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Effect of Body Mass Index on Lipid Profile of Type 2 DiabeticPatients at an Urban Tertiary Hospital in Nigeria

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Abstract:

Introduction: Diabetes mellitus is an important global public health problem. In addition to being a chronic disease, obesity is a key risk factor for Type 2 diabetes mellitus (T2DM) that leads to disability. This study aimed at investigating the effect of Body Mass Index on serum lipid profile in type II diabetic patients.

Material and Methods

This is a prospective study and observational study will be done in the Department of Physiology in collaboration with the Department of Medicine, Index Medical College, Hospital & Research Centre, Indore. Inclusion Criteria: Only Diagnosed cases of male and female Type II Diabetes Mellitus cases aged 30-60 suffering from more than 2yrs. The diagnosis of diabetes is made on the basis of (Revised AmericanDiabetic Association criteria). Fasting glucose >126mg/dl and 2hr postprandial plasma glucose >200mg/dl.

Results: The mean (± SD) of various motor nerve variables like latency, amplitude and nerve conduction velocity between control and cases. When compared these variables between controls and cases was statistically highly significant ($p<0.001$). Comparison of various motor nerve variables like latency, amplitude and nerve conduction velocity between control and cases are graphically shown.

Conclusion: A significant negative correlation between BMI and HDL-C was observed, while the correlationbetween BMI and LDL-C was observed to be insignificant. HDL-C was found significantly higher in patientswith normal BMI. These results are important to indicate that there is modest impact of BMI on lipid profile. Therefore, assessment and management for altered blood lipids should not be based on a patient's body weight or BMI.

Keywords: Type II diabetes, serum lipids profile, Body mass index, obesity, overweight.

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Introduction

Diabetes is associated with a greater risk of morbidity and mortality from cardiovascular disease (CVD), and heart disease is the leading cause of death among people with diabetes [1]. Diabetes mellitus is a chronic disease that requires long term medical attention both to limit the development of its devastating complications and manage them when they occur. It is more common amongst developed countries where affluent and overweight individuals live longer than in under developed countries [2].

Dyslipidemia is a well-recognized and modifiable risk factor for cardiovascular diseases which is currently a leading cause of morbidity and mortality world-wide.

Dyslipidemia is common in DM, as both insulin deficiency and resistance affect enzymes and pathways of lipid metabolism [3]. Diabetic dyslipidemia is characterized by raised triglycerides, low high-density lipoprotein, raised apo-B, and small dense low density lipoprotein particles. It may be present at the diagnosis of type 2DM and it is a component of the metabolic syndrome. The pathogenesis of heart disease in diabetes is complex, but serum lipids are frequently abnormal and likely contribute to the risk of coronary artery disease**.** Lipids and lipoproteins are well known risk factors for ischemic heart disease. Elevated levels of triglyceride, cholesterol, and LDL are documented as risk factors for atherogenesis. It is noteworthy that CRP plasma levels even slightly higher from the conventional upper limit of normal (1mg/dL) have been associated with a 2-3-fold increase in risk of future myocardial infarction, stroke, and peripheral atherosclerosis among apparently healthy middle-aged men and women [4].

A worldwide epidemic exists with respect to diabetes mellitus, primarily because of increased rates of obesity. Obesity has become widespread in developed countries along with a corresponding increase in the prevalence of diabetes [5]. Epidemiological studies have shown that, compared to lean individuals, very obese men and women (body mass index >35) have several folds increase in probability of developing Type 2 diabetes. It has been established that BMI is a significant predictor of cardiovascular disease and type 2 diabetes mellitus [6].

Obesity can be described as an imbalance between energy intake and expenditure such that excess energy is stored in fat cells, which enlarge or increase in number. Obesity is defined as a body mass index (BMI) of > 30 kg/m^2, according to WHO criteria. Obesity and overweight are significant public health problems worldwide, affecting an estimated 1 billion persons and contributing to hypertension, type 2 diabetes mellitus, cardiovascular disease, and death. Its prevalence in developed countries, such as the United States, is as high as 26.6% in men and 32.2% in women above age 20 years [7].

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MATERIAL AND METHODS

This is a prospective study and observational study will be done in the Department of Physiology in collaboration with the Department of Medicine, Index Medical College, Hospital & Research Centre, Indore.

Sample Size: 67 Controls and 67 cases (Type II Diabetes Mellitus)

Inclusion Criteria:

Control:

- Willingness (Informed consent)
- Normal healthy aged 30-60 years.

