

Lipid Profile in Normoglycemic Offspring of Patient with T2dm on Graded Exercise

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Abstract

Objectives: The primary causes of Type 2 Diabetes Mellitus (T2DM) are largely unknown but increase in lipid Profile (Total Cholesterol, Triglyceride, HDL and LDL) has been reported to be a risk factor for the T2DM through the alteration of Lipid Profile (LP). However, it is uncertain if exercise could influence the occurrence of T2DM in offspring of diabetic parents. Therefore this study was designed to assess the effect of exercise on lipid Profile on offspring of T2DM parents compared with offspring of non-diabetic parents.

Design: This study involved purposive selection of 42 offspring of T2DM parents attending University College Hospital, Ibadan and 53 offspring of non-diabetic parents who are undergraduate students of the University of Ibadan, Nigeria. Participants were randomly assigned into four groups; 27 Normal-weight Offspring of Non-Diabetic Parents (NONDP), 21 Normal-weight Offspring of Diabetic Parents (NODP), 26 Overweight Offspring of Non-Diabetic Parents (OONDP) and 21 Overweight Offspring of Diabetic Parents (OODP). Each participant followed a protocol of graded exercise using tummy trimmer everyday spending 30-45 minutes daily for 24 weeks. Blood samples were obtained after an overnight fasting for determination of lipid profile levels using standard methods at baseline, six week, 12 week, 18 week and 24 week, respectively. The LP determined from the fasting liquid Profile was measured spectrophotometrically using standard laboratory kits supplied by BIOLABO, France. Data were analyzed using descriptive statistic and repeated ANOVA with significant at $p < 0.05$.

Results: After exercise, there were reductions in total cholesterol (mg/dL) (NONDP: from 176.18 ± 10.78 to 166.85 ± 10.22 , NODP: from 120.91 ± 5.87 to 114.09 ± 5.78 $p = NS$. OONDP: 156.10 ± 6.37 to 147.73 ± 6.14 ($p < 0.05$), OODP: 131.29 ± 8.29 to 123.90 ± 10.33 $p = NS$). HDL in NONDP which was 41.67 ± 2.95 increased to 46.07 ± 3.06 , in NODP 39.81 ± 4.50 to 41.81 ± 4.29 in OONDP 43.27 ± 2.86 to 50.35 ± 2.47 $p = NS$, and in OODP 32.95 ± 3.57 to 42.57 ± 3.82). There were no significant changes in total cholesterol, triglyceride, HDL and LDL and between baseline and at 24 weeks.

Conclusions: Graded exercise reduced lipid profile in all the groups. The clinical importance of graded exercise in prevention of diabetes mellitus among offspring of diabetic parents looks promising.

Keywords: Graded exercise, Diabetic parents' offspring, Lipid Profile.

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I. Introduction

Diabetes mellitus, commonly known as diabetes, is a disorder of intermediary carbohydrate, protein and lipid metabolism. It is characterized by hyperglycemia, glucosuria, polydipsia, polyuria, polyphagia and weight loss. It is usually associated by secondary alterations in glucose, fat and protein metabolism, leading to many biochemical disorders. It is characterized by peripheral insulin resistance, impaired regulation of hepatic glucose production with declining β -cell function and eventually leading to β -cell failure [1]. Type 2 Diabetes Mellitus (Type 2DM) is characterized by a combination of peripheral insulin resistance and inadequate insulin secretion by pancreatic beta cells. Insulin resistance has been attributed to elevated levels of free fatty acids and pro-inflammatory cytokines in plasma, leads to reduced glucose transport into muscle cells, elevated hepatic glucose production, and pronounced break down of fat.

Researchers have found that obesity and diabetes are inter-connected. Individuals who are obese are at high risk of developing T2DM, particular if a close family member is affected with T2DM. Researchers have not yet discovered a specific gene that causes obesity although, several genes are considered to play a role. There seems to be a connection between abdominal fat and diabetes, hence anything that will reduce abdominal fat will likely reduce diabetes. Exercise has been known to ameliorate the effect of diabetes by improving

insulin sensitivity and reduced the lipid level. It is the aim of this to work to study the effect of exercise on lipid profile of normoglycemic offspring of patients with type 2 DM.

