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Original Article

The Relationship between Dyslipidemia, Antioxidants and Nerve Conduction Velocity in Overweight and Obese Persons in Aswan

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ABSTRACT

Background and Aim: Overweight and obesity are major health problems leading to peripheral neuropathy. The exact mechanism of peripheral neuropathy in obesity is uncertain. However, increased oxidative stress and dyslipidemia are potential pathways. To find out the relationship between obesity and peripheral neuropathy in adults in Upper Egypt. Also, to clarify the role of dyslipidemia and oxidative stress in obesity- related neuropathy. **Methods**: Study groups are: normal weight, overweight and obese group (25 subjects in each group). All individuals underwent nerve conduction studies (NCS) of both median and ulnar nerves. In addition, lipid profile, TAC and MDA were measured in the serum. **Result:** We found significant increase in LDL, TG and MDA and significant reduction in HDL in obese compared to normal weight subjects. NCS showed significant slowing of NCV of right median motor and left median sensory, decrease in amplitude of ulnar motor nerves on both sides and right median motor nerve. Overweight group showed significant increase in LDL, TC and MDA compared to normal weight group. **Conclusion**: Our study suggested that obesity induces peripheral neuropathy. It also suggested that oxidative stress and dyslipidemia play an important role in the pathogenesis of obesity-related neuropathy.

Key words: Body mass index, Nerve conduction study, Obesity, Median nerve, Ulnar nerve.

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INTRODUCTION

Overweight and obesity are major health problems.[1] Many risk factors lead to obesity such as; Genetic, socioeconomic status, behavioral and environmental factors.[2] The global prevalence of obesity almost tripled between 1975 and 2016.[3]

Peripheral nerve dysfunction affects mobility, physical functions and quality of life.[4] Obese individuals can develop multiple forms of peripheral neuropathy such as distal symmetric polyneuropathy, small fiber neuropathy and cardiovascular autonomic neuropathy.[5]

Obesity induces elevation of blood glucose level[6] and reduction in serum vitamin B_{12} level.[7-8] Both hyperglycemia and vitamin B_{12} deficiency are risk factors for the development of peripheral neuropathy.[9-10] The role of obesity-independent of blood glucose level or serum vitamin B_{12} level- on induction of peripheral neuropathy needs further investigation.

The exact mechanism of the development of peripheral neuropathy in obesity is uncertain, but increased oxidative stress, inflammation and dyslipidemia may be involved.[11] Malondialdehyde (MDA) is one of the products of lipid peroxidation and it is used as a marker of oxidative stress.[12] Lipid peroxidation generates free radical causing damage to cell structures.[13]

Although obesity is associated with numerous complications, yet obesity is a readily preventable metabolic disease.[14] Therefore, all complications resulting from obesity could be prevented or reversed if overweight and obesity were controlled. Because of the global epidemic of obesity and the association between obesity and peripheral neuropathy,[5] we hypothesized that obesity per se- regardless of hyperglycemia or vitamin B_{12} deficiency- has a role in the development of peripheral neuropathy in obese subjects. We also postulated that oxidative stress and dyslipidemia plays an important role in the development of peripheral neuropathy in obese subjects. Therefore, our aims of work are:

1. Assessment of peripheral nerve function in overweight and obese non-diabetic Upper Egypt population.
2. Investigate oxidative stress and dyslipidemia as possible mechanisms for peripheral neuropathy in the study groups.

PATIENTS AND METHODS

After approval by the Research Ethics Committee (15/5/2017), Faculty of Medicine, Aswan University, Egypt. Seventy-five individuals presenting to Aswan University hospitals, who are aged from 18 years to

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50 years were recruited to be studied. A written informed consent was obtained from each subject included in the study. They were allocated into three groups (25 individuals in each group) based on their BMI as follow:

Group 1: Control subjects with BMI from 18.5 to < 25kg/m^2 (10 males, 15 females).

Group 2: Patients with BMI ≥ 25kg/m^2 and < 30 kg/m^2 (8 males, 17 females).

Group 3: Patients with BMI ≥ 30kg/m^2 (10 males, 15 females).

Exclusion criteria included diabetes, thyroid disease, root lesions, other neurological or neuromuscular conditions, neurotoxic drugs, pregnancy, alcoholism, renal failure and hepatic failure.