Case:

- Willingness (informed consent)
- Only Diagnosed cases of male and female Type II Diabetes Mellitus cases aged 30-60 suffering from more than 2yrs.
- The diagnosis of diabetes is made on the basis of (Revised American Diabetic Association criteria) [295].
- Fasting glucose >126mg/dl and 2hr postprandial plasma glucose >200mg/dl.

Exclusion Criteria:

- No previous history of any systemic condition related to peripheral nerve dysfunction (Hypertension, Alcoholic nerve dysfunction, Renal failure).
- Any neuromuscular disordes such as myopathy, familial polyneuropathy or chronic polyneuropathy.
- Neuropathies associated with toxic agents, metals or drugs.
- Skin ailments or swelling that would affect with Nerve Conduction Study (NCS).

Result

The present study includes 67 cases (Type II Diabetes Mellitus subjects) who are attendants of the patients in OPD of Department of Medicine, Index Institute of Medical Sciences and research, and 367 Controls.

The cases were further divided into two groups i.e., Overweight and Obese. Groups Sex distribution of overweight group was, 120 male and 60 female and obese groups was, 70 male and 50 female.

The table no. 1 showed the mean (± SD) of various sensory nerve variables like latency, amplitude and nerve conduction velocity between control and cases. When compared these variables between controls and cases was statistically highly significant ($p<0.001$). Comparison of various sensory nerve variables like latency, amplitude and nerve

conduction velocity between control and cases are graphically shown.

Table No. 1: Comparison of Various Sensory Nerve Studies inControls and Cases

Nerves	Parameters	Control (n-300)	Case (n-300)	P Value
		Mean± SD	Mean± SD	
Right Median Nerve	Latency (ms)	3.06 ± 0.43	4.19 ± 0.38	< 0.001
	Amplitude (mv)	5.30 ± 0.64	4.28 ± 0.31	< 0.001
	NCV (m/s)	49.24 ± 2.89	47.21 ± 1.90	< 0.001
Left Median Nerve	Latency (ms)	2.91 ± 0.43	4.37 ± 0.42	< 0.001
	Amplitude (mv)	5.39 ± 0.71	4.67 ± 0.40	< 0.001
	NCV (m/s)	49.81 ± 2.98	47.13 ± 1.92	< 0.001
Right Ulnar Nerve	Latency (ms)	1.76 ± 0.37	3.64 ± 0.31	< 0.001
	Amplitude (mv)	4.83 ± 0.69	4.87 ± 0.27	< 0.001
	NCV (m/s)	48.23 ± 1.76	45.38 ± 1.83	< 0.001
Left Ulnar Nerve	Latency (ms)	1.86 ± 0.16	3.76 ± 0.39	< 0.001
	Amplitude (mv)	4.97 ± 0.74	4.52 ± 0.34	< 0.001
	NCV (m/s)	48.72 ± 2.81	43.57 ± 1.86	< 0.001
Right Sural Nerve	Latency (ms)	1.97 ± 0.44	3.82 ± 0.30	< 0.001
	Amplitude (mv)	8.36 ± 1.05	6.26 ± 0.36	< 0.001
	NCV (m/s)	46.43 ± 2.94	40.21 ± 1.56	< 0.001
Left Sural Nerve	Latency (ms)	1.93 ± 0.41	3.92 ± 0.25	< 0.001
	Amplitude (mv)	8.56 ± 1.08	6.46 ± 0.34	< 0.001
	NCV (m/s)	46.53 ± 2.94	40.71 ± 2.06	< 0.001

Data presented as Mean±SD, p value >0.05 Not significant < 0.05 = significant, <0.001 = highly significant.

The table no. 2 showed the mean (± SD) of various motor nerve variables like latency, amplitude and nerve conduction velocity between control and cases. When compared these variables between controls and cases was statistically highly significant ($p<0.001$). Comparison of various motor nerve variables like latency, amplitude and nerve conduction

velocity between control and cases are graphically shown.