II. Methods

Experimental interventional study was carried out in which blood sample was collected from offspring of patients with type 2 diabetes mellitus and normoglycemic offspring of non-diabetic parents. The parents of the test group were attending the medical out-patient clinic (MOP) of the University College Hospital (UCH), Ibadan and Catholic Hospital Oluyoro, Oke-Ofa, Ibadan, South Western, Nigeria. The normoglycemic offspring of non-diabetic parents aged 25 years and above were randomly selected from general population of Ibadan Community, Ibadan, and South-Western, Nigeria and undergraduate students of University of Ibadan. These are normoglycemic offspring of non-diabetic with normal weight that served as control subjects.

The participants were divided into four groups, N=42 as follows:

A – Overweight / Obese offspring of DM parents (OODP).

B – Normal weight / Normal Body Mass Index (BMI) offspring of DM parents (NODP).

C – Overweight / Obese offspring of non-diabetic parents (OONDP).

D – Normal BMI / weight offspring of non-diabetic parents (NONDP)

The study was approved by the University of Ibadan Teaching Hospital Ethical Committee (UI/UCH joint IRB) and Catholic Hospital Ethical Committee prior to its implementation. The parameters measured include: Total Cholesterol (TC); Low Density Lipoprotein (LDL); High Density Lipoprotein (HDL) and Triglycerides (TG) 10ml of venous blood specimen was obtained from each subject into plain bottles. Separation of plasma at centrifugal force of 3,000rpm was carried out at IMRAT (Institute of Medical Research and Training) of the College of Medicine, University of Ibadan. The plasma so obtained was stored at temperature not exceeding – 40°C for lipid profile estimation, each in a refrigerator at UCH Pharmacology Department until used for the determination of biochemical profile. Data was got from venous blood sampling and by measurement of anthropometric variables. This is repeated as follows: Baseline measurement and after 6, 12, 18 and 24 weeks. Total cholesterol level was measured spectrophotometrically using standard laboratory supplied by BIOLABO, France. Cholesterol esters in the presence of cholesterol esterase cholesterol and free fatty acids are separated. The cholesterol formed reacts with oxygen in the presence of cholesterol oxidase to form cholesten-4-one-3 and hydrogen peroxide. The hydrogen peroxide formed reacts with phenol and 4-amino-antipyrine in the presence of peroxidase to give aminoneimine (pinkish in colour) and water. The intensity of the pink/red colour formed is proportional to the cholesterol concentration. The procedure employed was as follows:

The reagent was prepared by adding 5ml of the buffer (1.75mool/L Amino-2-methyl-2-propanol-1) to 5ml of the Chromogen mixture (76umol/L 0-Cresolphtalein Complexon, 3.36mmol/L/L 8 – Hydroxy-Quinoline, 25mmol/L HCl) and allowed to stand for an hour at room temperature. The reagent solution was prepared by adding equal volumes of the buffer and 5mmol/L Chloro-4-phenol) and the enzyme mixture (100U/L Cholesterol oxidase, 70U/L Cholesterol esterase, 1200U/L peroxidase, 2mmol/L Cholic acid Sodium salt, 0.3mmol Amino antipyrine) and allowed to stand for 5 – 10 minutes while mixing gently at room temperature. To 10µL of each test sample or standard (5.17mmol/L Cholesterol) was added 1ml of the reagent mixture. This was incubated at 37°C for 5 minutes. The absorbance of the mixture was taken against the blank at a wavelength of 500nm. The blank was made up of 10µL of distilled water and 1ml of the reagent mixture. The cholesterol concentration was determined as follows.

$$\text{Total cholesterol concentration (mg/dl)} = \text{Absorbance}_{\text{sample}} \times \frac{\text{Standard concentration}}{\text{Absorbance standard}}$$

HDL cholesterol level was measured spectrophotometrically using standard lab kits supplied by BIOLABO, France. Low density lipoproteins (LDL) contained in serum are precipitated by addition of phosphotungstic acid and magnesium chloride. High density lipoproteins (LDL) which remains in the supernatant (obtained after centrifugation) react with the cholesterol reagent and proportionally with the cholesterol standard.