The study subjects and the control groups were matched with respect to age, gender and height. The body mass index (BMI: kg/m^2) of all participants was calculated. Detailed history regarding neuropathy was taken. Complete neurological examination was performed to look for signs of neuropathy such as diminished ankle jerk, diminished power. Sensory examination for loss of light touch, superficial pain, temperature sense, vibration and joint position was done. Prior to study, vitamin B_{12} was administered to all included groups by intramuscular injection for one month to exclude vitamin B_{12} deficiency.

Nerve Conduction Study (NCS)

The nerve conduction study was performed using NCV/EMG machine (Nihon Kohden, made in Japan) with standard equipment settings of sensitivity 2–5 μV/division, filter settings: low frequency 20 Hz, high frequency 2 kHz, sweep speed 1 msec/division. The room temperature was maintained between 21– 23°C. Patient's limb was placed in relaxed position. For motor conduction study, orthodromic recording was done and for sensory nerve study, antidromic recording was done. We used the ring type of electrodes for the sensory NCS and the disc type for motor NCS. Electrodes were fixed with adhesive tape to the skin and gel was applied under the electrode to reduce electrode impedance.[15]

Motor and sensory nerve conduction studies were performed for median and ulnar nerves in upper limb. Recordings were taken in both sides. The following parameters were measured:

a) For motor nerve conduction study, distal latency (msec), amplitude (mv), nerve conduction velocity in m/sec and F-wave latency in (msec).
b) For sensory nerve conduction study, the onset latency, peak-to-peak amplitude (μv) and nerve conduction velocity in m/sec.

Biochemical analysis

Five ml of venous blood was withdrawn after overnight fasting from all participants. Blood was collected and allowed to clot for 30 min. at 25°C. Then, blood was centrifuged at 2,000 xg for 15 min at 4°C. Then the top yellow serum layer was pipetted off without disturbing the white buffy layer. MDA and TAC were determined by colorimetric method according to manufacturer's instruction (Bio-diagnostic, Diagnostic and research reagent Catalog No. MD 25 28, Cat. No. TA 25 12 respectively). TC, TG, HDL-c and LDL-c were determined by enzymatic colorimetric method by kits purchased from Egyptian Company for Biotechnology (S.A.E) (REF: 230 002, REF: 314 002, REF: 266002 respectively). Glucose was determined by enzymatic colorimetric method, Biotecnica Instruments S.P.A., Italy.

Statistical Analysis

Data were verified, coded and analyzed using IBM-SPSS 21(IBM_SPSS. Statistical Package for Social Science. Ver.21. Standard version. Copyright © SPSS Inc., NY, USA. 2012). Descriptive statistics: Means, standard deviations and medians were calculated. One-way ANOVA was carried out to compare the means of normally distributed data, while Kruskal Wallis test was calculated to test the median differences of the data that do not follow normal distribution. Correlation analysis was used to test the association between variables (Spearman's rank correlation). $P \leq 0.05$ was considered significant.

RESULTS

Demographic results in the study groups

Our study showed that there was no significant difference between study groups in all demographic parameters used in this study such as age, gender and height, (Table 1).

Lipid Profile pattern in study groups

On assessment of lipid profile in our study, we found significant increase in LDL-c in overweight compared to normal weight group ($P < 0.05$) and in obese compared to normal weight group ($P < 0.05$). HDL-c showed no difference between overweight and control groups, however, there was significant reduction in HDL-c in obese compared to normal weight group ($P < 0.05$) (Figure 1B). As regard TG, there was no difference in serum TG between overweight and control groups. Significant increase in obese compared to normal weight group (P < 0.05) (Figure 1C). TC level showed significant elevation in overweight compared to normal weight group ($P < 0.05$). However, TC level in obese subject was not significantly different from normal weight group ($P >0.05$) (Figure 1D).

Levels of oxidative stress markers in study groups

To evaluate the oxidative stress status in our study groups we measured serum TAC and MDA. Serum TAC showed no difference between the study groups (Figure 2A). However, we found significant increase in MDA level in overweight compared to normal weight group ($P < 0.05$) and in obese vs. normal weight group ($P < 0.001$) (Figure 2B).

Results of nerve conduction velocity study in the studied groups.

In our study we found that, the only significant difference in NCV between groups was decrease of NCV of right median motor and left median sensory in obese compared to normal weight group ($P < 0.05$), (Table 2).

Table 1: Shows Socio-demographic differences between the studied groups.