Table No. 2: Comparison of various Motor Nerve Studies in Controls and Cases

Nerves	Parameters	Control (n-300)	Case (n-300)	P Value
		Mean± SD	Mean± SD	
Right Median Nerve	Latency (ms)	3.89 ± 0.21	4.48 ± 0.42	< 0.001
	Amplitude (mv)	5.78 ± 0.61	3.80 ± 0.71	< 0.001
	NCV (m/s)	54.20 ± 3.10	50.45 ± 2.75	< 0.001
Left Median Nerve	Latency (ms)	3.98 ± 0.12	4.87 ± 0.42	< 0.001
	Amplitude (mv)	5.70 ± 0.61	3.10 ± 0.76	< 0.001
	NCV (m/s)	54.1 ± 2.90	50.68 ± 3.78	< 0.001
Right Ulnar Nerve	Latency (ms)	4.49 ± 0.59	6.72 ± 0.48	< 0.001
	Amplitude (mv)	5.24 ± 0.79	4.97 ± 0.78	< 0.001
	NCV (m/s)	56.82 ± 2.93	52.54 ± 2.71	< 0.001
Left Ulnar Nerve	Latency (ms)	4.80 ± 0.50	6.52 ± 0.47	< 0.001
	Amplitude (mv)	5.34 ± 0.78	4.56 ± 0.72	< 0.001
	NCV (m/s)	56.63 ± 2.96	52.59 ± 2.83	< 0.001
Right Common Peroneal Nerve	Latency (ms)	4.16 ± 0.38	4.21 ± 0.34	< 0.001
	Amplitude (mv)	3.82 ± 0.372	4.10 ± 0.30	< 0.001
	NCV (m/s)	46.10 ± 2.51	42.17 ± 2.80	< 0.001
Left Common Peroneal Nerve	Latency (ms)	3.96 ± 0.37	4.87 ± 0.28	< 0.001
	Amplitude (mv)	3.62 ± 0.29	4.16 ± 0.26	< 0.001
	NCV (m/s)	46.50 ±0.38	42.18 ± 3.00	< 0.001

Data presented as Mean±SD, p value >0.05 Not significant < 0.05 = significant, <0.001 = highly significant

The table no. 3 showed the mean (± SD) of various sensory nerve variables like latency, amplitude and nerve conduction velocity between controls (normal BMI) and cases (overweight and obese group). When compared these variables between controls and cases was statistically highly significant ($p<0.001$). Comparison of various sensory nerve variables like latency, amplitude and nerve conduction velocity between control and cases are graphically shown.

Table No 3: Comparison of various Sensory Nerve Studies in Normal BMI,Overweight and Obese

Nerves	Parameters	Controls (n-300)	Cases (n-180+120)		P Value
		Normal BMI	Overweight	Obese	
		Mean± SD	Mean± SD	Mean± SD	
Right Median Nerve	Latency (ms)	2.98 ± 0.57	4.69 ± 0.38	4.64 ± 0.37	< 0.001
	Amplitude (mv)	5.20 ± 0.68	4.12 ± 0.39	4.21 ± 0.38	< 0.001
	NCV (m/s)	50.24 ± 3.09	46.15 ± 1.37	44.92 ± 1.92	< 0.001
Left Median Nerve	Latency (ms)	2.91 ± 0.50	4.69 ± 0.34	4.96 ± 0.30	< 0.001
	Amplitude (mv)	5.14 ± 0.73	4.12 ± 0.33	4.64 ± 0.36	< 0.001
	NCV (m/s)	50.85 ± 2.98	47.11 ± 1.50	44.85 ± 1.79	< 0.001
Right Ulnar Nerve	Latency (ms)	1.88 ± 0.35	3.79 ± 0.27	3.73 ± 0.22	< 0.001
	Amplitude (mv)	4.65 ± 0.63	4.74 ± 0.26	4.24 ± 0.04	< 0.001
	NCV (m/s)	48.23 ± 1.78	47.21 ± 1.81	44.58 ± 1.73	< 0.001
Left Ulnar Nerve	Latency (ms)	1.69 ± 0.24	3.53 ± 0.37	3.87 ± 0.21	< 0.001
	Amplitude (mv)	4.89 ± 0.66	4.24 ± 0.30	4.23 ± 0.07	< 0.001
	NCV (m/s)	48.72 ± 2.82	47.31 ± 1.93	44.38 ± 1.94	< 0.001
Right Sural Nerve	Latency (ms)	1.25 ± 0.39	3.94 ± 0.28	3.73 ± 0.24	< 0.001
	Amplitude (mv)	8.36 ± 1.07	6.51 ± 0.27	6.65 ± 0.26	< 0.001
	NCV (m/s)	46.43 ± 2.74	40.67 ± 1.83	38.39 ± 1.21	< 0.001
Left Sural Nerve	Latency (ms)	1.93 ± 0.32	3.82 ± 0.18	3.73 ± 0.18	< 0.001
	Amplitude (mv)	8.56 ± 1.09	6.59 ± 0.27	6.45 ± 0.20	< 0.001
	NCV (m/s)	46.53 ± 2.16	40.26 ± 1.89	38.15 ± 1.31	< 0.001

Data presented as Mean±SD, p value >0.05 Not significant < 0.05 = significant, <0.001 = highly significant.

