinical indicators. electroencephalographic and MRI studies. However, the etiological significance of mild brain injury in patients with PTE differs significantly from that in moderate and severe TBI, bringing to the fore a set of predisposition factors that determine the appearance of epileptic seizures and the development of the disease.

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