	Control (No.=25)	Overweight (No.=25)	Obese (No.=25)
Age	30.0±7.5	36.8±9.1	34.6±10.3
Gender	10 (male)	8 (male)	10 (male)
	15 (female)	17 (female)	15 (female)
Weight (kg)	62.0±5.4	73.8±16.3*	94.8±7.1** #
Height (cm)	161.3±6.1	163.3±7.9	164.4±7.4
BMI (kg/m^2)	23.8+101	27.6±5.3*	34.9+1.3

Significance level *P ≤ 0.01 vs normal group, **P ≤ 0.001 vs normal group, #P ≤ 0.001 vs overweight group. Data shown are the mean ± standard deviation.

Table 2: Shows the results of nerve conduction velocity (m/sec) of the bilateral motor-sensory median and ulnar nerves between the studied groups.

	Control (No.=25)	Overweight (No.=25)	Obese (No.=25)
Lt. Median Nerve Motor	51.5±3.2	52.6±8.3	52.7±13.6
Lt. Median Nerve Sensory	51.6±5.6	46.9±12.6	37.9±16.2+
Rt. Median Nerve Motor	51.7±2.5	53.9±7.5	43.6±13.8
Rt. Median Nerve Sensory	48.7±3.6	47.2±8.9	43.7±16.9
Lt. Ulnar Nerve Sensory	50.2±1.5	48.3±4.7	49.0±17.4
Lt. Ulnar Nerve Motor	58.2±9.7	62.0±14.1	55.3±18.5
Rt. Ulnar Nerve Motor	55.2±1.1	57.19±4.3	48.50±3.7 #
Rt. Ulnar Nerve Sensory	51.4±3.7	49.8±2.6	49.3±3.9

Significance level *P ≤ 0.05 vs normal group, +P ≤ 0.01 vs normal group, #P ≤ 0.05 vs overweight group. Data shown are the mean ± standard deviation.

Table 3: Shows the results of nerve Conduction amplitude (mv) in motor NCS, (μv) in sensory NCS) of the bilateral motor-sensory median and ulnar nerves between the studied groups.

	Control (No.=25)	Overweight (No.=25)	Obese (No.=25)
Lt. Median Nerve Motor	8.86±2.7	8.30±2.3	7.68±4.2
Lt. Median Nerve Sensory	18.9±9.9	18.2±9.8	18.2±13.5
Rt. Median Nerve Motor	9.59±4.8	8.88±4.0	6.97±3.2*
Rt. Median Nerve Sensory	17.9±5.5	16.2±5.9	17.4±6.6
Lt. Ulnar Nerve Motor	11.2±2.9	8.91±3.9	6.33±4.1 +#
Lt. Ulnar Nerve Sensory	14.04±4.0	17.58±5.8	16.75±9.9
Rt. Ulnar Nerve Motor	11.2±1.3	9.93±4.1	8.99±2.3*
Rt. Ulnar Nerve Sensory	18.2±6.2	19.0±6.5	19.4±6.6

Significance level *P ≤ 0.05 vs normal group, +P ≤ 0.001 vs normal group, #P ≤ 0.05 vs overweight group, n=number of participant /group. Data were expressed as the mean ± standard deviation.

Results of nerve conduction amplitude in the studied groups

Nerve conduction studies showed significant decrease in amplitude of ulnar motor nerve on both sides and right median motor nerve in obese compared to normal weight group ($P < 0.05$). Otherwise, there was no significant differences between groups, (Table 3).

Results of F-wave measurement in the studied groups.

F- wave studies showed that there was no difference between overweight and control subjects. However, there was significant prolongation of F waves in obese subject compared to normal weight group ($P < 0.05$) in all studied nerves, (Table 4).

Table 4: Shows results of F-wave (msec) measurement of the bilateral median and ulnar nerves between the studied groups.

	Control (No.=25)	Overweight (No.=25)	Obese (No.=25)
Left Median Nerve	25.0±2.2	27.1±2.5	27.8±3.8 +
Left Ulnar Nerve	24.5±1.7	26.2±2.5	26.2±3.0*
Right Median Nerve	25.6±2.1	26.9±2.5	28.5±3.5 +
Right Ulnar Nerve	24.7±1.9	25.9±2.4	28.0±4.0 +#

Significance level *P ≤ 0.05 vs normal group, +P ≤ 0.01 vs normal group, #P ≤ 0.05 vs overweight group, n=number of participant /group. Data were expressed as the mean ± standard deviation.

Correlations between BMI, MDA and Lipid Profile with NCV

To establish a relationship between BMI, markers of oxidative stress and peripheral neuropathy, we assessed their correlation. The results showed that BMI, high MDA level and high TC, high TG were negatively correlated to nerve conduction velocity. This correlation was statistically significant ($P \leq 0.05$), Low HDL-c, on the other hand showed positive correlation with decreased nerve conduction velocity ($P \leq 0.05$), (Table 5).

