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A Comparative study of Auditory Evoked Potential in Young Obese and Normal Subjects

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Abstract

Obesity is a metabolic syndrome associated with many established neurological conditions like meralgia paresthetica, migraine, carpal tunnel syndrome. Well documented defects in nerve conduction, dysfunction involving both peripheral motor and sensory nerves is observed in obese individuals. There is no documented study of cranial nerve involvement in obese individuals. Hence this study was performed to assess the Auditory nerve by auditory evoked potential in young obese Indian adult individuals. Auditory evoked potentials of young obese adults aged between 15 and 30 years were compared with normal. The study recruited 60 age and sex matched normal hearing (confirmed by Pure Tone Audiometry) obese and control subjects. One way ANOVA test was used for the inferential statistics. A significant (P>0.05) increase in the latencies of auditory evoked potential waves I, III, V and no significant change in the inter peak latencies of waves I-III, III-V and I-V were observed in both ears of obese individuals compared with normal subjects. This study demonstrates that the obese individuals are prone to develop early conduction defect to auditory nerve.

Keywords: Obesity, Auditory evoked potential.

Introduction

Obesity is a globally faced major health problem, growing as an epidemic in many nations like India, affecting wide spectrum of age groups, from young to elderly[1-3]. A BMI exceeding 30 kg/sq.m is considered as obese[4]. In urban North Indian population, prevalence of obesity is 5.5% in males and 12.6% in females[5]. Obesity is one of the leading avoidable causes of death worldwide[6-9]. The life expectancy of obese individuals reduces by two to four years with BMI 30-35 kg/sq. m. and ten years with BMI 40 kg/sq. m[10].

Many medical consequences occur as a result of obesity. It is an established risk factor for many diseases like ischemic heart disease, stroke, Type 2 Diabetes mellitus, hypertension, dyslipidemia, and osteoarthritis[11,12]. Obesity is associated with many established neurological conditions like meralgia paresthetica, migraine, carpal tunnel syndrome, dementia, idiopathic intracranial hypertension and learning impairment[13-18].

Obesity is also an inflammatory disease associated with excess production of oxygen free radicals, which causes damage to lipid membranes, proteins, deoxyribonucleic acid (DNA) and other cellular components[19,20]. This may contribute to the pathogenesis of metabolic syndrome and various co morbid conditions like aging, Alzheimer's disease, kidney disease, neurodegenerative disorders and cancer[21,22]. There is a documented defect in nerve conduction, dysfunction in both peripheral motor and sensory nerves by various nerve conduction studies[23,24]. There is no documented study in Indians to assess the cranial nerve involvement in obese individuals. Hence in this study the auditory nerve was assessed by Brain Stem Evoked Response Audiometry (BERA) in young obese and compared with normal healthy individuals.

Material and Methods

Proper ethical clearance was obtained from the Institutional Ethical Committee before the commencement of the procedures. Thirty normal hearing obese outpatients (BMI > 30Kg/sq.m) from the Institute of Endocrinology and Medicine, Madras Medical College and 30 normal individuals aged between 15 and 30 years were included in the study.

Case and control subjects with endocrine disorders, auditory defects like external/ middle/ inner ear pathology, diabetes mellitus, long duration of anaemia, history of taking long term ototoxic drugs, mental retardation and other neurological disorders were excluded from the study.

Both the case and control groups were subjected to Brain Stem Evoked Response Audiometry (BERA). The apparatus for BERA used in our study is RMS EMG EP MARK II, India. The apparatus was set as per the recommended standard for the clinical apparatus for practice of evoked potentials[25]. The three recording electrodes were placed on the scalp following the international electrode placement system on vertex (cz), right (A1) and left (A2) mastoid processes, with the reference electrode over fore head. The low filter set at 3Hz and high filter

150 Hz. A click sound of 80 dB intensity and 100 micro second duration was given as stimuli and white noise of 40-60 dB is used as masking to the subject's ear. A 1500 click with two repetitions was recorded in each subject.