The table no. 4 showed the mean (± SD) of various motor nerve variables like latency, amplitude and nerve conduction velocity between controls (normal BMI) and cases (overweight and obese group). When compared these variables between controls and cases was statistically highly significant (p<0.001). Comparison of various motor nerve variables like latency, amplitude and nerve conduction velocity between control and cases are graphically shown.

Table No. 4: Comparison of various Motor Nerve Studies in Normal BMI,Overweight and Obese

Nerves	Parameters	Controls (n-300)	Cases (n-180+120)		P Value
		Normal BMI	Overweight	Obese	
		Mean± SD	Mean± SD	Mean± SD	
Right Median Nerve	Latency (ms)	3.79 ± 0.12	4.45 ± 0.36	4.79 ± 0.36	< 0.001
	Amplitude (mv)	5.68 ± 0.55	3.91 ± 0.79	2.90 ± 0.20	< 0.001
	NCV (m/s)	54.35 ± 2.70	51.24 ± 1.84	48.79 ± 2.72	< 0.001
Left Median Nerve	Latency (ms)	3.68 ± 0.12	4.45 ± 0.37	4.80 ± 0.33	< 0.001
	Amplitude (mv)	5.40 ± 0.54	3.37 ± 0.74	2.71 ± 0.20	< 0.001
	NCV (m/s)	54.35 ± 1.75	51.78 ± 3.91	48.59 ± 1.72	< 0.001
Right Ulnar Nerve	Latency (ms)	4.69 ± 0.54	6.32 ± 0.48	6.43 ± 0.46	< 0.001
	Amplitude (mv)	5.74 ± 0.73	4.91 ± 0.76	4.54 ± 0.79	< 0.001
	NCV (m/s)	56.82 ± 2.79	53.49 ± 1.78	51.71 ±2.97	< 0.001
Left Ulnar Nerve	Latency (ms)	4.64 ± 0.35	6.32 ± 0.37	6.82 ± 0.41	< 0.001
	Amplitude (mv)	5.39 ± 0.77	4.28 ± 0.66	4.28 ± 0.73	< 0.001
	NCV (m/s)	56.63 ± 2.86	53.59 ± 2.88	51.71 ± 2.71	< 0.001
Right Common Peroneal Nerve	Latency (ms)	3.65 ± 0.35	4.36 ±0.35	4.87 ± 0.18	< 0.001
	Amplitude (mv)	3.72 ± 0.32	4.57 ± 0.23	4.43 ± 0.28	< 0.001
	NCV (m/s)	46.30 ± 1.93	44.21 ± 1.86	40.71 ± 1.94	< 0.001
Left Common Peroneal Nerve	Latency (ms)	3.65 ± 0.29	4.46 ± 0.37	4.61 ± 0.18	< 0.001
	Amplitude (mv)	3.73 ± 0.29	4.88 ±0.29	4.60 ± 0.24	< 0.001
	NCV (m/s)	46.35 ± 3.15	44.72 ± 2.74	40.81 ± 1.94	< 0.001

Data presented as Mean±SD, p value >0.05 Not significant, < 0.05 = significant, <0.001 = highly significant.

The table no. 5 showed the mean (± SD) of various sensory nerve variables like latency, amplitude and nerve conduction velocity between overweight male and female group. When compared these variables between overweight male and female group was statistically non-significant (p>0.05) except left ulnar nerve amplitude was statistically significant (p<0.05). Comparison of various sensory nerve variables like latency, amplitude and nerve conduction velocity between overweight male and female group are graphically shown.

Table No. 5: Comparison of various Sensory Nerve Studies in OverweightMale and Female

Nerves	Parameters	Male (n- 120)	Female (n- 60)	P Value
		Mean± SD	Mean± SD	
Right Median Nerve	Latency (ms)	4.70±0.23	4.58±0.28	0.70
	Amplitude (mv)	4.64±0.28	4.60±0.32	0.76
	NCV (m/s)	47.51±1.32	46.76±1.48	0.05
Left Median Nerve	Latency (ms)	4.80±0.23	4.89±0.28	0.71
	Amplitude (mv)	4.29±0.22	4.86±0.32	0.77
	NCV (m/s)	44.17±1.11	42.71±1.26	0.04
Right Ulnar Nerve	Latency (ms)	2.91±0.03	2.91±0.11	0.47
	Amplitude (mv)	4.89±0.13	4.84±0.20	0.46
	NCV (m/s)	47.46±1.93	46.87±1.75	0.14
Left Ulnar Nerve	Latency (ms)	3.78±0.23	2.73±0.28	0.05
	Amplitude (mv)	4.81±0.16	4.39±0.21	0.007
	NCV (m/s)	47.12±1.83	46.97±1.84	0.17
Right Sural Nerve	Latency (ms)	3.76±0.19	3.94±0.24	0.69
	Amplitude (mv)	6.78±0.25	6.19±0.26	0.65
	NCV (m/s)	41.49±1.82	42.12±1.83	0.60
Left Sural Nerve	Latency (ms)	3.72±0.18	3.82±0.24	0.80
	Amplitude (mv)	6.54±0.2	6.29±0.20	0.20
	NCV (m/s)	41.49±1.82	42.12±1.83	0.62

