Original Research

SOMATOSENSORY EVOKED POTENTIAL IN CHILDREN WITH BETA THALASSEMIA

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ABSTRACT

Background and Aim: Aim was to study somatosensory evoked potential in thalassemia children and effect of iron overload on somatosensory evoked potential.

Methods: 30 children with thalassemia on regular transfusions and iron chelation therapy and 30 healthy age and sex matched controls were subjected to somatosensory evoked potential. Statistical analysis used: means of quantitative variables were calculated in two groups and compared with student t- test. A p-value of <0.05 was taken as significant.

Results: On comparing the results between cases and controls, there was no significant difference in the latency of wave N9, N11 & P11 (p>0.05) and on comparing results of relation between serum ferritin level and somatosensory evoked potential in two groups of cases (group I with serum ferritin level <1000 ng/ml and group II with serum ferritin level >1000ng/ml), we found that serum ferritin level was associated significantly with greater latency in N9, N11 and P11 waves in group II of cases. No studies are available to support this data. This study was unique in the sense that there is relatively low awareness of this manifestation due to a very few studies in Indian population.

Keywords: Deferasirox, Ferritin, Somatosensory Evoked Potential, Thalassemia.

INTRODUCTION

Thalassemia is the most common genetic blood disease in the world and varies in different population group in the world.[1] Beta-thalassemia is an inherited disorder of hemoglobin characterized by an absence or reduced synthesis of beta globin chain. The net result is an excess of alpha chains, which precipitate and destroy the red cell precursors, leading to anemia, skeletal changes, splenomegaly and numerous other complications. There are two clinically important presentations of beta thalassemia. Homozygous beta thalassemia usually results in thalassemia major, a severe anemia which requires regular blood transfusion and iron chelation therapy from early infancy for survival. A milder form of this disorder exists called thalassemia intermedia, which encompasses a wide spectrum of clinical severity, ranging from transfusion independency to the occasional need for transfusion.

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Thalassemia intermedia may result from a variety of molecular processes, but its milder clinical presentation is usually due to a less marked, imbalance of the alpha:beta globin chain ratio.[2] The combination of transfusion and chelation therapy has dramatically extended the life expectancy of these patients, thus transforming thalassemia from a rapidly fatal disease of childhood to a chronic illness compatible with a prolonged life.[3] On the other hand, frequent blood transfusions leading to iron overload and chronic nature of the disease have contributed to a whole new spectrum of complications in patients suffering from thalassemia major.[4,5] Heart failure, arrhythmias, osteoporosis, bone pain, and bone changes, bile stone formation, increased risk of viral hepatitis, cirrhosis, delayed puberty, growth retardation, developmental delay, diabetes mellitus and hypothyroidism are the common complications.[6] Over the year several reports have demonstrated involvement of nervous system in beta thalassemia patients.[7] Neurological complications have been attributed to various factors such as chronic hypoxia, bone marrow expansion, iron overload and desferrioxamine neurotoxicity. In most cases neurological involvement does not initially present with relevant signs and symptoms (i.e., subclinical) and can only be detected during neurophysiological or neuroimaging evaluation.[8] There is relatively low

awareness of this manifestation due to a very few studies in Indian population. Hence, the need for this study. This study was planned to detect subclinical lesion in beta thalassemia patients by neurophysiological evaluation, enabling early detection of neural pathway impairment and allowing for appropriate management, in order to achieve a better life quality for this patient group.

METHODS

This study was cross sectional observational study conducted in a tertiary teaching hospital done over a period of one year from 1st July 2015 to 30th June 2016. The inclusion criteria for the case group was to include all beta thalassemia patients clinically diagnosed and confirmed by Hb electrophoresis with age >5 years of age. All thalassemic patients with preexisting neurological disease or congenital malformation and with vision and hearing problems were excluded. Age and sex matched healthy children attending pediatric OPD were included as control group. As per the inclusion criteria 30 thalassemic children were included in the case group and 30 age and sex matched children comprised the control group. Informed consent was taken from the parents or guardians of the beta thalassemia patients and the control group included in the study. All cases and control subjects were assessed clinically and were subjected to investigations like complete haemogram, serum ferritin levels, liver function test, renal function test, blood sugar level, and vision and hearing evaluation. All cases and control subjects were evaluated for somatosensory evoked potential. Somatosensory evoked potential was done by using MEB 2300 A 12 channel EMG machine of NIHON KOHDEN enterprises from Japan. Electrodes will be placed on CPc(between C3 and P3) , Fz (on forehead above nasion), C5S(5th spinous process) , EPi (ipsilateral Erb's point), REF(contralateral Erb's point). Following are the SEP settings-sensitivity: 10 microV/div, Hi-Cut Filter: 3 kHz, Low-Cut Filter: 20Hz, Analysis time: 3ms/div, Frequency: 5Hz, Stimulation: 0.2ms, Average count: 500. Latency was recorded.

Statistical Analysis

We analysed the data using SPSS software for windows. We calculated means, standard deviation of quantitative variables in both the groups, i.e., cases and controls. We also calculated means and standard deviation of quantitative variables in two groups of cases, i.e., group I (serum ferritin level <1000 ng/ml) and group II (serum ferritin level >1000ng/ml). We compared the means by the student t-test as relevant to the given statistical situation. A p-value of <0.05 was treated as statistically significant.