DISCUSSION

In the current study we addressed the effect of overweight and obesity on the development of peripheral neuropathy. We eliminated the effect of high blood glucose or vitamin B_{12} deficiency on nerve conduction function.

Overweight and obesity are growing health problem.[16] Although obesity is growing to be an epidemic, it is a readily preventable disease.[14] Therefore, treating or preventing obesity will help to prevent numerous complications resulting from it.[17] In the current study, we hypothesized that overweight and obesity could lead to neuropathy through dyslipidemia and increased oxidative stress.

Our data showed that in overweight subjects, there was only elevated levels of oxidative stress markers (MDA) associated with derangement of lipid profile. Nerve conduction studies didn't show significant changes between overweigh and control subjects, suggesting that metabolic derangement in overweight patients precedes neural tissue damage that takes place in obese subjects as we will discuss next.

In the current study, as the BMI increased, patients showed decreased HDL-c, increased TG level, increased MDA beside the development of peripheral neuropathy. These data suggest that the abnormal lipid profile and the increased oxidative stress can be causative factor for the development of peripheral neuropathy in obese subjects.

Nerve conduction velocity (NCV) showed significant negative correlation between nerve conduction velocity and BMI. NCV relies on age, temperature, height, gender, nerve fiber diameter, myelination and inter nodal distance.[18-19] Demyelinating neuropathy usually shows slowing of NCV.[20]

Table 5: Shows correlations between BMI, serum MDA level, and lipid profile with NCV (m/sec) of the motor/sensory median and ulnar nerves in the studied groups.

Nerve	BMI	MDA	HDL	LDL	TG	Cholesterol
LMN Motor	0.035	0.128	0.123	0.139	-0.078	0.298
LMN Sensory	0.397	*-0.374	*0.190	0.073	-0.031	-0.027
RMN Motor	0.293	0.022	0.223	-0.058	-0.276*	0.027
RMN Sensory	0.224*	-0.251*-	0.075	0.181	-0.043	-0.045
LUN Motor	0.109	0.266*	0.360*	0.096	-0.020	0.009
LUN Sensory	0.043	-0.102	0.014	-0.213	-0.164	-0.427*
RUN Motor	0.185	-0.132	0.069	-0.002	-0.164	-0.176
RUN Sensory	0.242	-0.219	-0.008	0.026	-0.112	-0.324 *

Correlation analysis was used to test the association between variables (Spearman's rank correlation). Correlation is significant (*) at the P ≤ 0.05 level