Data presented as Mean±SD, p value >0.05 Not significant < 0.05 = significant, <0.001 = highly significant.

The table no. 6 showed the mean (± SD) of various motor nerve variables like latency, amplitude and nerve conduction velocity between overweight male and female group. When compared these variables between overweight male and female group was statistically non-significant ($p>0.05$) except ulnar nerve NCV was statistically significant ($p<0.05$). Comparison of various motor nerve variables like latency, amplitude and nerve conduction velocity between overweight male and female group are graphically shown.

Table No. 6: Comparison of various Motor Nerve Studies in Overweight Maleand Female

Nerves	Parameters	Male (n- 120)	Female (n- 60)	P Value
		Mean± SD	Mean± SD	
Right Median Nerve	Latency (ms)	4.75±0.28	4.84±0.31	0.65
	Amplitude (mv)	3.46±0.76	3.87±0.71	0.06

	NCV (m/s)	51.21±1.87	51.77±2.31	0.78
Left Median Nerve	Latency (ms)	4.45±0.36	4.25±0.31	0.76
	Amplitude (mv)	3.72±0.69	3.70±0.91	0.07
	NCV (m/s)	51.27±4.15	51.94±2.37	0.38
Right Ulnar Nerve	Latency (ms)	6.12±0.32	6.92±0.35	0.60
	Amplitude (mv)	4.68±0.76	4.78±0.80	0.41
	NCV (m/s)	54.46±1.97	54.63±2.00	0.002
Left Ulnar Nerve	Latency (ms)	6.32±0.32	6.84±0.36	0.58
	Amplitude (mv)	4.14±0.67	4.98±0.82	0.45
	NCV (m/s)	53.56±1.86	54.70±2.11	0.002
Right Common Peroneal Nerve	Latency (ms)	4.76±0.27	4.75±0.29	0.20
	Amplitude (mv)	4.97±0.24	4.33±0.17	0.05
	NCV (m/s)	44.31±2.25	43.87±2.45	0.27
Left Common Peroneal Nerve	Latency (ms)	4.86±0.26	4.36±0.25	0.65
	Amplitude (mv)	4.87±0.27	4.91±0.07	0.20
	NCV (m/s)	44.92±2.51	44.36±3.12	0.41

Data presented as Mean±SD, p value >0.05 Not significant < 0.05 = significant, <0.001 = highly significant.

The table no. 7 showed the mean (± SD) of various sensory nerve variables like latency, amplitude and nerve conduction velocity between obese male and female group. When compared these variables between obese male and female group was statistically non-significant (p>0.05) except median nerve amplitude was statistically highly significant (p<0.001). Comparison of various sensory nerve variables like latency, amplitude and nerve conduction velocity between obese male and female group are graphically shown.

Table No 7: Comparison of various Sensory Nerve Studies in Obese Male andFemale

Nerves	**Parameters**	**Male (n-70)**	**Female (n-50)**	**P Value**
		Mean± SD	**Mean± SD**	
Right Median Nerve	Latency (ms)	5.12±0.26	5.90±0.69	0.37
	Amplitude (mv)	5.04±0.39	5.12±0.66	0.0008
	NCV (m/s)	46.13±2.00	50.62±2.64	0.92
Left Median Nerve	Latency (ms)	5.19±0.25	5.82±0.96	0.40
	Amplitude (mv)	4.95±0.27	5.12±0.74	0.0007
	NCV (m/s)	46.25±1.89	50.72±2.70	0.94
	Latency (ms)	3.89±0.15	5.83±0.70	0.21

