RESULTS AND DISCUSSION PHASE II

PHASE II (B): KNOWLEDGE ATTITUDE AND PRACTICES OF TYPE 2 DIABETES PATIENTS WITH NAFLD

I. KNOWLEDGE ABOUT TYPE 2 DIABETES AMONG TYPE 2 DIABETES PATIENTS WITH NAFLD (TABLE 4.63, TABLE 4.64, FIG 4.32)

1. Knowledge about diabetes: At baseline, subjects in both the arms had almost similar score on the aspect of definition of diabetes, wherein 76.6% of the intervention arm subjects and 66.6% controls described it as a state of elevated blood glucose. With the intervention, the score improved significantly (1.1 to 1.8, P 0.0001) as about 60% of the subjects cited insulin non-utilisation and 66.7% reported inadequate production of insulin defining the state of diabetes. The controls had marginal increase in score (1.06 to 1.2, P 0.043). However, the score of the intervention arm subjects remained significantly higher than the controls (1.8 vs. 1.2, P 9.26E).

2. Knowledge about risk factors for diabetes: At baseline the scores on knowledge about risk factors for diabetes differed non-significantly between both the groups. The major shifts in increase in knowledge owing to nutrition counselling were evident in the experimental arm, as citing family history of diabetes, presence of hypertension, sedentary lifestyle, high intake of fruits and sugars and low intake of fruits and vegetables as risk factors for diabetes was opined by a major chunk of these subjects post intervention. Moreover, the controls only had a marginal increase or no alteration in these responses. Post intervention, the proportion dropped to zero in the experimental arm and to 6.6% in the control arm. This led to a significant (P 4.88E) increase in scores of subjects of the intervention arm and the control arm also had a significant rise in scores (P 0.029), which were significantly lower from the scores of the intervention arm (2.33 vs. 5.8, P 8.3E).

3. Knowledge about symptoms of diabetes: The scores on symptoms of diabetes were non-significantly different between groups. Knowledge about symptoms of diabetes increased in terms of proportion as was evident by a substantial chunk of the experimental arm subjects responding to increased thirst, increased urination, excessive hunger and pain and numbness in hand and feet as major responses. The

controls, on the other hand, had no alteration in this aspect. Consequently, the scores improved significantly for the intervention arm subjects (P 2.61E) and were also significantly higher from the controls (3.56 vs. 2.2, P 5E).

4. Knowledge about diagnosis of diabetes: At baseline, both the groups had almost similar scores and majority of the subjects in both the arms opined that diabetes can be diagnosed by a fasting blood sugar test and the second major response was urine test. As a result of inter-personal counselling, majority of the subjects in the experimental arm responded that fasting blood sugar, followed by urine test and HbA1c test can be used for the diagnosis of diabetes and a marginal proportion quoted the OGTT test. In case of controls, there was just a marginal increase in proportion of subjects who cited HbA1c test for diagnosis of diabetes, while the other responses remained unaltered from baseline. Therefore, the scores improved significantly (P 9.16E) in the intervention arm and were also significantly higher than the scores of the controls (2.4 vs. 1.33, P 1.59E).

5. Knowledge about impact of diabetes on other organs: At baseline, the scores were similar for both the groups wherein majority of them cited that eyes, kidneys and the heart are impacted if one is diabetic. The liver was not responded of by any of these subjects. Nutrition counselling brought about improvements in responses as majority quoted eyes, kidneys, heart and the liver being impacted by diabetes, while more than one third also cited nerves to be affected. The controls had no alterations in responses other than mild increase in the proportion of subjects responding to nerves, feet, kidneys and the liver getting affected, which brought about a significant increase in their scores (P 0.002). The intervention arm subjects had a significant (P 1.37E) increase in their score which was also significantly higher from that of the controls (3.73 vs. 1.8, P 3.63E).

6. Knowledge about effective management of diabetes: At baseline, the intervention arm subjects had significantly higher score than the controls (1.6 vs. 1.06, P 1.71E). Post intervention about 96.7% opined that timely medication, physical activity and balanced diet are a must for effective management of diabetes. This brought about an increase in score of these subjects (P 9.73 E). Among the controls, minor shifts led a significant increase in score (P 3.15E). However, the score remained significantly lower than the intervention arm (2.73 vs. 1.66, P 1.69E).

TABLE 4.63: KNOWLEDGE ABOUT TYPE 2 DIABETES MELLITUS AMONG TYPE 2 DIABETES PATIENTS WITH NAFLD (N, %)

Aspects	Responses	Experimental group		Control group	
		Pre	Post	Pre	Post
Definition of diabetes	Blood glucose elevation	23 (76.7)	17 (56.7)	20 (66.7)	20 (66.7)
	Insulin non-utilisation	6 (20)	18 (60)	7 (23.3)	8 (26.7)
	Inadequate production of insulin	5 (16.7)	20 (66.7)	5 (16.7)	8 (26.7)
	Do not know	2 (6.7)	0 (0)	1 (3.3)	1 (3.3)
Risk factors for diabetes	Diabetes family history	20 (66.7)	30 (100)	19 (63.3)	19 (63.3)
	Excess body weight	5 (16.7)	18 (60)	6 (20)	7 (23.3)
	Hypertension	4 (13.3)	19 (63.3)	6 (20)	6 (20)
	Heart disease	1 (3.3)	1 (3.3)	0 (0)	0 (0)
	Sedentary lifestyle	4 (13.3)	22 (73.3)	5 (16.7)	7 (23.3)
	High intake of fats	6 (20)	24 (80)	3 (10)	5 (16.7)
	High intake of sugars	6 (20)	24 (80)	3 (10)	5 (16.7)
	Low intake of fruits	3 (10)	19 (63.3)	4 (13.3)	5 (16.7)
	Low vegetable intake	3 (10)	19 (63.3)	4 (13.3)	5 (16.7)
	Stress	19 (63.3)	19 (63.3)	17 (56.7)	17 (56.7)
	Do not know	2 (6.7)	0 (0)	3 (10)	2 (6.7)
Symptoms of diabetes	Increased thirst	14 (46.7)	25 (83.3)	15 (50)	15 (50)
	Increased urination	14 (46.7)	25 (83.3)	15 (50)	15 (50)
	Excess hunger	8 (26.7)	16 (53.3)	8 (26.7)	8 (26.7)
	Unexplained weight loss	9 (30)	11(36.7)	4(13.3)	4 (13.3)
	Blurred vision	1 (3.3)	2 (6.7)	2 (6.7)	2 (6.7)
	Fatigue	4 (13.3)	7 (23.3)	8 (26.7)	8 (26.7)
	Pain, numbness in limbs	7 (23.3)	14 (46.7)	5 (16.7)	6 (20)
	Delayed wound healing	4 (13.3)	7 (23.3)	8 (26.7)	8 (26.7)
	Do not know	3 (10)	0 (0)	1 (3.3)	1 (3.3)

