

clinico-toxicological analysis of the ethanol concentration in the blood. The study did not include patients with seizure, severe traumatic brain injuries in the anamnesis. EEG monitoring was performed from the moment of admission and until the restoration of consciousness. If the coma persisted for more than 6 hours, the study was performed after 12 hours and 24 hours. The EEG was recorded on the "Mizar-EEG-201" encephalograph (Ltd "Mizar").

Results: Of the 46 examined patients, five were diagnosed with epileptiform stigmata on the EEG. The convulsive syndrome was not observed in any case. In two cases, a relatively long unconscious state was formed in the outcome of comatose period. In one of these patients, the state of impaired consciousness lasted 76 hours, in the other case 48 hours. When EEG was registered in a clear consciousness, epileptiform activity was not observed in these patients.

Conclusions: In severe acute ethanol poisoning, the formation of a long-term state of disturbed consciousness in the outcome of a comatose period may have the formation of a stagnant determinant with hypersynchronous activity in pathogenesis. NCSE with severe ethanol poisoning is a relatively rare event compared with the clinic of other severe cerebral lesions (severe craniocerebral trauma, disorder of cerebral circulation).

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Absence status epilepticus in the postictal phase of a generalized tonic-clonic seizure as the potential final manifestation of a relapsing remitting genetic epilepsy syndrome

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Background: In 2012 nonconvulsive status epilepticus in the postictal phase of generalized tonic clonic seizures was characterized as being associated with periodic lateralized or generalized periodic discharges in EEG. In the classification of 2015 this category was omitted and among others the relapsing absence status in the elderly introduced.

Methods: We report the case of a 82-year-old woman who presented with recurrent absence status epilepticus in the postictal phase of generalized tonic-clonic seizures.

Results: Around 1946 the patient was diagnosed to suffer from Juvenile Absence Epilepsy and successfully treated with ethosuximide and primidone. After a miscarriage in 1968 seizures relapsed and the syndrome was now classified as Generalized Tonic-Clonic Seizures Alone. Seizure frequency increased to 4-5 per year in the early 90ies and she was advised to take valproic acid. Nevertheless, she stayed on ethosuximide 500 mg and primidone 625 mg. After all she remained seizure free from 1996 till January 2018, when under a medication of ethosuximide 250 mg and primidone 500 mg a series of generalized tonic-clonic seizures occurred followed by a nonconvulsive status epilepticus. Ethosuximide was stopped and levetiracetam was introduced to the therapy. After another series of generalized tonic-clonic seizures levetiracetam was increased to 3000 mg. In May 2018 after another generalized tonic-clonic seizure another nonconvulsive status epilepticus occurred, which was now recognized as absence

status as Relapsing Absence Status in Later Life. Valproate was introduced in the therapy and primidone was tapered off. The patient remained seizure-free for the next seven months at least.

Conclusions: In nonconvulsive status epilepticus in the elderly the diagnosis of relapsing absence status should be considered because valproate seems to be a very effective treatment in this situation. Additionally, this case shows that the features of a genetic epilepsy syndrome may change in the course of time.

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Absence status epilepticus – a case series

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Background: Absence status epilepticus (ASE) is a type of generalized non-convulsive status epilepticus in which continuous or almost continuous generalized spike-wave or polyspike-wave discharges are associated with a varying grade of consciousness impairment and at times with other clinical manifestations such as automatisms or subtle myoclonic, tonic, atonic, or autonomic phenomena. ASE can occur in all genetic generalized epilepsies (GGEs) with absence seizures, above all in juvenile absence epilepsy (JAE). The aim of the study was to present electroencephalographic (EEG) and clinical correlates of 8 cases of ASE.

Methods: EEG and clinical data of patients with ASE were prospectively registered.

Results: All patients (6 women and 2 men) suffered from GGEs; 6 had JAE, 2 had juvenile myoclonic epilepsy. The mean age at onset of epilepsy was 14 years (range 11-19) and the mean age at time of presentation was 41 years (range 28-72). ASE preceded or followed a tonic-clonic seizure in 4 patients. In one patient ASE occurred before the diagnosis of JAE was made, in other cases ASE was provoked. Triggering factors were as follows: antiepileptic drugs (AEDs) withdrawal/tapering in 3, treatment with inappropriate AEDs in 3, infection treated with antibiotics in 1 patient. EEG showed continuous or almost continuous generalized polyspike-wave discharges in 6 patients and bilateral sharp waves/ sharp and slow wave discharges in 2 patients treated with contraindicated drugs (gabapentin, tiagabine, carbamazepine) (figures 1-6). All cases of ASE were treated with iv diazepam or/and valproate with good outcome.

Conclusions: Absence status epilepticus is a rare form of nonconvulsive status epilepticus, in most cases provoked by withdrawal of medication or inappropriate medication. EEG is indispensable in diagnosis of ASE. ASE is usually easily treatable condition.

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Lateralized Periodic Discharges (LPDs) as ictal manifestation of Aphasical Status Epilepticus

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