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 occured before the diagnosis of JAE was made, in other cases ASE was provoked. Triggering factors were as follows: antiepileptic drugs (AEDs) withdrawal/tappering 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 Aphasic Status Epilepticus

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Background: Language disturbances can be usually found in various pathological acute pictures involving the dominant frontal and temporal lobes. Prolonged aphasia as the only manifestation of focal status epilepticus is rarely described and only a few cases have been documented. Several EEG patterns have been associated with Aphasic Status Epilepticus (ASE) including Lateralized Periodic Discharges (LPDs). LPDs pattern is usually correlated with structural lesions of cortical or subcortical areas due to some pathological conditions such as acute stroke, brain tumours, infections, traumas and metabolic diseases. The origin of LPDs is a controversial issue and only a few existing neurophysiological hypotheses address causes and circumstances of LPDs onset and if they represent an ictal or inter-ictal pattern.

Methods: We report two cases of ASE associated with LPDs. Aphasic Status Epilepticus was defined according to Rosenbaum's criteria modified by Grimes & Guberman. All these patients underwent a 21 derivations EEG recording according to the 10-20 international system, 3T Magnetic Resonance Imaging (MRI) of the brain and were tested with Aphasia Rapid Test (ART) to better define aphasia's severity. In addition, a review of the past literature was performed by the search terms "Aphasic Status Epilepticus" and "Lateralized Periodic Discharges" on PubMed. A total of 6 articles were available for further analysis.

Results: We stress the electro-clinical correlation between ASE and Lateralized Periodic Discharges. It has been recently reported that the association between LPDs and seizure is more consistent in the presence of particular LPDs features with an increased seizure risk with higher periodic discharges frequency and "Plus modifier" such as superimposed fast activity. In the previous literature, LPDs have been sometimes associated with ASE but they have not always been marked as ictal pattern even though, in some cases, a clear electro-clinical correlation was described with patient's good clinical response to the anti-seizure therapy.

Conclusions: We highlight the importance of considering focal SE in the differential diagnosis of patients presenting aphasia and how LPDs can represent an ictal EEG pattern with regard to ASE.

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Aphasic Status Epilepticus Revisited

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Background: Prolonged aphasic status epilepticus (ASE) in patients without previous seizures and unknown cerebral lesions is rare, and in many occasions an acute stroke is suspected. Some of these patients may be thrombolised and admitted into stroke units. The aim of the study is to describe electroclinical and neuroimaging characteristics, aetiologies and outcome of patients presenting as de novo ASE.

Methods: We designed an unicentric study including consecutive patients presenting to the Neurology Service with new onset status epilepticus of unknown origin (NORSE) between 2011 to 2018. Final diagnosis was obtained after an acute phase complete work-up and considering the follow-up as an outpatient (minimum one year). Patients with ASE (considering aphasia as the main seizure type) were selected. Aetiology and diagnostic procedures included: video-EEG monitoring, serum and CSF biochemistry, serologies and PCR for neurotropic agents, nonspecific immunological analysis and antineuronal antibodies and onconeuronal antibodies. Necropsic studies were performed in some cases. Neuroimaging studies included ictal SPECT, MRI with a protocol for status epilepticus and FDG-PET.

Results: From 35 patients with NORSE, 16 patients (43%) with ASE were selected. 13 (81%) were women, mean age 70.4 (SD14.5), mean age at ASE onset 66 (SD 15.9), 9 (56%) patients had died. TC scan, done in the first 24 hours, was normal in all patients. MRI done during the first week was normal only in 3 patients (17.5%), in 4 (25%) periictal changes were found. First available EEG was normal or showed minor abnormalities (focal slowing or generalized slowing) in 6 (40%), in 5 patients (31%) seizures were recorded and the rest showed a lateralized periodic pattern. SPECT and/or PET were available in 12 patients and showed focal hypermetabolism or hyperperfusion in 8 (66%). Final aetiologies were symptomatic epilepsy (6), toxic/metabolic (2), amyloid angeitis (2), SMART syndrome (1), infectious encephalitis (1), unknown (2), neurodegenerative disorder (1), autoimmune systemic disease (1). Only 4/16 (25%) responded to corticotherapy. No patient with limbic encephalitis debuted with ASE.

Conclusions: Aphasic status epilepticus is a severe entity in which high suspicion is needed. PET or SPECT studies may be specially helpful in diagnosing this entity.

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The features of status epilepticus in children with progressive myoclonus epilepsy - a single center experience

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Background: Progressive myoclonus epilepsy (PME) is characterized by various epileptic phenotype since PME has heterogeneous etiology. The main feature of PME is neurological devastating and resistant epilepsy. The aim of the study is evaluation of status epilepticus (SE) in children with progressive myoclonus epilepsy (PME).

Methods: The retrospective study included children with PME and SE with prominent motor symptoms, treated in Institute in period from 1998 to 2018. PME was diagnosed by enzyme, genetic and/or histopathology investigations. SE was defined as clinical seizure duration >30 min, and classified according the new classification (Trinka et al. 2015). Evaluated features were: age, type, duration, SE recurrence and response to the treatment.