Figures in parenthesis indicate percentage

TABLE 4.63: KNOWLEDGE ABOUT TYPE 2 DIABETES MELLITUS AMONG TYPE 2 DIABETES PATIENTS WITH NAFLD (N, %)

Aspects	Responses	Experimental group		Control group	
		Pre	Post	Pre	Post
Diagnosis of diabetes	OGTT	1 (3.3)	7 (23.3)	1 (3.3)	1 (3.3)
	Fasting blood sugar test	23 (76.7)	28 (93.3)	24 (80)	24 (80)
	HbA1c test	2 (6.7)	18 (60)	2 (6.7)	4 (13.3)
	Urine test	14 (46.7)	20 (66.7)	11 (36.7)	11 (36.7)
	Do not know	1 (3.3)	0 (0)	1 (3.3)	1 (3.3)
Impact of diabetes on other organs	Eye	10 (33.3)	22 (73.3)	12 (40)	12 (40)
	Kidney	16 (53.3)	26 (86.7)	15 (50)	15 (50)
	Nerves	1 (3.3)	11 (36.7)	4 (13.3)	7 (23.3)
	Feet	5 (16.7)	7 (23.3)	3 (10)	4 (13.3)
	Heart	7 (23.3)	21 (70)	7 (23.3)	9 (30)
	Liver	0 (0)	24 (80)	3 (10)	15 (50)
	Do not know	1 (3.3)	0 (0)	0 (0)	0 (0)
Effective manage-ment	Timely medication	16 (53.3)	26 (86.7)	16 (53.3)	18 (60)
	Balanced diet	15 (50)	24 (80)	16 (53.3)	17 (56.7)
	Physical activity	13 (43.3)	26 (86.7)	15 (50)	17 (56.7)
	Regular blood test	0 (0)	2 (6.7)	0 (0)	0 (0)

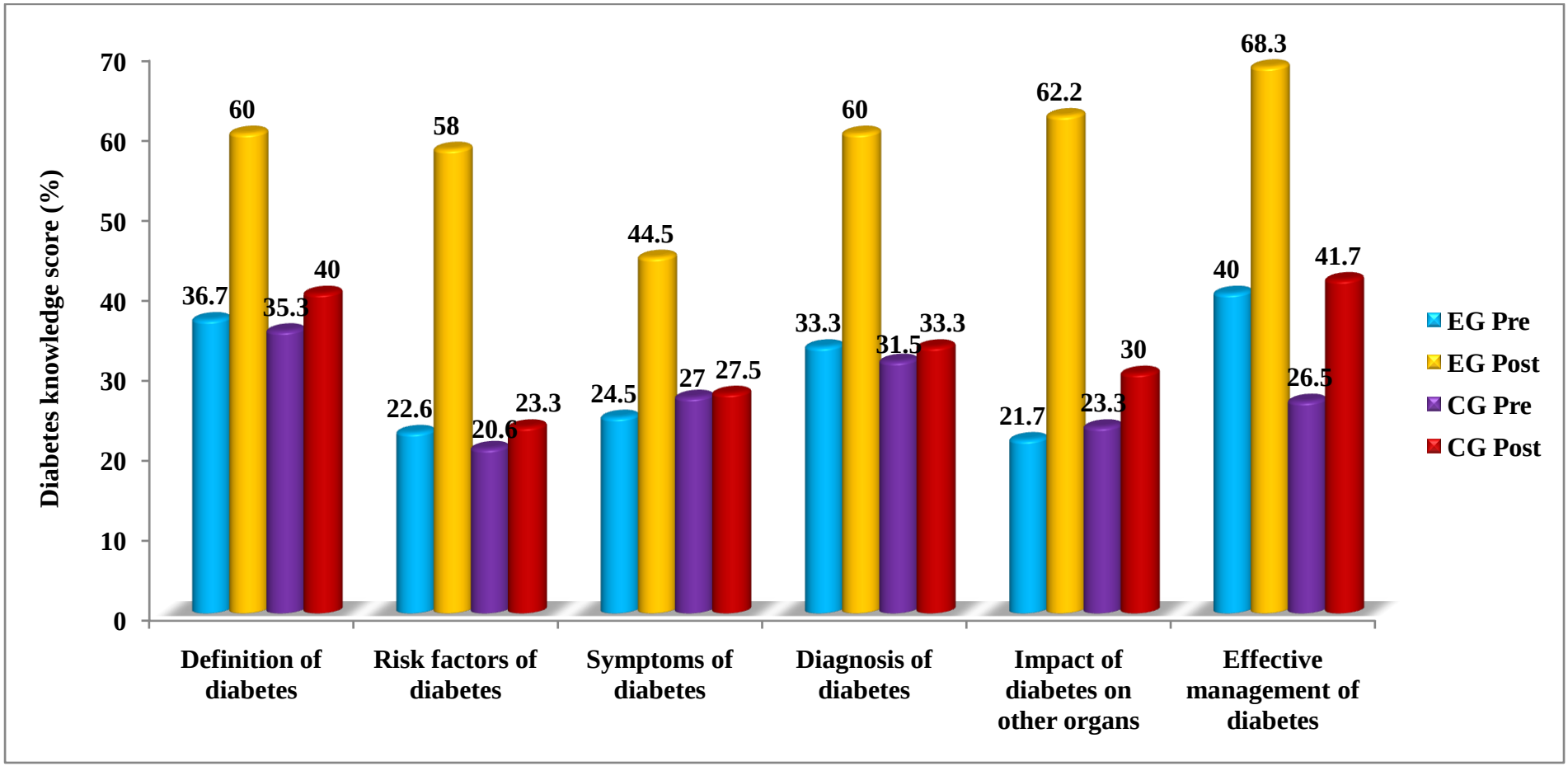
Figures in parenthesis indicate percentage