The procedure followed was as follows: equal volumes of the serum and reagent mixture (13.9mmol/L phosphotungstic acid and 570mmol/L magnesium chloride) were mixed together and allowed to stand for 10 minutes at room temperature. The reaction mixture was then centrifuged for 10 minutes at 4000rpm to get a clear supernatant. This supernatant was used as sample to get the HDL cholesterol concentration in the serum sample. 1000ul of the Cholesterol reagent was added to test tubes labeled blank, standard and sample containing 50µl water, 50µl of the cholesterol standard and 50µl of the sample respectively. This was well mixed and incubated for 10mins at 37°C. The absorbance of the end sample against the blank was taken at 505nm.

$$\text{HDL cholesterol concentration (mg/dl)} = \frac{\text{Absorbance}_{\text{sample}} \times \text{Standard concentration}}{\text{Absorbance}_{\text{standard}}}$$

Triglycerides level was measured spectrophotometrically using standard tab kits supplied BIOLABO, France Triglycerides in the presence of lipase form glycerol free fatty acids. Glycerol formed reacts reversibly with adenosine triphosphate (ATP) in the presence of glycerol lipase to form glycerol – 3 – phosphate and ADP. The glycerol 3 phosphate also reacts reversibly with oxygen in the presence of glycerol -3- phosphate oxidase to form dihydroxyacetone phosphate and hydrogen peroxide. The hydrogen peroxide then reacts with chlorophenol and amino antipyrine in the presence of peroxidase to form quinoneimine (pink) and water. The intensity of the pink/red colour formed is proportional to the triglyceride concentration.

The reagent solution was prepared by adding equal volumes of the buffer (3.5mmol/L chloro-4-phenol, 6mmol/L Magnesium chloride 100mmol/L PIPES) and the enzyme mixture 500U/I Lipase, 1800U/I peroxidase, 400U/I Glycerol 3-phosphate oxidase, 1000U/I Glycerol (lipase. 0.30mmol 4 Amino antipyrine. 1.72mmol/I Adenosine triphosphate Na) and allowed to stand for 5 – 10minutes. To 10 μ L of each test sample of standard (Glycerol 200mg/dl) was added 1ml of the reagent mixture. This was incubated at 37°C for 5minutes. The absorbance of the mixture was taken against the blank at a wavelength of 500nm. The blank was made up of 10 μ L of distilled H₂O and 1ml of the reagent mixture. The triglyceride concentration was determined as follows. Triglyceride concentration (mg/dl) = $\frac{\text{Absorbance}_{\text{sample}} \times \text{Standard concentration}}{\text{Absorbance}_{\text{standard}}}$

Heights of participants were taken using standard hospital adult vertical rule with sliding arms which had been recalibrated and certified by a Biomedical Engineering technician prior to use. The study subject stood erect, upright and bare-footed. Those who had extra clothes such as coats and sweater removed them while Omron equipment measurements of BMI were being taken. Body mass index (BMI) reading values for the subject were read off as displayed on the screen of Omron equipment (reliability and reproducibility index \pm 0.01%). The BMI values were used to group subject into four categories. Underweight – BMI < 18.5kg/m² Normal weight – BMI = 18.5 to 24.9kg/m² Overweight – BMI = 25-29.9kg/m² Obese – BMI = >30.0kg/m²

Omron fat estimator was used to measure the BMI. The subject stood uprightly bare-footed put on light clothing. The subject held his stretched hands forward as if he was riding a motor-bike. BMI readings were read off as displayed on the screen. The readings were recorded in the recording book. Tummy trimmer, a portable, aerobic exercise, lightweight equipment (European Home Choice Company, Lagos, Nigeria) was selected for the study. It is an in-door anaerobic equipment. It is compact and can fit right in the subject's brief case. During each phase of exercise the Tummy trimmer, a portable lightweight equipment, is held at the two handles and the sole of the two feet are put inside the pedal rest while the subject assume different positions. The subject will then pull the tummy trimmer's spring towards himself or herself either while lying flat or sitting up on the floor or carpeted hard surface. Subject sits up with leg straight, leans his or her body backwards until completely lying back with head on floor. He/she returns to sitting position in harmonic fashion. The subject was advised to start slowly and work up to repetition as she/he feels comfortable with harmoniously.

