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Short Communication

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Master's Degree in Nursing Case Report: Lennox Gastute.

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Abstract

The Lennox-Gastaut syndrome is the biggest target of current epilepsy research, and it contains almost all the fundamental problems in epilepsy. Highly different terms were given to the relatively broad area of the Lennox-Gastaut syndrome by many schools hitherto. But gradually a general outline of this syndrome has been defined and the name Lennox-Gastaut syndrome has been internationally fixed, leading to the holding of the International Symposium at Bad Kreuznach in 1987. Although the Lennox-Gastaut syndrome is a clinico-electrical entity which is clearly defined both clinically and electroencephalographically, diagnostic boundaries are not always precise in individual cases. According to the clinico-electroencephalographic investigation on typical and atypical cases,

Key Words: lennox-gastaut syndrome; antiepileptic drug; epilepsy;

Introduction

Lennox Gastaut Syndrome (LGS) is an epilepsy syndrome presenting in childhood, classically characterised by a triad of cognitive or developmental impairment, multiple seizure types and EEG features of slow-spike waves (SWW), with or without paroxysmal fast activity (PFA) in sleep. There is increasing scientific opinion in favour of a less rigid approach to LGS diagnosis and this clinical audit attempts to shed light on how the LGS diagnostic criteria used may have changed over time, in a large tertiary paediatric neurology unit

The onset of LGS is typically before 8 years of age, with a peak at 4–5 years [1] 1–2 years from seizure onset to a definite diagnosis of LGS [2] mong the identified 42 cases of late-onset LGS, 13 showed chromosome variations. Of the 29 remaining cases, 17 showed normal cognition and had a disease course of 5–32 years, while 12 showed cognitive impairment (mild cognitive impairment in two cases) and a course of 5–25 years. This indicates that unlike early-onset LGS, cognitive function, and daily viability are relatively preserved in late-onset LGS; additionally, cognitive function is not significantly related to disease course [3.4.5]

This is a 6 year old right handed patient with seizure disorder who was admitted for a surgical assessment. He was born after C/S with significant problem. He was diagnosed with seizure at age 5 years old.

The first seizure was consisted of falling and loss of consciousness followed by tonic of limbs(most right hand). The patient was medicated with anti epileptic medication. The habitual seizure consisted of staring and falling and tonic of limbs. There were no generalized tonic clonic seizure. The frequency of his seizure is many attack / day.

At present he is on Oxcarbamazepin 300 mg/d, Depakin 1000 mg/d, Pergabalin 300 mg/d

Possible Risk Factors

He was born with C/S with significant problem. He became hyperglycemia. No history of head trauma, CNS infection and encephalitis and meningitis. Developmental milestones were abnormal.

Social History

He is mental retard case.

Family History

There is no history of seizure disorder or any specific neurological disease.

Impression

This is a 6 year old right handed patient with history of seizure disorder who was admitted for pre surgical assessment considering medically intractable seizure.

Current Medications / Allergies

Antiepileptic: Oxcarbamazepin 300 mg/d

Antiepileptic: Depakin 1000 mg/d

Antiepileptic: Pergabalin 300 mg/d

Positive Findings on Physical and Neurological Examination

On exam, He is an oriented and alert boy who speaks fluency with no apparent dysfunction in other aspect of language function. The cranial nerve exam showed no abnormalities including in his visual field. Motor exam showed power 5/5 in distal and proximal muscle of upper and lower extremities. Fine repetitive movements are symmetric in his distal muscle. DTR is a symmetric 2+ in upper and lower limbs. The cerebral function tests are negative. On general exam no abnormalities was found in cardiovascular system and no carotid bruit.

MRI

Report:

In respect to patient's history, dedicated sequences were obtained. Focal gliosis is present in the parasagital aspect of bilateral parietooccipital lobe which are associated with volume loss thinning in the posterior aspect of corpus callosum.

Also atrophy of spelenium is seen which has CSF spaces.

It is related to a remote probably prenatal ischemic injury such as PVL.

In the let side it has extension to left temporal lobe border also.

However, both hippocampai are normal.

Other focal structure abnormality compatible with patient seizure is not seen.

The rest of supratentontorial structures is normal.

The sella and pituitary and parasellar structures are unremarkable.

The cerebellopontine angles, brain stem, cerebellar hemisphere, petromastoid

regions and orbits are unremarkable.

EEG:

EEG started with attenuation background then followed by bilateral fast activity in continuation there were bilateral slow activity.

Course In Hospital

On admission, he was on Oxcarbamazepin 300 mg/d, Depakin 1000 mg/d , Pergabalin 300 mg/d.

To bring on his seizures, his antiepileptic medications were cut down. During three days prolonged day and night EEG-video monitoring, many clinical and electrographic were recorded. Clinical manifestation consisted of staring and posture in right hand, unresponsiveness without generalized tonic clonic seizure.

EEG consisted of attenuation o9f background and followed by bilateral fast activity in Continuation

MRI showed Focal gliosis is in the parasagital aspect of bilateral parietooccipital lobe which are associated with volume loss thinning in the posterior aspect of corpus callosum.

Also atrophy of spelenium is seen which has CSF spaces.

Conclusions And Recommendations

This is a 6 year old right handed patient with history of seizure and medically intractable epilepsy.

During stay in hospital the patient had many attacks. Clinical manifestation consisted of staring, tonic of right hand.

Interictal abnormalities consisted of bilateral slow spike most posterior.

Epileptogenic zone is in bilateral maximum posterior head region.

At present he is Lennox gastute but not candidate for epilepsy surgery.

I recommended increasing dosage of Depakin and starting Keppra.

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