TABLE 4.64: IMPACT OF NUTRITION COUNSELLING ON KNOWLEDGE SCORES ON TYPE 2 DIABETES (MEAN ± SD)

VARIABLES	SCORE RANGE	STAGE	CONTROL GROUP (N=30)	EXPERIMENTAL GROUP (N=30)	P VALUE
Definition of diabetes	0-3	Pre	1.06 ± 0.36	1.1 ± 0.48	0.76
		Post	1.2 ± 0.48	1.8 ± 0.61	**9.26E*** **
		Paired t	**0.043***	**0.0001*** **	
Risk factors of diabetes	0-10	Pre	2.06 ± 1.11	2.26 ± 1.28	0.52
		Post	2.33 ± 1.21	5.8 ± 1.4	**8.3E*** **
		Paired t	**0.029***	**4.88E*** **	
Symptoms of diabetes	0-8	Pre	2.16 ± 1.05	1.96 ± 1.06	0.46
		Post	2.2 ± 1.06	3.56 ± 1.04	**5E*** **
		Paired t	0.32	**2.61E*** **	
Diagnosis of diabetes	0-4	Pre	1.26 ± 0.69	1.33 ± 0.54	0.68
		Post	1.33 ± 0.71	2.4 ± 0.67	**1.59E*** **
		Paired t	0.16	**9.16E*** **	
Impact of diabetes on other organs	0-6	Pre	1.4 ± 0.77	1.3 ± 0.53	0.56
		Post	1.8 ± 1.12	3.73 ± 0.78	**3.63E*** **
		Paired t	**0.002****	**1.37E*** **	
Effective management of diabetes	0-4	Pre	1.06 ± 0.36	1.6 ± 0.49	**1.71E*** **
		Post	1.66 ± 0.54	2.73 ± 0.52	**1.69E*** **
		Paired t	**3.15E*** **	**9.73E*** **	

P<0.05*, P<0.01**, P<0.001***

FIG 4.32: PERCENT IMPROVEMENT IN DIABETES KNOWLEDGE SCORES IN TYPE 2 DIABETICS WITH NAFLD AFTER NUTRITION COUNSELLING (%)



Impact of Nutrition Counselling On Knowledge Scores of Type 2 Diabetes

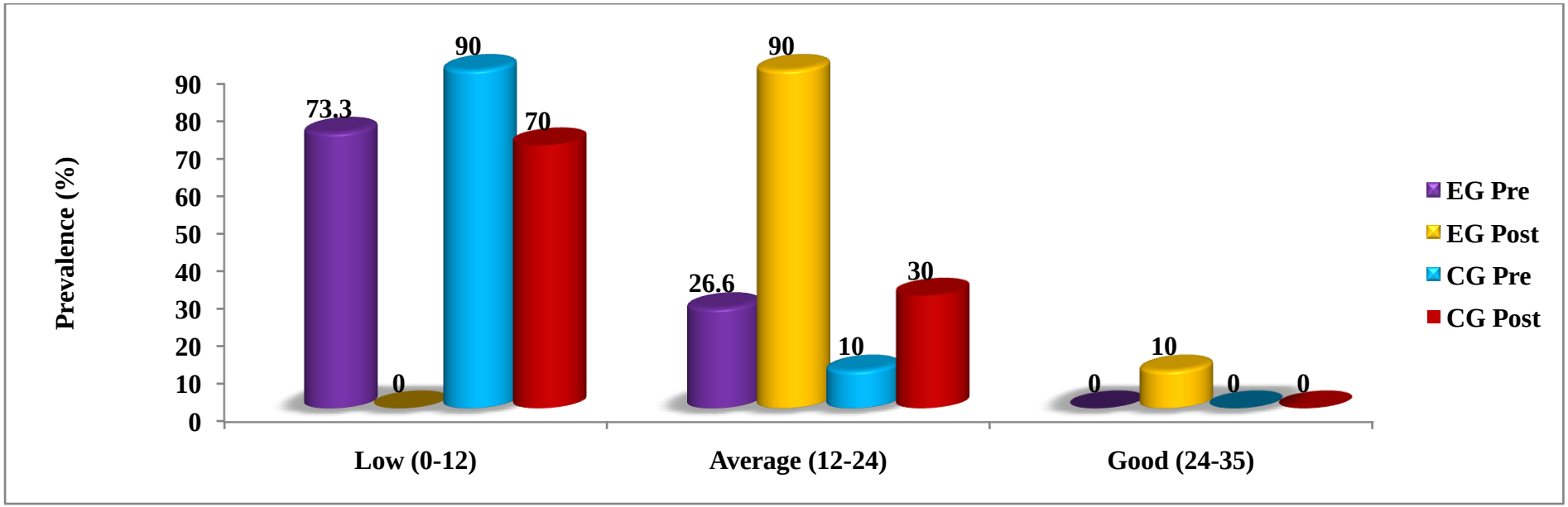
The nutrition counselling intervention to adopt lifestyle changes brought about a significant 110.3% (P 4.3E) improvement in knowledge scores of diabetes shifting the mean score to average category as knowledge scores improved significantly on all the parameters of diabetes (table 4.65). The controls only had 16.3% (P 2.51E) increase in scores falling in the poor category. However, despite improvement in controls, the scores of the experimental arm were significantly higher from the controls at the end of the intervention on knowledge on diabetes (20.1 vs. 10.5, P 2.39E). Majority of the subjects had low scores on diabetes at baseline, which reduced to nil from 73.3% in the intervention arm subjects (P 0.000) and lowered non-significantly in the controls from 90% to 70%. As a result, the controls had significantly high prevalence of low score vs. nil in the intervention arm (70% vs. 0%, P 0.000). Nutrition counselling brought about a significant increase in the average knowledge scores on diabetes from 26.6% to 90% (P 0.0000008) and improved non-significantly from 10% to 30% in controls. Hence, the prevalence of average scores on knowledge about diabetes was significantly higher in subjects in the intervention arm than the controls (90% vs. 30%, P 0.000). None of the controls had good knowledge scores about diabetes whereas 10% did in the intervention arm at post stage of the study (fig 4.33).