The subject was advised to lie flat on floor, extend his/her legs straight up in the air. He will be keeping his/her back on the floor and raise lower legs without bending them. The subject was advised to sit erect with legs straight horizontally, he/she raises handle to tummy height using arms only. Finally, subject was advised to lie flat on the floor while he/she bends knees up to his/her chest. He/she makes a circular motion push feet up and then round towards the floor again. The different positions were observed for exercise period of 30 to 40 minutes (a video clip of the exercise procedure was shown to the subject before the commencement of the exercise).

Each subject was advised as follows:

- (1) He/she to undergo the 4 phases of exercise between 30 and 40 minutes daily (either in the mornings or evenings).
- (2) He/she to contact the researcher on cell phone anytime when he/she has any problems with the unit.
- (3) There were regular cell phone calls made to each of the subjects by the research assistant to ensure compliance with exercise schedule.
- (4) The research assistant called them on cell phone and sent s.m.s (Short Message Service) to them to keep return appointments every six weeks. This was done one or two days before appointment schedule.

Statistical analysis was carried out by using the ANOVA. The data obtained was analyzed using computer statistical programme package SPSS version 15.0 Probability value of **P** less than 0.05 was considered statistically significant.

III. Results

Lipid profiles of the subjects with variations in intra-group and extra-group measurements are shown below. This is shown in tables 1a to 1c, 2a to 2c and Figures 1 to 4. There were reductions in the levels of cholesterol, TTG and LDL in all the groups. There was increased in the levels of HDL in all the groups after six months of graded exercise. Tables 1a to 1c show the comparison before exercise. Table 1a showed comparison in lipid profile between OODP and NODP which showed reduction in TTG from 166.85±10.22mg/dl to 103.00±9.74mg/dl after six months of exercise and the reduction is statistical significant (p<0.05). The reduction of NODP from 78.09±7.50mg/dl to 75.67±7.34mg/dl is statistically significant (p<0.05). Table 1b showed the comparison in lipid profile of OODP and OONDP before and after six months of exercise. The total cholesterol reduction is statistically significant (p<0.05). Table 1c showed comparison of lipid profile of OODP and NONDP before and after exercise where the reductions in total cholesterol and LDL were statistically significant (p<0.05).

Tables 2a- 2c show comparison after 24 weeks of exercise. Table 2a showed comparison of lipid profile in NODP and OONDP before and after six months of exercise where the reduction of total cholesterol and LDL are statistically significant (p<0.05).Table 2b showed comparison of NODP and NOND before and after exercise where Total cholesterol and LDL reductions were statistically significant (p<0.05). However, the increases in levels of HDL in all groups were not statistically significant.Table 2c showed comparison of OONDP and NONDP before and after exercise where Total cholesterol, TTG and LDL reductions and increase in HDL were not statistically significant.Figure 1 showed the comparison in the total cholesterol in offspring of diabetic and non-diabetic parents. The reductions in NONDP and OONDP are statistically significant (p<0.05). Figure 2 showed the comparison of TG in all the groups where the reduction in NODP and NONDP were statistically significant (p<0.05) after six months of exercise.Figure 3 showed the comparison of HDL in all the groups where the increase in all groups were not statistically significant after six months of exercise.Figure 4 showed the comparison of LDL in all the groups. The reductions in NONDP and OONDP were statistically significant (p<0.05) after six months of exercise.

Table 1a to 1c: Comparison of Lipid Profile before and after 24 weeks of exercise.

Table 1a: Comparison of lipid profile of OODP and NODP before and after 24 weeks of exercise.

