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CONFERENCE ABSTRACTS

3. EPILEPSY

NEUROPHYSIOLOGIC FEATURES OF EPILEPTIC SYNDROME OF ALCOHOLIC GENESIS

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In the last years, a steady tendency of increasing neurologic manifestations of chronic alcoholic intoxication (CAI) was observed. Epileptic syndrome as an aftereffect of alcohol toxic impact is one of the leading disorders of the nervous system by CAI together with lower extremities polyneuropathy caused by toxic-destructive changes in peripheral nerves. Analysis of the main neurophysiological features of patients with epileptic syndrome and lower extremities polyneuropathy of alcoholic genesis is necessary for high-quality diagnosis and adequate therapy of the mentioned neurologic disorders. We examined 65 men — patients of the neurologic department of the Central Medical-Sanitary Unit № 58 in the city of Severodvinsk. 57 persons (<30 y.o.: 17.5%, 31–55 y.o.: 73.7%, >55 y.o.: 8.8%) had primary generalized epilepsies and the syndrome of alcohol dependence (SAD). 8 patients with symptomatic epilepsy had craniocerebral injuries and repeated epilepsies in their anamneses. The clinical-psychopathologic, neurologic, instrumental (brain EEG, computer tomography CT) and laboratory research was implemented. Patients with SAD did not inherit epilepsy, epiphenomena, craniocerebral injuries and insults in their anamneses, personality disorders of epileptoid type. There were no focal symptoms detected in their neurologic status, but in 75.4% of cases lower extremities neuropathy was detected. In all these patients, EEG detected light diffusive changes, deorhythmia, low bioelectric activity, overlapping of muscular tremor and artefacts. 82.5% of patients had mixed hypotrophic hydrocephalus at CT; in 98.2% — rise in activity of blood serum alanine aminotranspherase, aspartate aminotranspherase, gammaglutamate-ranspherase was fixed (only in 1 patient <30 y.o., borderline transferase values were present). During neurological assessment of 8 persons with symptomatic epilepsy, we found focal symptoms of pyramidal deficiency type in 37.5%, and hemiparesis of I-IV degree in 62.5% of patients. Lower extremities polyneuropathy was detected in 25% of patients. At EEG in all the patients, slow indulatory activity in injured cerebral hemisphere was determined; at CT also in all the cases, cystic-gliosic changes were detected; in 62.5%, blood serum transferase values were higher. Conclusions: 1. Epileptic syndrome of alcoholic genesis is characterized by a set of the following neurophysiological features: — absence of epileptic heredity, craniocerebral injuries and insults and presence of alcoholic anamnesis; — absence of focal and common cerebral symptoms during a clinical examination; — presence of primary paroxysms generalization, fits of being awake; — lower extremities polyneuropathy — as a concomitant pathology; — absence of epileptic and focal activity, low bioelectric activity, cerebral diffusive changes and overlapping of muscular tremor at EEG; — symptoms of mixed hypotrophic hydrocephalus at CT; — higher blood serum transferase activity. 2. For symptomatic epilepsy, alcohol does not represent a causal factor. Steady changes in a clinical picture (focal symptoms) as well as slow indulatory activity at EEG and cystic-gliosic changes at CT indicate that patients like this even without alcohol impact can have epilepsy and so selection of adequate anticonvulsants is necessary. 3. By presence of epileptic activity at EEG, epilepsy genesis may not be alcoholic per se. In such cases, intake of strong drinks may be a provoking factor. 4. Higher blood serum transpherase activity is a biological marker of CAI. 5. Overwhelming majority of persons with epileptic syndrome of alcoholic genesis are at the able-bodied age. 6. Complex examination of patients is necessary for timely detection of pathogenesis and selection of adequate treatment and rehabilitation.