[a]University G. D'annunzio, "SS Annunziata" Hospital, Chieti, Italy
[b]Catholic University of the Sacred Heart, Rome, Italy

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

Sonia Jaraba Armas[a,b], Jacint Sala[a], Misericordia Veciana[c], Jordi Pedro[c], Àngels Camins[d], Laura Rodríguez Bel[e], Jaume Mora[f], Cristina Gámez[e], Mercè Falip[a]
[a]Epilepsy Unit, Neurology Department, Hospital Universitari de Bellvitge, L'Hospitalet De Llobregat, Barcelona, Spain
[b]Neurology Department, Hospital De Viladecans, Viladecans, Barcelona, Spain
[c]Neurophysiology Unit, Neurology Department, Hospital Universitari de Bellvitge, L'Hospitalet De Llobregat, Barcelona, Spain
[d]Radiology Department, Institut de Diagnòstic per la Imatge (IDI), Centre Bellvitge, Hospital Universitari de Bellvitge, L'Hospitalet De Llobregat, Barcelona, Spain
[e]PET Unit, Institut de Diagnòstic per la Imatge (IDI), Hospital Universitari de Bellvitge-IDIBELL, L'Hospitalet de Llobregat, Barcelona, Spain
[f]Nuclear Medicine Department, Hospital Universitari de Bellvitge - IDIBELL, L'Hospitalet de Llobregat, Barcelona, Spain

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

Ruzica Kravljanac, Biljana Vucetic Tadic
Institute For Mother and Child Healthcare of Serbia, Faculty of Medicine University of Belgrade, Beograd, Serbia

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.