Right Ulnar Nerve	Amplitude (mv)	4.50±0.04	5.80±0.36	0.94
	NCV (m/s)	45.98±2.35	49.21±3.43	0.87
Left Ulnar Nerve	Latency (ms)	3.87±0.04	5.26±0.84	0.27
	Amplitude (mv)	4.18±0.05	5.92±0.40	0.31
	NCV (m/s)	45.78±2.36	49.67±4.00	0.84
Right Sural Nerve	Latency (ms)	3.84±0.16	5.03±0.89	0.91
	Amplitude (mv)	6.66±0.21	8.15±0.82	0.90
	NCV (m/s)	40.12±1.21	42.69±1.88	0.15
Left Sural Nerve	Latency (ms)	3.94±0.14	5.12±0.70	0.67
	Amplitude (mv)	6.72±0.20	8.43±0.83	0.40
	NCV (m/s)	40.31±1.14	42.58±2.06	0.22

Data presented as Mean±SD, p value >0.05 Not significant < 0.05 = significant, <0.001 = highly significant.

The table no. 8 showed the mean (± SD) of various motor nerve variables like latency, amplitude and nerve conduction velocity between obese male and female group. When compared these variables between obese male and female group was statistically non-significant (p>0.05) except median and ulnar nerve NCV was statistically significant (p<0.05). Comparison of various motor nerve variables like latency, amplitude and nerve conduction velocity between obese male and female group are graphically shown.

Table No. 8: Comparison of various Motor Nerve Studies in Obese Male andFemale

Nerves	Parameters	Male (n-70)	Female (n-50)	P Value
		Mean± SD	Mean± SD	
Right Median Nerve	Latency (ms)	5.15±0.30	6.86±1.04	0.59
	Amplitude (mv)	3.11±0.11	4.28±0.81	0.96
	NCV (m/s)	50.24±2.24	54.68±4.08	0.01
Left Median Nerve	Latency (ms)	5.12±0.30	6.36±0.89	0.74
	Amplitude (mv)	3.22±0.10	4.28±0.80	0.97
	NCV (m/s)	50.22±2.31	54.62±3.99	0.01
Right Ulnar Nerve	Latency (ms)	6.23±0.33	8.63±0.86	0.76
	Amplitude (mv)	4.95±0.45	6.29±1.82	0.15
	NCV (m/s)	54.46±2.87	55.24±3.42	0.04
Left Ulnar Nerve	Latency (ms)	6.452±0.35	8.83±0.99	0.39
	Amplitude (mv)	4.85±0.41	6.45±1.61	0.30
	NCV (m/s)	53.36±2.88	55.73±3.60	0.03
	Latency (ms)	4.42±0.17	6.25±0.72	0.08

Right Common Peroneal Nerve	Amplitude (mv)	4.15±0.25	6.24±0.76	0.19
	NCV (m/s)	42.75±1.84	45.42±2.50	0.89
Left Common Peroneal Nerve	Latency (ms)	4.27±0.17	6.64± 0.80	0.88
	Amplitude (mv)	4.42±0.26	3.40±0.86	0.51
	NCV (m/s)	42.61±2.00	45.63±2.48	0.89

Data presented as Mean±SD, p value >0.05 Not significant < 0.05 = significant, <0.001 = highly significant.

The table no. 9 shows the correlation of fasting blood sugar level mean ±SD 158.85±17.33 with three variables (latency, amplitude and NCV) of various sensory nerve conduction studies in cases. Fasting blood sugar level positively correlated with all variables but negatively correlated with left ulnar nerve and sural nerve in cases. No statistically significant (p>0.05) correlations were found of fasting blood sugar level with any sensory nerve conduction studies in cases.

Table No. 9: Correlation between Fasting blood sugar level and Sensory Nerve Conduction studies in Cases (n-300)

			Latency (ms)			Amplitude (mv)			NCV (m/s)		
			Mean± SD	**r-Value**	**p-Value**	**Mean± SD**	**r-Value**	**p-Value**	**Mean± SD**	**r-Value**	**p-Value**
Fasting Blood Glucose Level 158.85±17.33	Median Nerve	Right	4.72±0.26	0.035	0.36	4.81±0.28	0.066	0.13	46.01±1.90	0.017	0.65
		Left	4.82±0.45	0.042	0.23	4.82±0.23	0.068	0.13	46.23±1.92	0.013	0.76
	Ulnar Nerve	Right	3.84±0.26	0.010	0.74	4.36±0.23	0.007	0.80	45.59±2.43	0.035	0.44
		Left	3.69±0.24	0.010	0.71	4.64±0.21	-0.044	0.08	45.59±2.56	0.034	0.40
	Sural Nerve	Right	3.81±0.20	0.015	0.61	6.37±0.26	-0.046	0.10	40.95±2.13	0.037	0.26
		Left	3.91±0.17	0.014	0.65	6.67±0.26	-0.031	0.44	40.20±2.22	0.043	0.30

Data presented as Mean±SD, p value >0.05 Not significant < 0.05 = significant, <0.001 = highlysignificant.