RESULTS

In the present study, analysis was done on 30 thalassemia patients and 30 age and sex matched controls aged >5 years with mean age of 12.43 yrs (SD 5.19). Both cases and controls consisted of 20 (66.7%) males and 10 (33.3%) females, with a male to female ratio of 2:1. The haemoglobin values in cases ranged between 9.0-10.8 (mean-9.88, SD-0.55). Patients were transfused with packed red blood cells at intervals of 3-4 weeks with the goal being to maintain a hemoglobin level of >9g/dl as per the departmental protocol. The serum ferritin level in cases was less than 1000 ng/ml in 8 patients (26.7%), between 1000-2000ng/ml in 20 patients (66.7%) and more than 2000 ng/ml in 2 patients (6.7%). (Mean-1373 with SD-474.48). These patients also received deferasirox at a dose ranging between 20mg/kg/d to 40mg/kg/d depending on serum ferritin level i.e. >1000ng/ml. It was given once daily on an empty stomach as per protocol of the department. 15 patients (50%) received deferasirox @ 20-30mg/kg/day, 8 patients (26.7%) received deferasirox @30-40mg/kg/day and 6 patients were not on deferasirox as the serum ferritin level was less than 1000ng/ml. In SSEP, on comparing the results between cases and controls, there was no significant difference in the latency of wave N9, N11 & P11 (p>0.05). Latency of N9 wave in cases was 7.78 (SD-1.49) and control was 7.68 (SD-1.49) which was not significant. Latency of N11 wave in cases was 10.31 (SD-1.75) and in control was 10.22(SD-1.76) which was also not significant. In P11 wave, latency in case was 9.29 (SD-0.31) and in control was 9.20 (SD-0.21) which was also not significant. (TABLE 1). Serum ferritin level was associated significantly with greater latency in N9, N11 and P11 waves of group II (serum ferritin level >1000ng/ml) of cases on comparing with group I (serum ferritin level <1000 ng/ml) of cases. In N9 wave, latency increased from 6.84ms (SD-0.80) in group I to 8.12ms (SD-1.55) in group II which was significant (p<0.05). In N11 wave, latency increased from 9.14ms (SD-1.48) in group I to 10.74ms (SD-1.67) in group II which was also significant (p<0.05). In P11 wave, latency increased from 9.20ms (SD-0.12) in group I to 9.33ms (SD-0.23) in group II which was also significant (p<0.05) (TABLE 2).

DISCUSSION

The present study was conducted on children with thalassemia between 5-18 yrs of age, with a mean age of 12.43 yrs. On comparing the results between cases and controls, there was no significant difference in the latency of wave N9, N11 & P11 (p>0.05). Latency of N9 wave in cases was 7.78 (SD-1.49) and control was 7.68 (SD-1.49) which was not significant. Latency of N11 wave in cases was 10.31 (SD-1.75) and in control was 10.22(SD-1.76) which was also not significant. In P11 wave, latency in case was 9.29 (SD-0.31) and in control was 9.20(SD-0.21) which was also not significant whereas Wong et al. describe abnormal

somatosensory evoked potential findings in 4 out of 34 study cases (11.76%), two of which were complicated by diabetes mellitus, a fact significantly correlating to somatosensory neural pathway impairment[9]. Zafeiriou et al in 1998, reported abnormal somatosensory evoked potential in 7.5% of thalassemia patients and was mainly attributed to deferoxamine toxicity[10]. This difference could be attributed to the fact that the patients in these studies received deferoxamine as chelation therapy where as our patients received deferasirox. In our study, on comparing results of relation between serum ferritin level and somatosensory evoked potential in two groups of cases**,** we found that serum ferritin level was associated significantly with greater latency in N9, N11 and P11 waves in group II of cases. In N9 wave, latency increased from 6.84ms (SD-0.80) in group I to 8.12ms (SD-1.55) in group II which was significant ($p<0.05$). In N11 wave, latency increased from 9.14ms (SD-1.48) in group I to 10.74ms (SD-1.67) in group II which was also significant ($p<0.05$). In P11 wave, latency increased from 9.20ms (SD-0.12) in group I to 9.33ms (SD-0.23) in group II which was also significant ($p<0.05$). No studies are available to support this data. This study was unique in the sense that there is relatively low awareness of this manifestation due to a very few studies in Indian population.

CONCLUSION

The results of our study revealed that there was no significant difference on comparing cases with controls in somatosensory evoked potential. On dividing cases into further two groups and comparing serum ferritin level with somatosensory evoked potential, a significant relation was found. With progressive increase in serum ferritin level, abnormalities were found in somatosensory evoked potential, these are subclinical peripheral and central sensory neuropathy. Frequent blood transfusions leading to iron overload have contributed to new spectrum of complications. Hence, the use of neurophysiologic monitoring becomes imperative, enabling early detection of neural pathway impairment and allowing appropriate management. Therefore, it is recommended somatosensory evoked potential should be done periodically in beta thalassemia patients in order to provide, better quality of life.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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Table 1: Comparison of Ssep: Cases Vs Controls

Somatosensory Evoked Potential (Latency)	Cases (N=30)		Controls(N=30)		P Value
	Mean ± Sd	Min - Max	Mean ± Sd	Min - Max	
N9	7.78 ± 1.49	6.4 - 10.8	7.68 ± 1.49	6.3 - 10.7	0.809

N11	10.31 ± 1.75	8.5 - 12.8	10.22 ± 1.76	8.4 - 12.8	0.838
P11	9.29 ± 0.31	9.1 - 9.8	9.20 ± 0.21	9.0 - 9.7	0.088

Table 2: Relation of Ssep with Serum Ferritin Level in Cases

Ssep (Latency)	**Group I(Serum Ferritin<1000ng/Ml)**	**Group Ii(Serum Ferritin>1000ng/Ml)**	**P Value**
	Mean ± Sd	**Mean ± Sd**	
N9	6.84 ± 0.80	8.12 ± 1.55	0.007
N11	9.14 ± 1.48	10.74 ± 1.67	0.024
P11	9.20 ± 0.12	9.33 ± 0.23	0.048