A study efficacy of pyridoxine in early-onset idiopathic intractable seizures in pediatric patient at tertiary health care center

Jagadish N Bhadbhade

1Assistant Professor, Department of Paediatrics, Vedaantaa Institute of Medical Sciences, At Saswand, Dhundalwadi, Talk. Dahanu, Dist. Palghar 401602, Maharashtra, INDIA.
Email: jagadish_bhadbhade@yahoo.co.in

Abstract

Background: Pyridoxine is used for management of seizure disorder in three settings viz., seizures that respond to pyridoxine and require life-long supplementation with therapeutic doses of pyridoxine, pyridoxine dependent seizures

Aims and Objectives: study Efficacy of Pyridoxine in Early-Onset Idiopathic Intractable Seizures in pediatric patient at tertiary health care center.

Methodology: This was a cross-sectional study carried out in the patients of Early-Onset Idiopathic Intractable Seizures in pediatric patient during the one year period i.e. June 2017 to June 2018, there were 64 pediatric patients with Idiopathic Intractable Seizures were enrolled for the study. All details of the patients like Age, sex etc. was noted. All the patients were undergone EEG testing. Out of 64 patients Group A received 30 mg/kg/day pyridoxine with 4 mg/kg/day of oral prednisolone Group B received 4 mg/kg/day of oral prednisolone and 14, 15 randomly. The outcome of patients like episodes of seizure etc. was noted after 1 month of treatment. The statistical analysis was done by Chi-square test and analyzed by SPSS 19 version software. Result: In our study the average age in both the group was comparable with each other i.e. 3 ±1.21Yrs. and 3.52±1.31 Yrs. (p>0.05, t=1.12,df=62). The ratio of Male and Female was 2.9:1 and 1.90: 1 was comparable with each other (X2=0.29, df=1,p>0.05). Complete seizure freedom was found in 34.38% and 9.38%; >50% seizure reduction but not complete cessation in 40.63% and 28.13%; <50% seizure reduction in 25.00% and 62.50% respectively in Group A and Group B which was statistically significant (X2=10.44, df=2,p<0.005). EEG findings Normal in 37.50% and 12.50%; Decreased Epileptiform discharges in 46.88% and 31.25%; Persistent Epileptiform discharges in 15.63% and 56.25% respectively in Group A and Group B this observed difference was statistically significant (X2=12.35, df=2,p<0.002) Conclusion: It can be concluded from our study that the patients who received the pyridoxine treatment improved much as compared to not received so the role of pyridoxine in the treatment of intractable seizure should not be underestimated.

Key Word: Pyridoxine, Early-Onset Idiopathic Intractable Seizures, EEG

INTRODUCTION

Pyridoxine is used for management of seizure disorder in three settings viz., seizures that respond to pyridoxine and require life-long supplementation with therapeutic doses of pyridoxine, pyridoxine dependent seizures (PDS, MIM 266100); early-onset seizures responsive to pyridoxine but not requiring life-long pyridoxine supplementation, pyridoxine-responsive seizures (PRS); and, high-dose pyridoxine for the treatment of major seizure disorders of young children e.g., West syndrome1. It has been suggested that pyridoxine dependency is often underdiagnosed, both because of occasional atypical presentation2, and infrequent use of pyridoxine in
Intractable seizures of early onset in infants and children in India. So in our study we have studied efficacy of Pyridoxine in Early-Onset Idiopathic Intractable Seizures in pediatric patient at tertiary health care center.

METHODOLOGY
This was a cross-sectional study carried out in the patients of Early-Onset Idiopathic Intractable Seizures in pediatric patient during the one year period i.e. June 2017 to June 2018, there were 64 pediatric patients with Idiopathic Intractable Seizures were enrolled for the study. All details of the patients like Age, sex etc. was noted. All the patients were undergone EEG testing. Out of 64 patients Group A received 30 mg/kg/day pyridoxine with 4 mg/kg/day of oral prednisolone Group B received 4 mg/kg/day of oral prednisolone and randomly. The outcome of patients like episodes of seizure etc. was noted after 1 month of treatment. The statistical analysis was done by Chi-square test and analyzed by SPSS 19 version software.

RESULT

Table 1: Distribution of the patients in two groups with respect to age and sex

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Group A (n=32)</th>
<th>Group B (n=32)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average age (Yrs.)</td>
<td>3 ± 1.21</td>
<td>3.52 ± 1.31</td>
<td>p&gt;0.05</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td>t=1.12, df=62</td>
</tr>
<tr>
<td>Male</td>
<td>23</td>
<td>21</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>9</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>(X²=0.29, df=1, p&gt;0.05)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The average age in both the group was comparable with each other i.e. 3 ±1.21Yrs. and 3.52±1.31 Yrs. (p>0.05, t=1.12,df=62). The ratio of Male and Female was 2.9: 1 and 1.90: 1 was comparable with each other (X²=0.29, df=1,p>0.05).