	OODP before exercise	NODP before exercise	p	OODP after exercise	NODP after exercise	p
Cholesterol	131.29±8.29	120.91±5.87	0.412	123.90±10.33	114.09±5.78	0.400
TTG	166.85±10.22	78.09±7.50	0.014*	103.00±9.74	75.67±7.34	0.020*
HDL	32.95±3.57	39.81±4.50	0.187	42.57±3.82	41.81±4.29	0.880
LDL	71.14±7.29	61.4±2.90	0.386	66.05±7.07	57.9±3.20	0.404

All values are mean±SE *Significant at p<0.05

Table 1b: Comparison of lipid profile of OODP and OONDP before and after 24weeks of exercise

	OODP before exercise	NODP before exercise	p	OODP after exercise	NODP after exercise	p
Cholesterol	131.29±8.29	156.10±6.37	0.041*	123.90±10.33	147.73±6.14	0.034*
TTG	166.85±10.22	103.27±6.14	0.628	103.00±9.74	94.58±5.70	0.438
HDL	32.95±3.57	43.27±2.86	0.038*	42.57±3.82	50.35±2.47	0.109
LDL	71.14±7.29	86.62±6.13	0.144	66.05±7.07	78.92±5.66	0.162

All values are mean±SE *Significant at p<0.05

Table 1c: Comparison of lipid profile of OODP and NONDP before and after 24weeks of exercise

	OODP	NODP	p	OODP after	NODP	p
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	before exercise	before exercise		exercise	after exercise	
Cholesterol	131.29±8.29	176.18±10.78	0.000*	123.90±10.33	166.85±10.22	0.000*
TTG	166.85±10.22	111.14±8.73	0.854	103.00±9.74	103.37±7.94	
0.983						
HDL	32.95±3.57	41.67±2.99	0.076	42.57±3.82	46.07±3.06	
0.464						
LDL	71.14±7.29	95.19±9.63	0.023*	66.05±7.07	85.56±7.66	
0.034*						

All values are mean±SE *Significant at p<0.05

Table 2a to 2c: Comparison of lipid Profile before and after 24weeks of exercise.

Table 2a: Comparison of lipid profile of NODP and OONDP before and after 24weeks of exercise

	NODP before exercise	OONDP before exercise	p	NODP after exercise	OONDP after exercise	p
Cholesterol	120.91±5.87	156.10±6.37	0.004*	114.09±5.78	147.73±6.14	0.003*
TTG	78.09±7.50	103.27±6.14	0.035*	75.67±7.34	94.58±5.70	
0.089						
HDL	39.81±4.50	43.27±2.86	0.482	41.81±4.29	50.35±2.47	
0.079						
LDL	61.4±2.90	86.62±6.13	0.020*	57.9±3.20	78.92±5.66	
0.025*						

All values are mean±SE *Significant at p<0.05

Table 2b: Comparison of lipid profile of NODP and NONDP before and after 24weeks of exercise

	NODP before exercise	NONDP before exercise	p	NODP after exercise	NONDP after exercise	p
Cholesterol	120.91±5.87	176.18±10.78	0.000*	114.09±5.78	166.85±10.22	0.000*
TTG	78.09±7.50	111.14±8.73	0.006*	75.67±7.34	103.37±7.94	
0.013*						
HDL	39.81±4.50	41.67±2.99	0.703	41.81±4.29	46.07±3.06	
0.373						
LDL	61.4±2.90	95.19±9.63	0.002*	57.9±3.20	85.56±7.66	
0.003*						

All values are mean±SE *Significant at p<0.05

Table 2c: Comparison of lipid profile of OONDP and NONDP before and after 24weeks of exercise

	NODP before exercise	OONDP before exercise	p	NODP after exercise	OONDP after exercise	p
Cholesterol	156.10±6.37	176.18±10.78	0.077*	147.73±6.14	166.85±10.22	0.068
TTG	103.27±6.14	111.14±8.73	0.477*	94.58±5.70	103.37±7.94	
0.395						
HDL	43.27±2.86	41.67±2.99	0.728	50.35±2.47	46.07±3.06	
0.345						
LDL	86.62±6.13	95.19±9.63	0.386*	78.92±5.66	85.56±7.66	
0.440						