The table no. 10 shows the correlation of postprandial blood sugar level mean ±SD 193.85±20.91 with three variables (latency, amplitude and NCV) of various sensory nerve conduction studies in cases. Postprandial blood sugar level positively correlated with all variables except latency of right ulnar and sural nerve but negatively correlated with amplitude of left ulnar nerve in cases No statistically significant (p >0.05) correlation were found of postprandial blood sugar level with any sensory nerve conduction studies in cases.

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Table No. 10: Correlation between Post Prandial blood sugar level and SensoryNerve Conduction studies in Cases (n-300)

			Latency (ms)			Amplitude (mv)			NCV (m/s)		
			Mean± SD	**r-Value**	**p-Value**	**Mean± SD**	**r-Value**	**p-Value**	**Mean± SD**	**r-Value**	**p-Value**
Post Prandial blood sugar level 193.85 ±20.91	Median Nerve	Right	4.72±0.28	0.019	0.60	4.78±0.26	0.06	0.10	46.10±2.03	0.02	0.50
		Left	4.68±0.31	0.020	0.54	4.61±0.32	0.077	0.10	46.23±2.09	0.024	0.55
	Ulnar Nerve	Right	3.42±0.17	-0.010	0.73	4.66±0.21	0.024	0.45	45.59±2.28	0.050	0.21
		Left	3.41±0.24	0.0061	0.14	4.43±0.24	-0.030	0.40	45.59±2.39	0.050	0.22
	Sural Nerve	Right	3.13±0.20	-0.0061	0.10	6.34±0.25	0.0051	0.78	40.95±2.23	0.081	0.10
		Left	3.92±0.18	0.010	0.73	6.67±0.24	0.026	0.50	40.30±2.22	0.074	0.11

Data presented as Mean±SD, p value >0.05 Not significant < 0.05 = significant, <0.001 = highly significant.

Discussion

Research literature support a relationship between BMI and TG, and the relationship of blood lipids and body fat distribution has been under discussion over the past few decades. [8] Body fat and blood lipids have been observed to be key determinants of metabolic disorders, like cardiovascular diseases (CVD), diabetes, dyslipidemia, hypertension, hyperinsulinemia and elevated serum uric acid. [9]

Dyslipidemia, a well-known risk factor for cardiovascular manifestations, is mostly observed in the population of the Asian continent. People with T2DM have an increased cardiovascular morbidity and mortality, and are affected more by CVD compared with non-diabetics. Prompt recognition and management of DM associated dyslipidemia might be one step in controlling the risk of CVD. [10]

Obesity, which is considered to be potentially linked with abnormal lipids and poor cardiovascular outcomes, is becoming a highly prevalent condition in Pakistan. The current study intends to evaluate the correlation between BMI and lipids in patients with T2DM in a Khyber Pakhtunkhwa population; an area from where information in this regard is uptill now not available.

In this study, 300 diabetic patients attending the outpatient department (diabetic clinic) at the Northwest General Hospital and Research Centre, Peshawar were randomly selected for the study. The participants were already diagnosed as having T2DM and were under treatment at the diabetic clinic. The patients included 180 (60%) men and 120 (40%) women. The female population was more than that of the male counterpart. This compares

well with a study on WHO global data which stated that the prevalence ratio of DM between men and women varies markedly, with no consistent trend. [11]

Age, BMI, HbA1c and lipids values were stratified in gender wise manner; in male patients mean values of BMI and TC were slightly lower and that of TG and HDL-C were higher compared to female patients but the mean differences were not statistically significant which is consistent with previous studies results in TG and TC. [12] However the mean difference of LDL-C in male and female patients was statistically significant. This is in agreement to the findings of a study which showed similar result in LDL-C values between men and women, although the mean values of TG and HDL-C were differ significantly whereas TC was comparable in both genders. [13] In a study by Omotoye FE et al. [14] which showed that mean TC, TG, and LDL-C were elevated more among the T2DM female patients than males.