Table 2: Distribution of the patients as per the treatment outcome

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Group A (n=32)</th>
<th>Group B (n=32)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete seizure freedom</td>
<td>11 (34.38)</td>
<td>3 (9.38)</td>
<td></td>
</tr>
<tr>
<td>&gt;50% seizure reduction but not complete cessation</td>
<td>13 (40.63)</td>
<td>9 (28.13)</td>
<td></td>
</tr>
<tr>
<td>&lt;50% seizure reduction</td>
<td>8 (25.00)</td>
<td>20 (62.50)</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>32 (100.00)</td>
<td>32 (100.00)</td>
<td></td>
</tr>
</tbody>
</table>

(X²=10.44, df=2, p<0.005)

Complete seizure freedom was found in 34.38% and 9.38%; >50% seizure reduction but not complete cessation in 40.63% and 28.13%; <50% seizure reduction in 25.00% and 62.50% respectively in Group A and Group B which was statistically significant (X²=10.44, df=2, p<0.005)

Table 3: Distribution of the patients as per the EEG findings

<table>
<thead>
<tr>
<th>EEG findings</th>
<th>Group A (n=32)</th>
<th>Group B (n=32)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>EEG Normal</td>
<td>12 (37.50)</td>
<td>4 (12.50)</td>
<td></td>
</tr>
<tr>
<td>Decreased Epileptiform discharges</td>
<td>15 (46.88)</td>
<td>10 (31.25)</td>
<td></td>
</tr>
<tr>
<td>Persistent Epileptiform discharges</td>
<td>5 (15.63)</td>
<td>18 (56.25)</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>32 (100.00)</td>
<td>32 (100.00)</td>
<td></td>
</tr>
</tbody>
</table>

(X²=12.35, df=2, p<0.002)

EEG findings Normal in 37.50% and 12.50%, Decreased Epileptiform discharges in 46.88% and 31.25%, Persistent Epileptiform discharges in 15.63% and 56.25% respectively in Group A and Group B this observed difference was statistically significant (X²=12.35, df=2, p<0.002)

DISCUSSION
Epilepsy accounts for about 1% of global disease burden. Since the report of Spies et al in 1940, 6 several studies regarding the use of PN in the treatment of epilepsy have been reported. This pyridoxine (Vitamin B6) deficiency seizures condition was first described in 1954 by Hunt et al., as responsive to an intravenous multivitamin. The point prevalence of pyridoxine (Vitamin B6) deficiency seizures in the UK is 1/687000 and birth incidence is 1/783,000 population, and approximately only 100 such cases have been reported so far in literature.

The value of pyridoxine (PN) in the treatment of epilepsy cannot be overemphasised. Since the report of Spies et al in 1940, 6 several studies regarding the use of PN in the treatment of epilepsy have been reported. This pyridoxine (Vitamin B6) deficiency seizures condition was first described in 1954 by Hunt et al., as responsive to an intravenous multivitamin. The point prevalence of pyridoxine (Vitamin B6) deficiency seizures in the UK is 1/687000 and birth incidence is 1/783,000 population, and approximately only 100 such cases have been reported so far in literature.

Pyridoxine-dependent

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seizures are a group of extremely rare autosomal recessive disorder which is a typical example of metabolic epilepsy. These seizures occur despite normal Vitamin B6 level due to defective binding of pyridoxine to its apoenzyme which converts glutamic acid to GABA. Therefore, GABA levels are very much reduced causing very much lowered seizure threshold.8 Seizures can start prenatally, natally, postnatally, or neonatal period and typically resistant to antiepileptic drugs. The underlying genetic defect has been identified as a mutation in ALDH7A1 causing deficiency of alpha-aminoadipic semialdehyde dehydrogenase. This is involved in cerebral lysine catabolism.8 There are atypical types of pyridoxine-dependent epilepsies which may first respond to anticonvulsants but relapse later, seizures not controlled by pyridoxine initially but which respond later.9 In our study The average age in both the group was comparable with each other i.e. 3 ±1.21 Yrs. and 3.52±1.31 Yrs. (p>0.05, t=1.12,df=62). The ratio of Male and Female was 2.9:1 and 1.90:1 was comparable with each other (X²=0.29,df=1,p>0.05). Complete seizure freedom was found in 34.38% and 9.38%; >50% seizure reduction but not complete cessation in 40.63% and 28.13%; <50% seizure reduction in 25.00% and 62.50% respectively in Group A and Group B which was statistically significant (X²=10.44, df=2,p<0.005) EEG findings Normal in 37.50% and 12.50%, Decreased Epileptiform discharges in 46.88% and 31.25%, Persistent Epileptiform discharges in 15.63% and 56.25% respectively in Group A and Group B this observed difference was statistically significant (X²=12.35, df=2, p<0.002)

CONCLUSION
It can be concluded from our study that the patients who received the pyridoxine treatment improved much as compared to not received so the role of pyridoxine in the treatment of intractable seizure should not be underestimated.

REFERENCES

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Conflict of Interest: None Declared