All values are mean±SE

*Significant at p<0.05

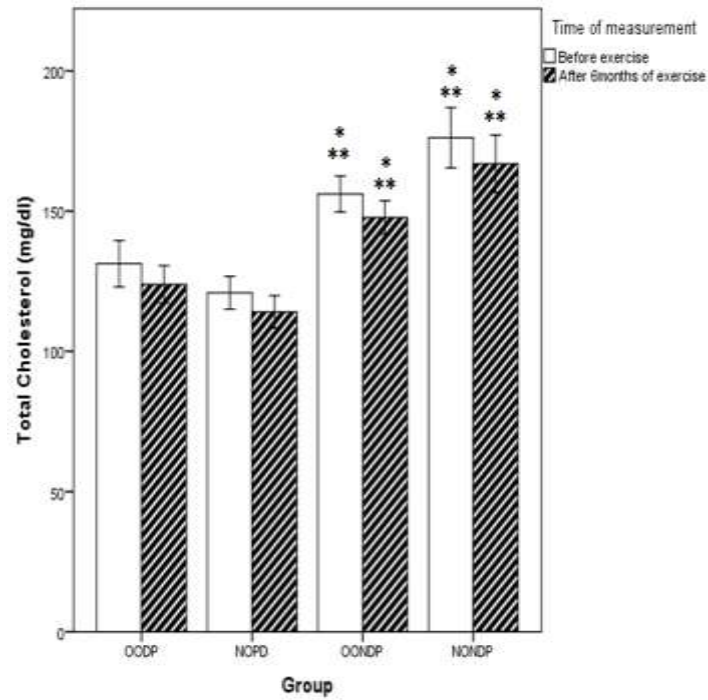


Figure 1 showed the comparison in the total cholesterol in offspring of diabetic and non-diabetic parents.

All values are mean±SE.

*Significant at $p < 0.05$.

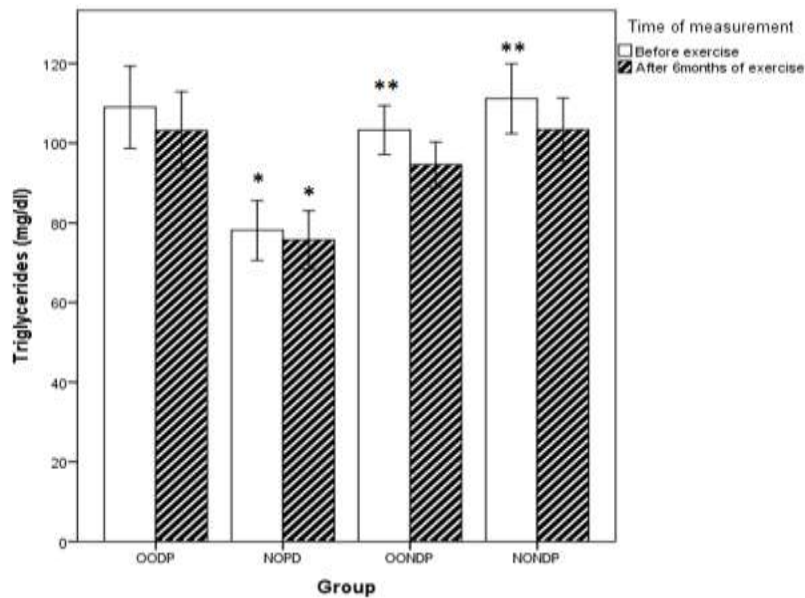


Figure 2 showed the comparison of TG in all the groups.