TABLE 4.65: IMPACT OF NUTRITION COUNSELLING ON TOTAL SCORES ON KNOWLEDGE ON DIABETES (MEAN ± SD)

Variables	Score	Stage	Control Group (N=30)	Experimental Group (N=30)	P Value
Knowledge score on diabetes	0-35	Pre	9.03 ± 2.5	9.56 ± 3.24	0.48
		Post	10.5 ± 2.8	20.1 ± 2.6	**2.39E*** **
		Paired t	**2.51E*** **	**4.3E*** **	

P<0.05*, P<0.01**, P<0.001***

FIG 4.33: IMPACT OF NUTRITION COUNSELLING ON KNOWLEDGE SCORES OF TYPE 2 DIABETICS WITH NAFLD (%)



II. KNOWLEDGE ABOUT NAFLD AMONG TYPE 2 DIABETES PATIENTS WITH NAFLD (TABLE 4.66, TABLE 4.67, FIG 4.34)

1. Awareness about the term NAFLD: At baseline, 96.7% of the experimental arm subjects and 93.3% controls had never heard about the term NAFLD. However, with the intervention, all of the experimental arm subjects knew about NAFLD (P 5.7E) and so did 83.3% controls (P 1.14E). The reason behind the rise in proportion in controls can be attributed to explanation given about NAFLD as a part of the research protocol during the consent stage. However, the scores of the intervention arm subjects remained significantly higher than the controls (1 vs. 0.83, P 0.022).

2. Knowledge about the definition of NAFLD: At baseline, about 96.7% of the experimental arm subjects and 93.3% controls could not describe what NAFLD meant. After the intervention for the experimental arm and the protocol description to the control arm, 93.3% in the experimental arm and 66.7% in the control arm described NAFLD as the accumulation of fat in the liver. Hence, the mean scores increased significantly for the intervention arm subjects (0.03 to 0.93, P 4.88E) and for controls (0.06 to 0.66, P3.15E). However, controls had significantly lower scores than the intervention arm subjects (0.66 vs. 0.93, P 0.009).

3. Risk factors for NAFLD: At baseline, both the groups had no knowledge about the risk factors for NAFLD. With the nutrition counselling intervention, a substantial 90% of these subjects quoted IR and T2DM, 76.7% said obesity as risk factors for NAFLD. This led to a significant increase in score (0.03 to 2.1, P 9.22E). The controls also had a significant increase in score (0.13 to 1.03, P 5.99E) owing to majority of them quoting IR and T2DM as risk factors. However, the score of the intervention arm subjects remained significantly higher than the controls (2.1 vs. 1.03, P 2.11E).

4. Knowledge about predisposition to NAFLD due to diabetes: None of the subjects at baseline in the experimental group were aware about predisposition to NAFLD owing to their diabetic state vs. only 3.3% controls being aware about it. With the nutrition counselling intervention, about 73.3% of the subjects were able to describe that the fat present in the liver will determine the quantity of insulin required and another 6.7% cited that fat in the liver will determine how severe the IR would be. This resulted in an increase in the score of intervention arm subjects from nil at

baseline to 0.93 (P 9.54E). The controls had no alteration in their score and therefore the score of the intervention arm subjects remained significantly higher than the controls (0.93 vs. 0.06, P 9.54E).

5. Knowledge about occurrence and progression of NAFLD: At baseline, both the arms had near nil scores on knowledge about occurrence and progression of NAFLD. The intervention brought about a significant rise in scores (0.03 to 2, P 6.9E) in the experimental arm subjects owing to majority of the subjects (83.3%) claiming diet rich in fats and sugars and one third claiming IR as the pathogenic and progressive factor. Because a few of the controls had reverted to the doctor and also accessed the internet, 20% claimed that NAFLD occurs and progresses due to a diet rich in sugars and fats and 10% said IR that led to a significant rise in their scores (0.03 to 0.5, P 0.013). Yet, 73.3% controls remained unaware about the pathophysiology and progression of NAFLD at the end of the study and hence the scores of the intervention arm subjects was significantly higher than the controls (2.0 vs. 0.5, P 1.85E).

6. Knowledge about symptoms of NAFLD: At baseline, none of the subjects in the intervention arm were aware about the symptomatology of NAFLD and so were 96.7% of the controls. After the intervention, about 80% of the intervention subjects were able to cite that NAFLD is asymptomatic. This led to a significant increase in score from nil at baseline to 0.93 (P 1.34E). The controls had mild alterations in their responses which led to a significant rise (0.03 to 0.23, P 0.011), which however, was significantly lower from the scores of the intervention arm subjects (0.23 vs. 0.93, P 7.76E). At the termination of the study, 76.7% of the controls remained unaware about the symptoms of NAFLD.