Following the WHO, ADA and NCEP ATP III criteria for BMI, HbA1c and Lipid profile values. The most common lipid abnormality was seen in TGs with 69% of the study participants, followed by the LDL-C (54.1%). This result is in agreement with published studies in Northwest Ethiopia (63.5%), Hyderabad-India (60%) and Sudan (48.8%). [15] These findings may be due to the increased secretion of LDL-C by the liver and slow removal of TGs rich lipoproteins, as well as raised levels of substrates for TG production from augmented mobilization of free fatty acid (FFA) from adipose tissue in people with diabetes. [16] High TG levels are a prominent lipid abnormality in T2DM and also occur in individuals with pre-diabetes states. A fasting TG level of > 150 mg/dl is one of the benchmarks for characterizing peoples at high risk for CVD and T2DM. Our results showed raised LDL-C and low HDL-C levels in DM patients. These results are in agreement to Asian Pacific Cohort Studies Collaboration. [17] These findings are thought to be due to differences in genetic makeup, differences in life style and the management of specific population of DM being studied.

This study showed that in the study population mean BMI was not different in males and females (29.24 vs. 29.32) kg/m^2. These BMI results were higher than the previously published mean BMI of participants from an urban community in Yemen (23.9 ± 5.1) kg/m^2 and 21.8 ±8.9) kg/m^2 in females and males, respectively [18] and also the work of Al- Sharafi which showed that the overall mean BMI was considerably higher in females than in males (28 vs. 25.4). [19]

In our study two patients were underweight (17.5 kg/m^2) which was considered normal. Whereas, 10.5% were normal weight, 15.4% were overweight and obese (BMI>25) accounted for 74.1% of the total investigated population with diabetes mellitus. This figure was lower than that of a study by Bansal P et al. [20] but higher than the findings of a previous study in Yemen that overweight and obesity accounted only for 26.2% of patients with T2DM aged 20-65. [21] The international data Analysis with reference to the relationship between BMI and both morbidity and mortality recommended that the

association of BMI with most diseases was rather continuous and commonly, women had a higher mean BMI than men. With regards to relationship between BMI and lipid profile this study showed that BMI had a negative correlation (r =-0.125, *p*=0.029) with HDL-C value while the others parameters like TC (r=-0.048, *p*=0.300), TG (r=-0.004, *p*=0.890), LDL-C (r=-0.017, *p*=0.684) did not reveal any correlation with BMI. Our results are different from other similar studies. Results of a study conducted in Korea described that there was a positive correlation between BMI with TC and LDL-C respectively; whereas a study from India illustrated the existence of only BMI vs. LDL-C correlation. A weak negative correlation of HDL-C with BMI was also reported by a similar study conducted in Nigeria. Likewise in a study by Shamai et al [22] there was an association between BMI, HDL-C and TG.

In this study T2DM patient with normal BMI when compared to overweight and obese BMI did not show significant differences in the mean values of TC, TGs and LDL-C except HDLC. In a study by Yadav NK et al., [23] reported that obese T2DM patients, in comparison with on-diabetic obese control patients revealed statistically significant increase in the levels of TC, TGs, LDL-C whereas HDL-C levels in the two groups did not show statistically significant difference. Another study on Iraqi diabetic premenopausal women showed that high BMI is consistently coupled with abnormal lipid profile marked by elevated TGs and LDL-C, and low HDL-C. Comparison of HDL-C levels showed that Group one individuals had the most favorable values (62.61 ± 41.56). This was followed by individuals in Group two (48.63 ± 24.85) and Group three (46.53 ± 28.64). This difference in HDL-C levels across the three groups was significant ($p < 0.036$). Our results are consistent with a study by Bora K et al. [24]

Both body fat and lipid parameters have been revealed to be the significant predictors for metabolic disturbances including diabetes, dyslipidemia, hypertension, hyperinsulinemia, and cardiovascular diseases. Lipid profiles association is reported with lifestyle, age, intra-abdominal adiposity, Obesity and BMI.

Limitations of the study: The contribution of diet and socio-economic factors in influencing lipid profile and obesity were not considered. In addition, a randomly drawn larger sample would have been more advantageous. Constraint resources and lack of time was the chief causes for these limitations**.** Similarly, waist circumference (WC) was not measured.

Conclusion

This study showed high percentage of abnormal TG. LDL-C level was observed to be significantly higher in female. A significant negative relationship between HDL-C and BMI level was also seen, while the correlation between LDL-C and BMI was observed to be insignificant. HDL-C was found significantly higher in patients with normal BMI. These results are imperative as they back up that there is modest impact of BMI on lipid profile. Diabetic patients are more likely to have dyslipidemia which is a key determinant for atherosclerosis and CVD. Normal BMI significantly improves

dyslipidemia in T2DM patients. Further studies with large sample size are needed to identify the causes of obesity that would help in better understanding of its influence on lipid profile.

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