Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 9, Issue, 02, pp.46708-46709, February, 2017

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

ISSN: 0975-833X

CASE STUDY

Unusual complication of levetiracetam – a case report from a teritary teaching hospital

***Leny Thomas Mathew, Suresh Kumar and Stephen Abraham Suresh Kumar, S.**

Department of Neurology and Neuroscience Center, Sree Balaji Medical College and Hospitals, Chromepet, Chennai, Tamilnadu, India

ARTICLE INFO

Article History:
Received 18th November, 2016
Received in revised form
08th December, 2016
Accepted 02nd January, 2017
Published online 28th February, 2017

Key words:
Levetiracetam (LEV)

ABSTRACT

Levetiracetam (LEV) is a broad spectrum anti-epileptic drug with renal elimination and no hepatic metabolism. LEV is a relatively well-tolerated both in adults and children. Various behavioral and psychiatric side-effects associated with LEV is been reported. Encephalopathy can occur concomitant with levetiracetam accumulation in a patient with chronic renal failure and also when administered along with valproate. This condition like encephalopathy and psychosis are reversible after down-titration of levetiracetam with no change of the renal function. Encephalopathy resulting from LEV administration without renal dysfunction is a rare occurrence. The knowledge of these facts hold an important role in clinical practise. We hereby present two case reports enlightening the complication of LEV in different clinical scenario.

Citation: Leny Thomas Mathew, Suresh Kumar and Stephen Abraham Suresh Kumar, S. 2017. "Unusual complication of levetiracetam – a case report from a teritary teaching hospital", *International Journal of Current Research*, 09, (02), 46708-46709.

INTRODUCTION

Levetiracetam (LEV) is a relatively well-tolerated antiepileptic drug (AED) in both adults and children. The behavioral and psychiatric side-effects associated with LEV include irritability, nervousness, hostility, anxiety and depression. (Cereghino *et al*., 2000; Ben-Menachem and Falter, 2000) Encephalopathy resulting from LEV administration is a rare occurrence and has been reported previously in a patient with renal failure (Vulliemoz *et al*., 2009) and in another patient when the drug was added to valproate. (Bauer, 2008) Here we present two case reports enlightening the complication of LEV in different clinical scenario.

Case 1: A 63 year old right handed male presented to our hospital with breakthrough seizures after having two late nights. He is a known case of seizure disorder for past 26 years, hypothyroidism for 15 years and hypertension for 10 years. Initially treated with Phenytoin 300 mg OD and later on tapered off. He had seizure episode in 2000 and was put on T. Oxcarbazepine 600 mg BD. In view of recurrent episodes once in 3 months despite AED, adjuvant therapy of T. Levetiracetam 500 mg BD and T. Sodium valproate 300 mg BD was started. During this first admission he needed intravenous levetiracetam and sodium valparoate. Oxcarbazepine were continued. At time of discharge patient's treatment was stepped up from Levetiracetam 500 mg BD to 1000 mg BD, Sodium valproate 500 mg BD to 500 mg TDS and Oxcarbazepine 600 mg BD. He was well controlled on 500mg Sodium Valparoate TDS, Levetiracetam 1000 mg BD, Oxcarbazepine 600 mg BD. He needed ventilatory support for 24 hours in view aspiration. Subsequently over a week the patient settled and had no further seizures and walked back home. After 10 days he presented with difficulty in walking–was unsteady jumpy, jerky movements in both hands, body and confused sensorium with areas of lucent talk for 7 days. On examination vitals were normal. CNS examination patient was conscious, not oriented to time, place or person. Speech was normal and no cranial nerve abnormalities was noted. Bulk, tone, power and reflex of muscles were normal. Myoclonic jerky movements of hands and body was noted. There was masked facies andbradykinessia. There was no sensory deficits. Bladder & bowel were normal. No meningeal signs. Skull and spine normal. Basic laboratories showed normal renal, liver, thyroid function and complete blood count tests. Serum electrolyte levels (sodium, potassium, calcium and magnesium) were within normal limits. Serum ammonia level was elevated (115µg/dL). Serum Valproic Acid Level: Normal (98.4 ug/mL) (50-100), Serum Levetiracetam Level: 57.7 mg/L (10-40), Serum Carbamazepine level: Normal (1.25 ug/mL) (4-12 ug/mL). EEG showed diffuse non-specific cerebral slowing and MRI Brain was normal.