7. Knowledge about diagnosis of NAFLD: At baseline, subjects in both the arms had score falling in the poor category on knowledge about the diagnosis of NAFLD. The intervention brought about a significant improvement in the experimental arm subjects (0.03 to 1.3, P 2.26E) wherein 90% opined that NAFLD can be diagnosed with the help of USG and a little less than half also cited liver function test as the response. Because the study protocol was explained in depth to the controls also, 70% of them could respond USG as the method of diagnosis of NAFLD. This led to a significant rise in the score (0.03 to 0.8, P 1.14E) but was significantly lower from the scores of the intervention arm subjects (0.8 vs. 1.3, P 9.44E).

8. Knowledge about necessity of treating NAFLD: At baseline, there was much unawareness about the necessity for treating NAFLD in both the arms. The nutrition counselling intervention brought about a significant increase in score (0.03 to 1.53, P 4.88E), as about 86.7% of the subjects cited prevention of liver complication and 60% said prevention of heart disease as the reasons for treating NAFLD. The controls also had a significant increase in score (0.1 to 0.43, P 0.015) as one fifth of them reported occurrence of hepatic and cardiac consequences if NAFLD was left untreated. However, 80% of the controls remained unaware about the consequences of NAFLD at the end of the study and hence their scores were significantly lower from the subjects of the intervention arm (0.43 vs. 1.53, P 1.65E).

9. Knowledge about treatment modalities for NAFLD: At baseline, 96.7% of the subjects in both the arms had no knowledge about the treatment of NAFLD. With the intervention, the scores improved significantly (0.1 to 2.9, P 7.74E) as physical activity (80%), balanced diet (70%), weight control (66.7%) were cited as responses for the treatment of NAFLD. The controls also improved significantly on their score (0.06 to 0.9, P 0.0013) as one third of them said physical activity and one third said medications could be used to treat NAFLD. However, the scores of the intervention arm subjects remained significantly higher than the controls at the end of the study (2.9 vs. 0.9, P 1.17E) as about 66.7% of the controls did not know about NAFLD's treatment.

TABLE 4.66: KNOWLEDGE ABOUT NAFLD AMONG TYPE 2 DIABETES PATIENTS WITH NAFLD (N, %)

Questions	Responses	Experimental group (N=30)		Control group (N=30)	
		Pre	Post	Pre	Post
Awareness about NAFLD	Yes	1 (3.3)	30 (100)	2 (6.7)	25 (83.3)
	No	29 (96.7)	0 (0)	28 (93.3)	5 (16.7)
Definition of NAFLD	Fat accumulation in liver	1 (3.3)	28 (93.3)	2 (6.7)	20 (66.7)
	Do not know	29 (96.7)	2 (6.7)	28 (93.3)	10 (33.3)
Risk factors for NAFLD	Obesity	0 (0)	23 (76.7)	0 (0)	5 (16.7)
	IR / T2DM	1 (3.3)	27 (90)	1 (3.3)	20 (66.7)
	Hypertension	0 (0)	5 (16.7)	0 (0)	0 (0)
	Altered lipid profile	0 (0)	6 (20)	1 (3.3)	4 (13.3)
	Surgery	0 (0)	0 (0)	1 (3.3)	1 (3.3)
	Hepatotoxic drugs	0 (0)	2 (6.7)	1 (3.3)	1 (3.3)
	Do not know	29 (96.7)	2 (6.7)	29 (96.7)	6 (20)
Predisposition to NAFLD owing to diabetes	Fatty liver influences the severity of IR	0 (0)	2 (6.7)	0 (0)	0 (0)
	Hepatic fat predicts insulin requirement	0 (0)	22 (73.3)	1 (3.3)	1 (3.3)
	Do not know	30 (100)	6 (20)	29 (96.6)	29 (96.6)
How does NAFLD occur and progress?	Diet rich in sugars	0 (0)	25 (83.3)	0 (0)	6 (20)
	Diet rich in fats	0 (0)	25 (83.3)	0 (0)	6 (20)
	Insulin resistance	1 (3.3)	10 (33.3)	1 (3.3)	3 (10)
	Do not know	29 (96.7)	2 (6.7)	29 (96.6)	22 (73.3)

Figures in parenthesis indicate percentage

TABLE 4.66: KNOWLEDGE ABOUT NAFLD AMONG TYPE 2 DIABETES PATIENTS WITH NAFLD (N, %)

Questions	Responses	Experimental group (N=30)		Control group (N=30)	
		Pre	Post	Pre	Post
Symptoms of NAFLD	Asymptomatic	0 (0)	24 (80)	1 (3.3)	6 (20)
	Fatigue	0 (0)	2 (6.7)	0 (0)	0 (0)
	Abdominal discomfort	0 (0)	2 (6.7)	0 (0)	1 (3.3)
	Do not know	30 (100)	2 (6.7)	29 (96.7)	23 (76.7)
Diagnosis of NAFLD	Ultrasound	1 (3.3)	27 (90)	1 (3.3)	21 (70)
	Liver function test	0 (0)	14 (46.7)	0 (0)	3 (10)
	Lipid profile	0 (0)	4 (13.3)	0 (0)	0 (0)
	Do not know	29 (96.7)	0 (0)	29 (96.7)	6 (20)
Need for treating NAFLD	Prevent liver complication	1 (3.3)	26 (86.7)	2 (6.7)	6 (20)
	Prevent heart disease	0 (0)	18 (60)	1 (3.3)	6 (20)
	Prevent diabetes complications	0 (0)	2 (6.7)	0 (0)	0 (0)
	Do not know	29 (96.7)	0 (0)	28 (93.3)	24 (80)
Treatment of NAFLD	Weight control	0 (0)	20 (66.7)	0 (0)	0 (0)
	Balanced diet	0 (0)	21 (70)	0 (0)	5 (16.7)
	Insulin sensitizing drugs	1 (3.3)	18 (60)	1 (3.3)	6 (20)
	Fat lowering drugs	1 (3.3)	5 (16.7)	1 (3.3)	6 (20)
	Physical activity	1 (3.3)	24 (80)	0 (0)	10 (33.3)
	Do not know	29 (96.7)	2 (6.7)	29 (96.7)	20 (66.7)

