National Journal of Physiology, Pharmacy and Pharmacology

RESEARCH ARTICLE

Comparison of auditory evoked potential changes with the duration of diabetes mellitus

Sudha D[1], Chandraselvi E[2], Saikumar P[2]

[1]Department of Physiology, Melmaruvathur Adhiparasakthi Institute of Medical Sciences and Research, Melmaruvathur, Tamil Nadu, India, [2]Department of Physiology, Sree Balaji Medical College and Hospital, Chennai, Tamil Nadu, India

Correspondence to: Sudha D, E-mail: ssudha91@yahoo.com

Received: December 24, 2019; **Accepted:** January 13, 2020

ABSTRACT

Background: Type II diabetes is typically a chronic disease associated with decreasing life expectancy. This is partly due to the number of complications with which it is associated. With increasing duration, the glycemic control in Diabetes Mellitus is also closely associated with hearing loss. **Aim and Objective:** This study aims to compare the auditory evoked potential (AEP) changes with the duration of Type II diabetic mellitus. **Materials and Methods:** A total of 135 diabetic patients were involved. They were divided into three groups depending on the duration of the disease: Group-I: 0–5 years; Group-II: 5–10 years; and Group-III: 10–15 years. Blood investigation such as glycosylated hemoglobin (HbA1c) level and fasting blood sugar (FBS) levels was done in all the patients. AEP was done in all the participating diabetic patients. **Results:** The FBS and HbA1c level values were increased as disease progressed up to 5–10 years of duration. Further, disease progression that is more than 10–15 years of FBS and HbA1c levels was reduced. The results of the study showed that a significant delay in AEP latencies (wave I, wave III, and wave V) and interpeak latencies (IPL I-III and IPL III-V) was prolonged in Group A-II when compared with the other two groups. The results were analyzed by mean standard deviation and using ANOVAs. **Conclusion:** According to our study, the AEP latencies were prolonged in increased duration of diabetes mellitus patients. This gives a clear idea in managing the disease progress and pathological changes taking place to have a better life.

KEY WORDS: Auditory Evoked Potential; Glycosylated Hemoglobin; Fasting Blood Sugar

INTRODUCTION

International Diabetes Federation stated that in 2011, 61.8 million people had diabetes in India. Diabetic patients with uncontrolled hyperglycemia are affected by both central and peripheral neuropathy. The complex signaling process in the auditory system requires glucose and high levels of energy utilization. Cochlea gets affected more in hyperglycemia. The metabolic disturbance even in short duration of hyperglycemia causes disturbance in cochlea both anatomically and physiologically. The most serious problem of hyperglycemia in the auditory system is the microangiopathy. Stria vascularis which is highly microvascular-dependent organ in cochlea gets affected more. In the endothelium, there is an increased permeability which leads to changes in auditory electrolyte homeostasis within the endolymph which causes interferences with the hair cell transduction and signal transmission.[1]

The evoked potentials that appear following transduction of the acoustic stimulus by the ear cells create an electrical signal that is carried through the auditory pathway to the

Access this article online	
Website: www.njppp.com	Quick Response code
DOI: 10.5455/njppp.2020.10.01003202013012020	

brain stem and from there to cerebral cortex. When signal travels, it generates an action potential in all fibers which are recorded as waves. Five waves are recorded. Waves I, II, III, IV, and V are recorded.

MATERIALS AND METHODS

The study was undergone in the Department of Physiology, Sree Balaji Medical College and Hospital. The diabetic patients were recruited from the Diabetology Outpatient Department in Sree Balaji Medical College and Hospital. In this study, 135 Type II diabetes mellitus patients were taken. Ethical approval was obtained. Informed and written consent was obtained. The study procedure was clearly explained to all the participants. They were divided into three groups depending on the duration of the disease: Group-I: 0–5 years; Group-II: 5–10 years; and Group-III: 10–15 years. Blood investigation such as glycosylated hemoglobin level (HbA1c) and fasting blood sugar levels (FBS) was done in all the patients. Auditory evoked potential (AEP) was done in all the participating diabetic patients using RMS Polyrite in the Department of Physiology, Sree Balaji Medical College and Hospital.