Statistical analysis: The latencies and inter peak latencies of AEP waves I, III, V and waves I-III, III-V, I-V respectively, of two groups were compared by one way analysis of variance. p value less than 0.05 was considered significant.

Results and Discussion

The latencies of waves I, III, V of BERA showed a significantly prolongation in both ears of obese individuals in comparison with normal. Where as inter-peak latencies I-III, I-V and III-V waves of both ears doesn't show statistically significant change in obese with normal control. Prolonged latencies of waves I, III, V of BERA and normal inter peak latencies I-III, I-V and III-V waves of both ears suggests the conduction damage to auditory nerve. This damage could be explained by the following facts in obesity, the expanding adipose tissue mass with fat well in advance of angiogenesis, results in hypoxia. This hypoxia results in recruitment of transcription factor, hypoxia-inducible factor-1, which induces infiltration with inflammatory cell particularly macrophages and results in local and systemic inflammation[26,27,28]. More than 50 different adipokines like tumour necrosing factor- α, interleukin 1 β, interleukin 6, interleukin-10 and inflammation-related signals such as monocyte chemo attractant protein-1, macrophage migration inhibiting factor, nerve growth factor, and adiponectin are known to be released from adipocytes, which are highly heterogeneous both in terms of protein structure and of function[15,29-34]. This inflammation results in imbalance between the production of free radical (especially high levels of reactive oxygen species) and its scavenging mechanism – (Low antioxidant enzyme protein content like copper, zinc, manganese super oxide dismutase and catalase), which results in disturbances of cellular redox homeostasis[21,35]. This would result in damage to neuron and surrounding Schwann cell by associated oxidative stress[12,23]. Hence this study suggests that the obese individuals are proven to develop early damage to auditory nerve.

Conclusion

This study suggests that obese persons are prone to develop early conduction changes of auditory nerve in comparison with normal healthy young adults. This would result in early defects in hearing, which might affect the performance of individuals with obesity in par with healthy individuals both academically and socially in the society. Usage of antioxidants likes vitamin E, A, C and minerals like selenium, zinc, in obese individuals might reduce these effects. But the effects are least and short lived, as the locally produced and pre existing antioxidants play a major role in protection of neuron at the cellular level[19,35,36].

Hence the study suggest the importance of reduction in fat mass, a source of lipid peroxidation and oxidative stress events, is the only mode that might prevent the damage on neurons. Hence, diet restriction from hyperlipidaemic foods is recommended[28,31,37,38]. The recording of auditory evoked potential can be included in the regular package of investigations in obese individuals for early diagnosis and intervention. Hence, this AEP test can be useful for both preventive and prognostic purpose in obesity.

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Table-1
Latencies of AEP waves in test and control groups

Latencies AEP waves	Control Right ear (ms)	Test Right ear(ms)	Control Left ear(ms)	Test Left ear(ms)	F value
I	1.20 ± 0.06	1.69 ± 0.13	1.21 ± 0.07	1.66 ± 0.13	201.79*
III	3.26 ± 0.11	3.61 ± 0.14	3.23 ± 0.07	3.61 ± 0.14	95.03*
V	5.23 ± 0.06	5.68 ± 0.14	5.21 ± 0.06	5.64 ± 0.12	189.40 *

A significant increase in latencies of AEP waves was observed in test group in comparison with normal subjects in both ears.

Table-2
Interpeak Latencies of AEP waves in test and control groups

Interpeak Latencies AEP waves	Control Right ear (ms)	Test Right ear (ms)	Control Left ear (ms)	Test Left ear (ms)	F value
I – III	2.06 ± 0.12	1.93 ± 0.11	2.00 ± 0.15	1.97 ± 0.11	5.78
III - V	1.97 ± 0.13	2.05 ± 0.12	2.00 ± 0.15	1.97 ± 0.11	2.80
I – V	4.03 ± 0.08	3.98 ± 0.11	4.00 ± 0.09	3.98 ± 0.08	2.12

No significant change was observed in interpeak latencies of AEP waves in test group in comparison with normal subjects in both ears.

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