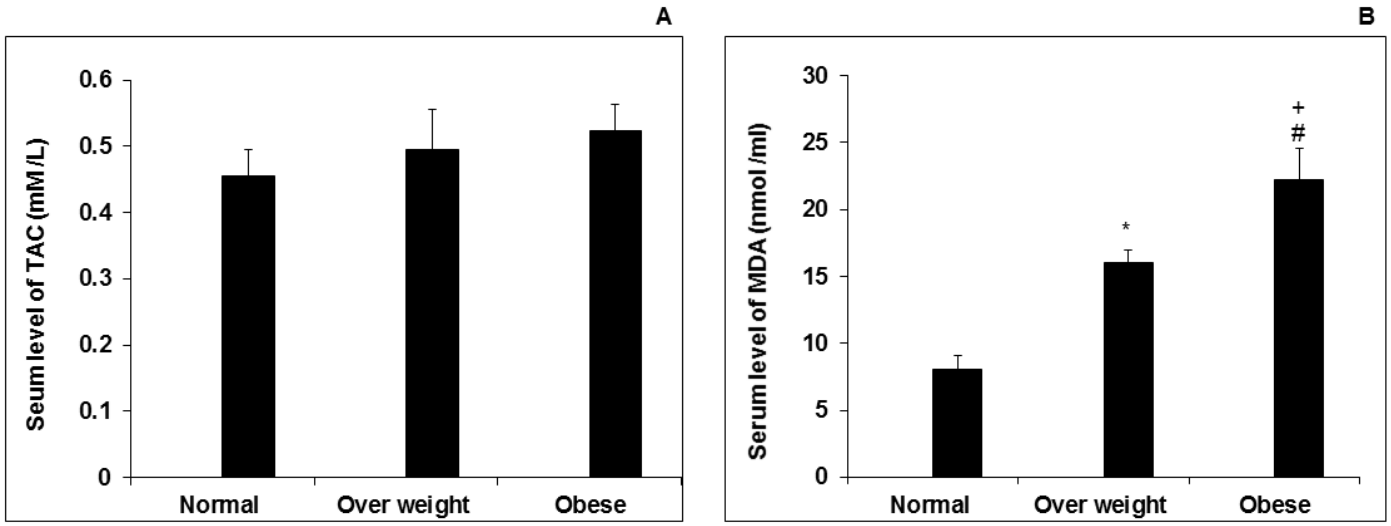

Figure 2A: shows a comparison of serum total antioxidant capacity (TAC) (mM/L) between the studied groups. Data were expressed as the mean ± standard deviation. **Figure 2B:** shows a comparison of serum malondialdehyde (MDA) (nmol/ml) of the studied groups. Data shown are the mean ± standard deviation. *P ≤ 0.05 vs normal group, +P ≤ 0.001 vs normal group, #P ≤ 0.05 vs overweight group.

Suggesting that the prolongation of NCV in obese subjects in the current study can be attributed to demyelination.

In addition to reduction in NCV in obese subjects, we observed reduction in amplitude in motor ulnar nerves and median motor nerves in obese groups. However, sensory ulnar and median nerves did not show significant difference among the three groups. A reduction in amplitude means loss of axons, loss of muscle fibers or conduction block.[9] Reduction in amplitude may also be due to compression by adipose tissue,[21] metabolic effect of obesity,[22] or due to increased skin fold thickness.[23]

F-wave results from antidromic activation of anterior horn cell (backfiring).[24] It reflects conduction along whole motor pathway of the peripheral axons including proximal segment.[25] F-wave changes may be the earliest and the only electrophysiological defect detected in mild cases of peripheral neuropathy.[26] There was significant prolongation of F waves latencies of ulnar and median nerves with increasing BMI, suggesting proximal segment dysfunction in obese subjects. These results are in agreement with Huang *et al.* (2009),[27] who observed that individuals with higher body weights have longer latencies of the median F-wave studies compared to those with lower body weights. In contrast, Thakare *et al.*(2016) did not find association between body mass index and F- wave latency.[28] These differences could be attributed to the difference in the age of the study groups.

Lipids are major structural contents of the nerve cell and therefore dyslipidemia affects neuronal structure and functions.[29] In the current study, we found significant correlation between low HDL-c and decreased conduction velocity of ulnar motor nerve in obese subjects. Furthermore, there was positive correlation between high TG and decreased nerve conduction velocity of median motor nerve. These data suggest that dyslipidemia plays an important role in peripheral nerve dysfunction.

Previous studies demonstrated that high fat diet leads to neuronal damage due to oxidative stress resulting from lipid metabolism.[11,30]

Our data showed that there was elevated level of lipid peroxidation product, MDA. Which showed negative correlation with conduction velocity of median sensory nerve. It is very well known that high MDA level triggers neurotoxic cascade, apoptosis and cell death in peripheral nerves.[31] These data suggest that oxidative stress induces peripheral nerve dysfunction during obesity.

CONCLUSION

Our study showed that overweight and obese subjects developed dyslipidemia and increased markers of lipid peroxidation.

In addition, obese subjects showed reduced nerve conduction velocity and reduced nerve conduction amplitude when compared to normal participants. These findings suggest that obesity - through dyslipidemia and increased lipid peroxidation - results in peripheral nerve demyelination and axonal degeneration. Which results in peripheral neuropathy in obese subject.

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None

CONFLICT OF INTEREST

The author declare no confict of interest.

ABBREVIATIONS USED

NCS: Nerve conduction studies; **HDL:** High density lipoprotein; **LDL:** Low density lipoprotein.

SUMMARY

The aim of the present study was to find out the relationship between obesity and peripheral neuropathy in adults of Upper Egypt. Also, to clarify the role of dyslipidemia and oxidative stress in obesity- related neuropathy. Study groups are: normal weight, overweight and obese group (25 subjects in each group). All individuals underwent nerve conduction studies (NCS) of both median and ulnar nerves. In addition, lipid profile, TAC and MDA were measured in the serum. We found significant increase in LDL, TG and MDA and significant reduction in HDL in obese compared to normal weight subjects. NCS showed significant slowing of NCV of right median motor and left median sensory, decrease in amplitude of ulnar motor nerves on both sides and right median motor nerve. Overweight group showed significant increase in LDL, TC and MDA compared to normal weight group. Our study suggested that obesity induces peripheral neuropathy. It also suggested that oxidative stress and dyslipidemia play an important role in the pathogenesis of obesity-related neuropathy.

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