Table 1: Physical characteristic feature in the study population

Parameters	Patients (*n*=135)
Age (years)	44.6±6.7
Male (*n*)	44
Female (*n*)	91
Group D-I	26.9±5.2
Group D-II	28.4±4.1
Group D-III	27.2±4.6

Values are expressed in mean±standard deviation. Group D-I: 0–5 years; Group D-II: 5–10 years; Group D-III: 10–15 years

Table 2: Comparison of AEP and duration of disease in diabetic patients

Duration of DM	AEP				
	I	III	V	I–III	III–V
Group D-I	1.7±0.2	4.0±0.3	6.0±0.3	2.3±0.3	2.3±0.4
Group D-II	3.2±0.9	4.4±0.4	6.4±0.4	2.7±0.4	2.7±0.3
Group D-III	2.5±0.3	4.2±0.2	6.2±0.5	2.6±0.4	2.5±0.3

Values are expressed in mean±standard deviation. Group D-I: 0–5 years; Group D-II: 5–10 years; Group D-III: 10–15 years. AEP: Auditory evoked potential, DM: Diabetes mellitus

RESULTS

The findings of the present study are depicted in Tables 1-3.

DISCUSSION

In conformity with other authors,[2] significant difference in FBS levels ≤120 mg/dl and >120 mg/dl was demonstrated in our study population. Craft *et al*.[3] found a significant correlation between HbA1c and duration of diabetes in patients with poorly controlled diabetes. The results of the study showed that a significant delay in AEP latencies (wave I, wave III, and wave V) and interpeak latencies (IPL I-III, IPL III-V) was prolonged in Group A-II when compared with the other two groups. The results of our study showed a positive correlation between latencies and metabolic control.

Duby *et al*.[4] observed that HbA1c >8% was significantly related to the duration of diabetes. Bril *et al*.[5] proved that the amount of carbohydrates attached to HbA1c increases with the duration of disease. Pudar *et al*.[6] correlated the duration of diabetes and prolongation of interval I and III while

Table 3: Comparison between metabolic control and duration of disease with AEP

Glycemic state	Duration of disease	AEP				
		I	III	V	I–III	III–V
FBS ≤120 mg/dl	Group D-I	1.7±0.2	3.7±0.1	5.6±0.2	1.9±0.2	1.9±0.3
	Group D-II	2.1±0.4	4.0±0.3	6.0±0.3	2.3±0.4	2.3±0.3
	Group D-III	1.9±0.1	3.8±0.2	5.8±0.1	2.1±0.3	2.1±0.3
FBS >120 mg/dl	Group D-I	2.1±0.2	4.1±0.2	6.1±0.2	2.5±0.1	2.6±0.1
	Group D-II	2.6±0.2	4.6±0.2	6.5±0.2	2.8±0.2	2.8±0.2
	Group D-III	2.4±0.4	4.4±0.3	6.4±0.2	2.7±0.3	2.6±0.2
HbA1c ≤6%	Group D-I	1.7±0.2	3.7±0.1	5.6±0.2	1.9±0.2	1.9±0.3
	Group D-II	2.2±0.4	4.1±0.5	6.1±0.4	2.3±0.2	2.3±0.3
	Group D-III	1.9±0.1	3.8±0.2	5.8±0.2	2.1±0.3	2.1±0.4
HbA1c >6%	Group D-I	2.1±0.3	4.2±0.3	6.2±0.2	2.4±0.3	2.4±0.2
	Group D-II	2.7±0.3	4.6±0.3	6.6±0.3	2.8±0.3	2.8±0.3
	Group D-III	2.4±0.4	4.5±0.2	6.4±0.3	2.8±0.3	2.6±0.2

Values are expressed in mean±standard deviation. Group D-I: 0–5 years; Group D-II: 5–10 years; Group D-III: 10–15 years. AEP: Auditory evoked potential, FBS: Fasting blood sugar, HbA1c: Glycosylated hemoglobin

Virtaniemi *et al*.[7] found latency time of wave V prolongation of interval I–V and III–V, but this was not proved by Ottaviani *et al*., Di Leo *et al*., and Dąbrowski *et al.*[8] Recent study[9] had inversely correlated wave V latency and interval I–V with the duration of diabetes.

In 1981, Donald *et al*.[10] were first observed the correlation between AEP with central diabetic neuropathy. Verma *et al*. found that AEP latency was not correlated with diabetes mellitus.[11] In our study, waves I, III, and V were prolonged in poorly controlled diabetes patients (FBS >120 mg/dl and HbA1c >6%). Our results correlated with the results of Kovacic *et al*.[12] who explained that ischemia and lacunar demyelination and the reduction in the nervous fibers of acoustic nerves, superior olivary complex and the lateral lemnisci, the axons or nuclei of the lateral lemnisci, and the inferior colliculi were responsible for the prolongation of waves I, III, and V.