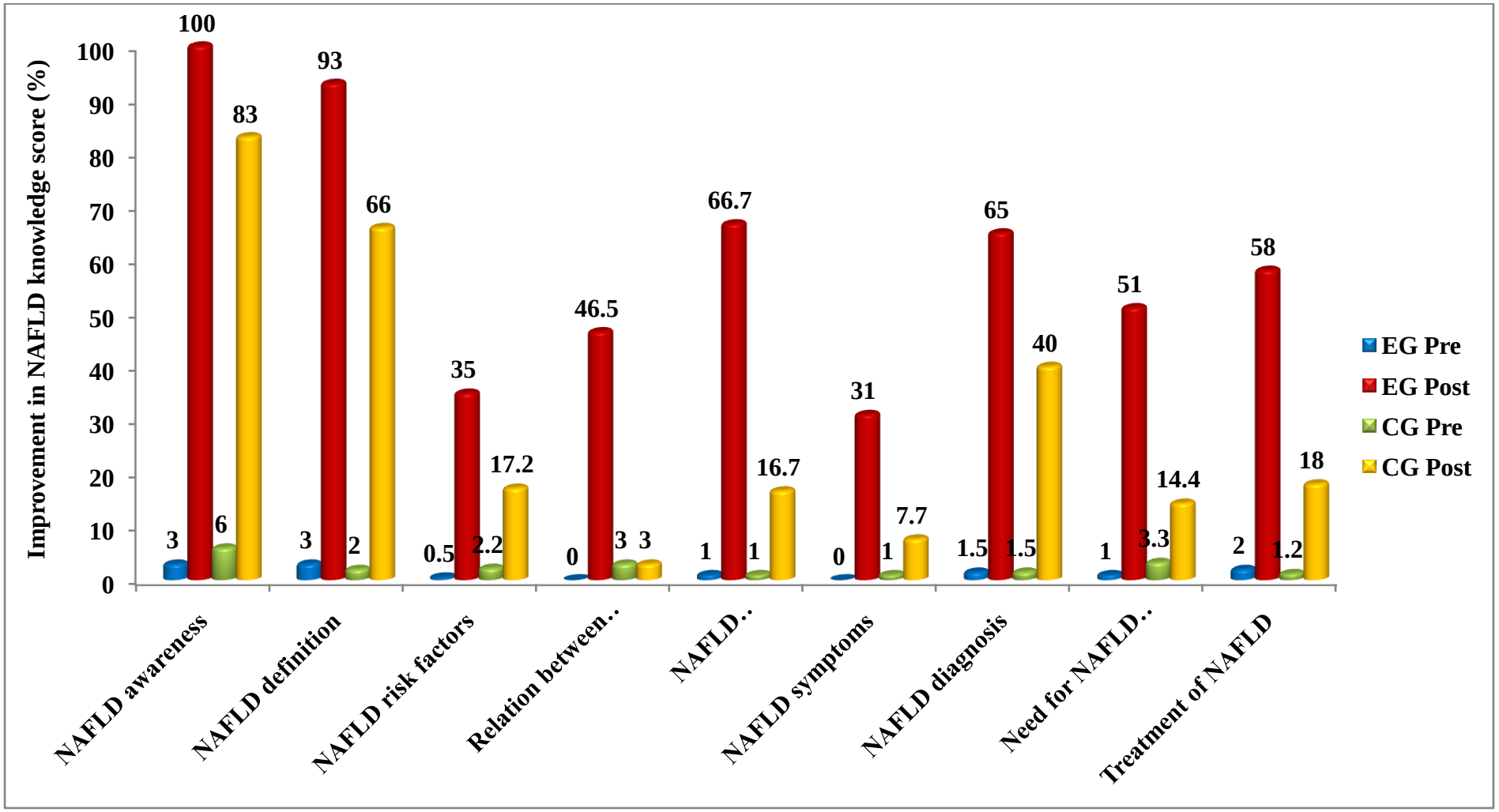
Figures in parenthesis indicate percentage

TABLE 4.67: IMPACT OF NUTRITION COUNSELLING ON KNOWLEDGE SCORES OF NAFLD (MEAN ± SD)

Variables	Score	Stage	Control Group (N=30)	Experimental Group (N=30)	P Value
Awareness about the term NAFLD	0-1	Pre	0.06 ± 0.25	0.03 ± 0.18	0.56
		Post	0.83 ± 0.37	1± 0	**0.022***
		Paired t	**1.1E*****	**5.7E*****	
Definition of NAFLD	0-1	Pre	0.06 ± 0.25	0.03 ± 0.18	0.56
		Post	0.66 ± 0.47	0.93 ± 0.25	**0.009****
		Paired t	**3.15E*****	**4.88E*****	
Risk factors for NAFLD	0-6	Pre	0.13 ± 0.73	0.03 ± 0.18	0.47
		Post	1.03 ± 0.8	2.1 ± 0.75	**2.11E*****
		Paired t	**5.99E*****	**9.22E*****	
Predisposition to NAFLD due to T2DM	0-2	Pre	0.06 ± 0.36	0.0 ± 0.0	0.32
		Post	0.06 ± 0.36	0.93 ± 0.58	**9.54E*****
		Paired t	1	**1.2E*****	
NAFLD occurrence, progression	0-3	Pre	0.03 ± 0.18	0.03 ± 0.18	1
		Post	0.5 ± 0.97	2.0 ± 0.78	**1.85E*****
		Paired t	**0.013***	**6.9E*****	
Symptoms of NAFLD	0-3	Pre	0.03 ± 0.18	0 ± 0	0.32
		Post	0.23 ± 0.43	0.93 ± 0.25	**7.76E*****
		Paired t	**0.011***	**1.34E*****	
Diagnosis of NAFLD	0-2	Pre	0.03 ± 0.18	0.03 ± 0.18	1
		Post	0.8 ± 0.4	1.3 ± 0.49	**9.44E*****
		Paired t	**1.14E*****	**2.26E*****	
Relevance of treating NAFLD	0-3	Pre	0.1 ± 0.4	0.03 ± 0.18	0.41
		Post	0.43 ± 0.81	1.53 ± 0.57	**1.65E*****
		Paired t	**0.015***	**4.88E*****	
Treatment modalities of NAFLD	0-5	Pre	0.06 ± 0.36	0.1 ± 0.54	0.78
		Post	0.9 ± 1.3	2.9 ± 1.2	**1.17E*****
		Paired t	**0.0013****	**7.74E*****	