T. Sodium Valproate tapered and stopped, serum ammonia

**Corresponding author: Leny Thomas Mathew,*

jerking while walking or doing activity, none during sleep. He had parkisonian features (Masked facies and bradykinesia) for which syndopa was added. We still felt it was drug induced and tapered LEVETIRACETAM and stopped. 3 days after stopping he was symptomatically better. Myoclonus had reduced significantly, gait was better. T.Oxcarbamezepine was decreased to 450 mg BD. T.Clobazam 10 mg HS was added in view of rapid drug taper. T.Lamotrigine was intially started with 25 mg OD and gradually stepped up to 100 mg BD . 2 weeks post discharge patient had no myoclonus and gait disturbances. Thereby Syndopa was stopped.

Case 2: A 62 year old male presented with complaints ofunsteadiness of gait and giddiness of 7days duration, Frequent myoclonus while in bed and at night which were never there earlier. Hallucinations and irrelevant talks were also noticed. He was a known case of Type 2 Diabetes mellitus and Seizure disorder For 5 years and last episode of seizure being 4 years back. There was no slurring of speech, no motor deficit, no cerebellar signs, no history of trauma, fever, headache, vomiting and diarrhea. Patient was on T.Levetiracetam 1 g BD, T. Sodium Valproate 600 mg BD, T. Clobazam 10 mg BD and T.Gliclizide 80 mg BD for the past 4 years. There was no relevant family history and had no addictions.

On examination vitals were normal. CNS examination patient was conscious, not oriented to time, place or person. Speech was normal and no cranial nerve abnormalities was noted. Bulk, tone, power and reflex of muscles were normal. Myoclonic movements were noticed while simply sitiing, walking and sleeping. There was no sensory deficits. Bladder & bowel were normal. No meningeal signs. Skull and spine normal.Basic laboratories showed normal liver, thyroid function and complete blood count tests. Serum urea was slightly elevated (65 mg/dL). Serum electrolyte levels (sodium, potassium, calcium and magnesium) were within normal limits. Serum ammonia were mildly elevated (97 µg/dL). Fasting Lipid Profile were normal. Serum Valproic Acid level was 45.5 $_{u}$g/mL (50-100). Serum Levetiracetam level was 53.1 mg/L (10-40). EEG and MRI Brain with diffusion was normal. Levetiracetam was tapered and stopped. Patient symptomatically improved, no halluciantions, myoclonus or confused talk. Patient was discharged on T. Sodium Valproate 600 mg BD, T. Clobazam 10 mg BD, T. Gliclizide 80 mg BD.

Points of interest:

1) Encephalopathy with or without myoclonus can be a manifestation of Levetricetam toxicity.
2) Precipitation of Hyperammonemia can occur when Levetiracetam is added with Sodium valproate.
3) Cautious dosing of Levetiracetam in cases of renal dysfunction.
4) Parkinsonium features like Tremors ± myoclonus can be a manisfestation of Levetricetam toxicity.

Conflict of Interests

The authors declare that there is no conflict of interest.

REFERANCES

Bauer J. 2008. Encephalopathy induced by levetiracetam added to valproate. *ActaNeurol Scand*., 117:374–6. (PubMed)

Ben-Menachem E, Falter U. 2000. European Levetiracetam Study Group. Efficacy and tolerability of levetiracetam 3000 mg/d in patients with refractory partial seizures: A multicenter, double-blind, responder-selected study evaluating monotherapy. Epilepsia. 41:1276–83. (PubMed)

Cereghino JJ, Biton V, Abou-Khalil B, Dreifuss F, Gauer LJ, Leppik I. 2000. Levetiracetam for partial seizures: Results of a double-blind, randomized clinical trial. *Neurology*, 55:236–42. (PubMed)

Vulliemoz S, Iwanowski P, Landis T, Jallon P. 2009. Levetiracetam accumulation in renal failure causing myoclonic encephalopathy with triphasic waves. *Seizure*, 18:376–8. (PubMed)

****