All values are mean±SE

*Significant at $p < 0.05$

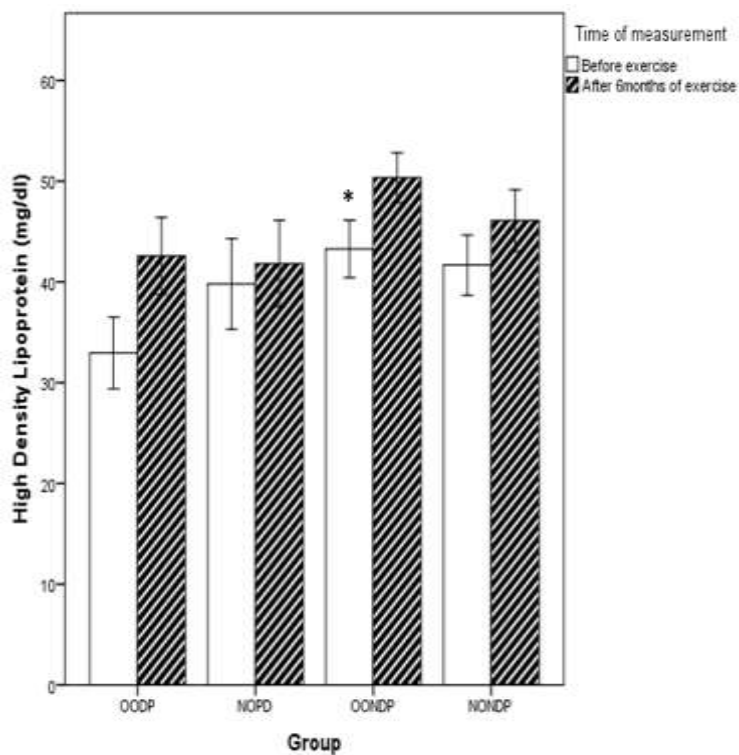


Figure 3 showed the comparison of HDL in all the groups.

All values are mean \pm SE
 *Significant at $p < 0.05$

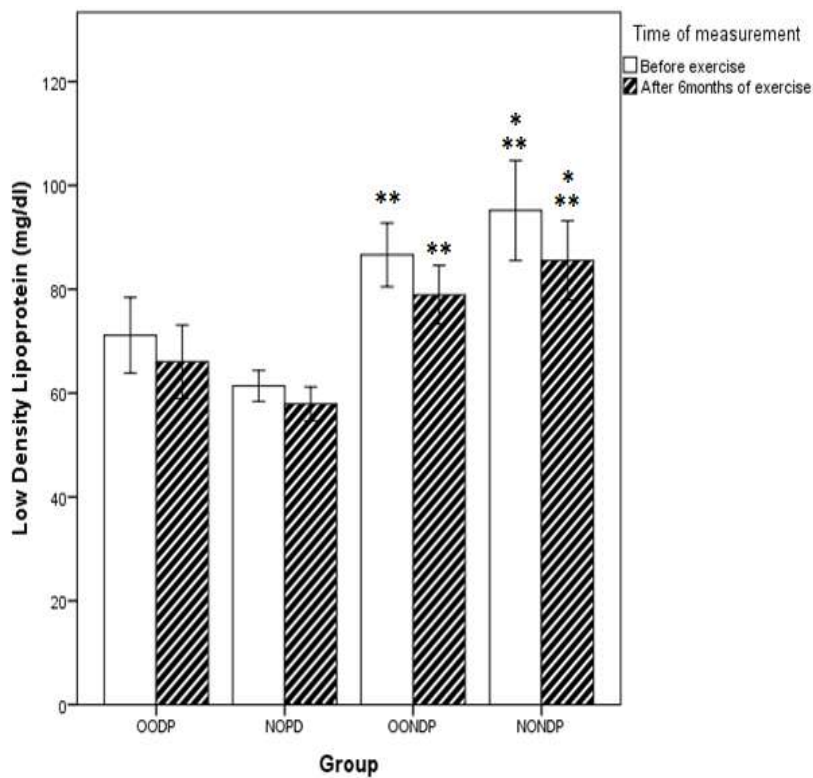


Figure 4: showed the comparison of LDL in all the groups.