P<0.05*, P<0.01**, P<0.001***

FIG 4.34: IMPROVEMENT IN NAFLD KNOWLEDGE SCORES AMONG TYPE 2 DIABETICS WITH NAFLD (%)



Impact of Nutrition Counselling on Knowledge Scores on NAFLD

At baseline, the knowledge on NAFLD was similar in both the arms with practically near nil scores on all the aspects (table 4.68). Post intervention, the scores improved significantly in the experimental arm from poor category 0.13 to average category 13.7 (P 1.08E) on knowledge scores on NAFLD. The control arm also showed improvement in knowledge scores on NAFLD (0.6 to 5.4, P 9.84E) owing to indepth explanation of the protocol of the study that required explaining the pros and cons of the diseased condition to all the participants' irrespective of their groups. However, the experimental arm performed better than the control arm at the end of the intervention as reflected by significantly higher total scores on knowledge on NAFLD than the controls, whose mean score still remained in the poor category (13.7 vs. 5.4, P 1.18E).

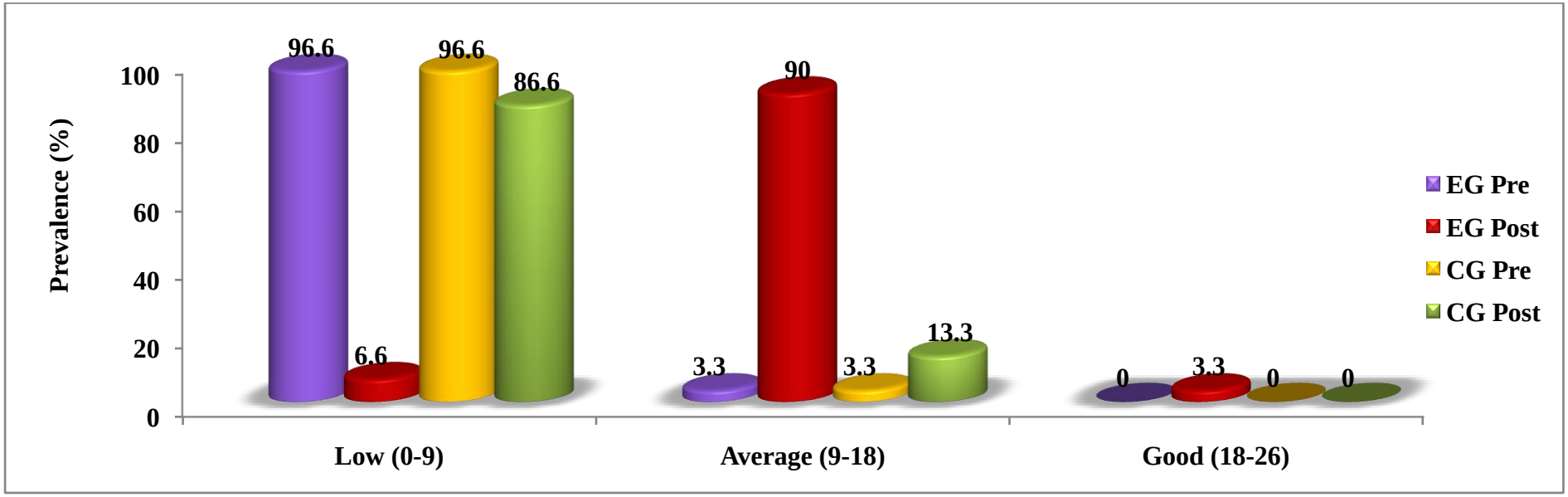
At baseline, 96.7% of the subjects of both the arms had low knowledge scores of NAFLD. Post intervention, the figure went down to 6.6% in the experimental arm (P 0.000) and became 86.6% in controls and hence they had a significantly higher prevalence of low knowledge scores on NAFLD (P 0.000). At baseline 3.3% had average knowledge scores on NAFLD in both the arms. With the intervention, it increased to 90% in the experimental arm (P 0.000) vs. only 13.3% controls (P 0.000). While none in the control arm had good scores on NAFLD pre and post study, 3.3% of the experimental arm subjects had good knowledge scores on NAFLD after the intervention (fig 4.35).

TABLE 4.68: IMPACT OF NUTRITION COUNSELLING ON THE TOTAL KNOLWEDGE SCORES OF NAFLD (MEAN ± SD)

Variables	Score	Stage	Control Group (N=30)	Experimental Group (N=30)	P Value
Knowledge scores on NAFLD	0-26	Pre	0.6 ± 2.6	0.3 ± 1.6	0.59
		Post	5.4 ± 3.3	13.7 ± 2.8	**1.18E*** **
		Paired t	**9.84E*** **	**1.08E*** **	

P<0.05*, P<0.01**, P<0.001***

FIG 4.35: IMPACT OF NUTRITION COUNSELLING ON NAFLD KNOWLEDGE SCORES IN TYPE 2 DIABETICS WITH NAFLD (%)



III. ATTITUDE AND PRACTICES OF TYPE 2 DIABETES PATIENTS WITH NAFLD (TABLE 4.69, TABLE 4.70, FIG 4.36)

1. Satisfaction with current exercise regime: At baseline, both the groups had similar score on satisfaction with exercise regime with 60% of the intervention arm subjects and 56.6% controls. The intervention brought along a significant increase in satisfaction with exercise regime (0.40 to 0.73, P 0.0006) with 73.3% of the subjects being satisfied as against half of the controls who still remained dissatisfied.