Tóth *et al*. reported a positive correlation between glycemic level and AEP abnormalities.[13] Di Leo *et al*. also showed prolonged AEP latencies with poor glycemic control.[8] Talebi *et al*.[14] did not detect any correlation between HbA1c, FBS, and the AEP results. Díaz de León-Morales *et al*.[15] found that AEP abnormalities in Type II diabetes mellitus were not associated with HbA1c and FBS.

Strength and Limitations

More number of diabetic patients should be involved in further studies.

CONCLUSION

In our study, the FBS and HbA1c level values were increased as disease progressed up to 5–10 years of duration. Further, disease progression that is more than 10–15 years of FBS and HbA1c levels was reduced. There were significant changes in AEP latencies with uncontrolled blood sugar levels. This gives us a widespread vision on regulation of hyperglycemia for a longer duration which can reduce the abnormalities in auditory pathway.

REFERENCES

1. Frisina ST, Mapes F, Kim S, Frisina DR, Frisina RD. Characterization of hearing loss in aged type II diabetics. Hear Res 2006;211:103-13.
2. de Courten-Myers GM, Kleinholz M, Holm P, DeVoe G, Schmitt G, Wagner KR, *et al*. Hemorrhagic infarct conversion in experimental stroke. Ann Emerg Med 1992;21:120-6.
3. Craft S, Foster TC, Landfield PW, Maier SF, Resnick SM, Yaffe K. Session III: Mechanisms of age-related cognitive change and targets for intervention: Inflammatory, oxidative, and metabolic processes. J Gerontol A Biol Sci Med Sci 2012;67:754-9.
4. Duby JJ, Campbell RK, Setter SM, White JR, Rasmussen KA. Diabetic neuropathy: An intensive review. Diabetic Care 2004;61:37-53.
5. Bril V, England J, Franklin GM, Backonja M, Cohen J, Del Toro D, *et al*. Evidence-based guideline: Treatment of painful diabetic neuropathy: Report of the American academy of neurology, the American association of neuromuscular and electrodiagnostic medicine, and the American academy of physical medicine and rehabilitation. Neurology 2011;76:1758-65.
6. Pudar G, Vlaski L, Filipović D, Tanackov I. Correlation of hearing function findings in patients suffering from diabetes mellitus type 1 in regard to age and gender. Med Pregl 2009;62:395-401.
7. Virtaniemi J, Kuusisto J, Karjalainen L, Karjalainen S, Laakso M. Improvement of metabolic control does not normalize auditory brainstem latencies in subjects with insulin-dependent diabetes mellitus. Am J Otolaryngol 1995;16:172-6.
8. Di Leo MA, Di Nardo W, Cercone S, Ciervo A, Lo Monaco M, Greco AV, *et al*. Cochlear dysfunction in IDDM patients with subclinical peripheral neuropathy. Diabetes Care 1997;20:824-8.
9. Dąbrowski M, Mielnik-Niedzielska G, Nowakowski A. Involvement of the auditory organ in type 1 diabetes mellitus. Endokrynol Pol 2011;62:138-44.
10. Donald MW, Bird CE, Lawson IS, Letemendia FJ, Monga TN, Surridge DH, *et al*. Delayed auditory brainstem responses in diabetes mellitus. J Neurol Neurosurg Psychiatry 1981;44:641-4.
11. Verma A, Bisht MS, Ahuja CK. Involvement of central nervous system in diabetes mellitus. J Neurol Neurosurg Psychiatry 1984;47:414-6.
12. Kovacic J, Lajtman Z, Ozegovic I, Knezevic P, Cerir T, Vlasic A. Investigation of auditory brainstem function in elderly diabetic patients. Int Tinnitus J 2009;15:79-82.
13. Tóth F, Várkonyi TT, Rovó L, Lengyel C, Légrády P, Jóri J, *et al*. Investigation of auditory brainstem functions in diabetis patients. Int Tinnitus J 2003;9:84-6.
14. Talebi M, Moosavi M, Mohamadzade NA, Mogadam R. Study on brainstem auditory evoked potentials in diabetes mellitus. Neurosciences 2008;13:370-2.
15. Díaz de León-Morales LV, Jáuregui-Renaud K, Garay-Sevilla ME, Hernández-Prado J, Malacara-Hernández JM. Auditory impairment in patients with type 2 diabetes mellitus. Arch Med Res 2005;36:507-10.

How to cite this article: Sudha D, Chandraselvi E, Saikumar P. Comparison of auditory evoked potential changes with the duration of diabetes mellitus. Natl J Physiol Pharm Pharmacol 2020;10(03):206-208.

Source of Support: Nil, **Conflicts of Interest:** None declared.