All values are mean \pm SE
 *Significant at $p < 0.05$

IV. Discussion

From the results of the Lipid Profile, the total cholesterol reduced in all the groups after six months of exercise (24 weeks). The triglyceride was also reduced in all the groups after six months of tummy trimmer exercise. The HDL (good fat) increased in all the groups after six months of exercise. The LDL (bad fat) reduced in all the groups after six months of exercise. The extensive works done by Mathur et. Al. (1960) found out that when blood cholesterol was increased in level it is associated with atherosclerosis, diabetes mellitus, myocardial infection and angina pectoris. Hence, exercise which reduced the level of cholesterol will prevent development of diabetes mellitus in the offspring for example, regular and intensive exercise will eventually increase the level of HDL which is the cholesterol that is good for the heart. A single bout of prolonged dynamic exercise reduces postprandial chylomeron tryglyceridemia in type 2 diabetic patients (3). Furthermore, regular exercise has been shown to reduce fasting triglyceride concentration in some patients (3). Hannele et al, (1983) studied the effect of body composition and maximal aerobic power on insulin sensitivity. They found that body sensitivity to insulin is directly related to the muscle mass and inversely proportional to adiposity. They also reported that one factor contributing to decrease in insulin sensitivity is obesity which occurred during physical inactivity. Therefore in this present study, reduction in the lipid profile after six months of exercise confirm the importance of physical activity in improving insulin sensitivity in offspring of T2 diabetes patients. Insulin resistance may play a pivotal role in the development of diabetic dyslipidemia by influencing several factors such as insulin resistance and type 2 diabetes, increased efflux of free fatty acids into the liver (5,6). Hepatic lipase activity is responsible for hydrolysis of phospholipids in LDL and HDL particles and lead to smaller and denser LDL particles and decrease in HDL (7,8) and so leading to increase in serum lipids which is seen in obesity.

Lifestyle interventions such as diet, physical activity weight loss, and smoking cessation are integral part of any diabetes management plan. Epidemiologic and intervention studies have shown significant improvements in the features of diabetic dyslipaemia such as medical nutrition therapy and physical activity (9,10)..Exercise is a major therapeutic modality in the treatment of diabetes mellitus [11]. Exercise training has been known to be effective in type 2 diabetes mellitus by increasing insulin sensitivity [12], and regular exercise can strengthen antioxidant defenses and may reduce oxidative stress [13]. Exercises including yoga postures have been shown to play a role in preventing type 2 diabetes [14]. The yoga postures are slow rhythmic movements which emphasize the stimulation of the organs and glands by easy bending and extensions which do not over-stimulates muscles but concentrate on glandular stimulation [15]. A major benefit of non-exhaustive exercise such as yoga is to induce a mild oxidative stress that stimulates the expression of certain antioxidant enzymes. This is mediated by the activation of redox-sensitive signaling pathways [16].

Over the past three decades, the etiology of insulin resistance and beta-cell dysfunction has been subject to intense study [17, 18]. Obesity, as a result of inactivity in combination with overeating, plays a key role in the development of pancreatic beta-cell dysfunction as well as insulin resistance. Several mechanisms mediating this interaction have been identified. It is now well established that a number of circulating hormones, cytokines, and metabolic fuels, such as non-esterified fatty acids (NEFAs), are being released by adipose tissue and can modulate insulin action. An increased mass of stored triglyceride, especially in visceral or deep subcutaneous adipose depots, leads to large adipocytes that are themselves resistant to the ability of insulin to suppress lipolysis. This results in increased release and circulating NEFA and glycerol levels both of which aggravate insulin resistance in skeletal muscle [5] and the liver [19,20].

Ectopic fat storage in hepatocytes, so-called intrahepatic lipids (IHL), has also been related to the development of hepatic insulin resistance [21] and hepatic inflammation, initiating non-alcoholic fatty liver disease [22]. In rodents, 3 days of a high-fat diet induces hepatic insulin resistance, while no significant changes in fat content in muscle or visceral tissue could be detected [23]. Experimental research now suggests that hepatic insulin resistance arises from DAG-induced activation of protein kinase C, which directly binds to and inhibits insulin receptor tyrosine kinase activity [24]. As such, fat-induced hepatic insulin resistance and hepatic inflammation are considered important etiological factors in the development of systemic insulin resistance.

Our study, however, examine the lipid level of offspring of diabetes on graded exercise using tummy trimmer as exercise apparatus. In conclusion, graded exercise using tummy trimmer is an important tool which improves dyslipidemia. It should be recommended for offspring of diabetes patients to delay or prevent the onset of diabetes mellitus.

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