2. Dietary restrictions: Subjects in both the arms had almost similar scores wherein majority (96.7%) avoided sweets, followed by sweet fruits and fatty foods. The score improved significantly (1.9 to 2.53, P 9.21E) in the intervention arm subjects. The controls too had a significant rise in score (1.83 to 2.03, P 0.031) but was significantly lower from the intervention arm subjects (P 0.014).

3. Regularity in medicines: Regularity in medicines was similar in both the arms at baseline with >70% adhering to the regime. The intervention led to a significant increase in score (0.76 to 0.90, P 0.043) as the proportion increased to 90%.

4. Visit to diabetologist: At baseline, only 13.3% of the intervention subjects and 16.7% controls went for a health check up. With the intervention, that emphasized on seeing the doctor once in a quarter of a year, the proportion increased to 26.7% (0.13 to 0.26, P 0.043) and the statistics remained unaltered in controls.

5. Monitoring of FBS: FBS prior to the intervention was monitored on a monthly basis by 46.6% of the intervention arm subjects and 36.6% of the controls. The intervention significantly improved the FBS monitoring in these subjects (P 0.011) with monthly monitoring going upto 66.6% vs. only 4.4% improvement in the controls. Hence, the practice of FBS monitoring was significantly improved in the intervention arm subjects and better than the controls (P 0.035).

6. Monitoring of HbA1c: At baseline, only 33.3% of the subjects in the intervention arm and 36.7% controls used to get their HbA1c monitored once in six months. With

intervention, 56.7% of the experimental arm subjects said they would get it checked half yearly, improving the scores significantly (0.33 to 0.56, P 0.0059), with no alteration in controls. Although non-significant, the prevalence of timely Hba1c monitoring was better in intervention arm than the controls (56.7% vs. 36.7%, P 0.12).

7. Monitoring of BP: With the intervention, the score of BP monitoring improved significantly (0.53 to 0.86, P 0.0006) as majority of the subjects started measuring their BP monthly/fortnightly and weekly. The controls had no alteration in their BP measurement frequency and their score remained non-significantly lower than the intervention arm subjects.

8. Monitoring of lipid profile and kidney profile: The prevalence of estimation of lipid and hepatic profile estimation was similar in both the groups at baseline as 83% got it estimated as per the ADA norms of atleast once in a year. With the intervention, the score became hundred percent and hence improved significantly (0.83 to 1.0, P 0.022) and that of controls improved marginally which led to intervention arm subjects having better score than the controls (1.0 vs. 0.86, P 0.043).

9. Visit to the ophthalmologist: At baseline, subjects of both the arms (about half) visited the ophthalmologist once in a year. However, with the intervention, the prevalence of subjects quoting that they would go for yearly visit increased from 50% to 80% that increased the score significantly (0.5 to 0.8, P 0.0014). As the prevalence remained unaltered among controls, the intervention arm subjects performed better than the controls (0.53 vs. 0.8, P 0.028).

10. Abdominal ultrasound for liver scan: About 96.7% of the subjects in both the arms had never appeared for a liver ultrasound. With the intervention, about 83.3% of the subjects said that they would go yearly for liver ultrasound because of which their score improved (0.0 to 0.83, P 1.19E). Among the controls, 1/3rd of them stated that they too would go for yearly scan and hence their score improved (0.03 to 0.33, P 0.004) but was significantly lower from the intervention arm (0.33 vs. 0.83, P 3.81E).

TABLE 4.69: ATTITUDE AND PRACTICE OF TYPE 2 DIABETES PATIENTS WITH NAFLD (N, %)

Questions	Responses	Experimental group		Control group	
		Pre	Post	Pre	Post
Satisfaction with exercise regime	Yes	12 (40)	22 (73.3)	13 (43.3)	15 (50)
	No	18 (60)	8 (26.7)	17 (56.6)	15 (50)
Dietary restrictions kept in mind while eating	Avoid sweets	29 (96.6)	30 (100)	29 (96.6)	29 (96.6)
	Avoid sweet fruits	18 (60)	22 (73.3)	15 (50)	17 (56.6)
	Avoid fatty, oily food	10 (33.3)	22 (73.3)	11 (36.7)	15 (50)
Regularity in medication	Yes	23 (76.6)	27 (90)	22 (73.3)	25 (83.3)
	No	7 (23.33)	3 (10)	8 (26.7)	5 (16.7)
Visit to diabetologist (pre and post)	Once in 3 months	4 (13.33)	8 (26.7)	5 (16.7)	5 (16.7)
	Once in 6 months	14 (46.7)	17 (56.7)	16 (53.3)	16 (53.3)
	Once in a year	6 (20)	3 (10)	4 (13.33)	4 (13.33)
	Once in 2 years	6 (20)	2 (6.66)	5 (16.7)	5 (16.7)
FBS estimation (pre and post)	Fortnightly	2 (6.7)	2 (6.7)	2 (6.66)	2 (6.7)
	Once a month	14 (46.6)	20 (66.6)	11 (36.6)	12 (40)
	Once in 2 months	5 (16.7)	6 (20)	11 (36.6)	9 (30)
	Once in 3 months	9 (30)	2 (6.7)	6 (20)	7 (23.33)
HbA1c estimation (pre and post)	Once in 6 months	10 (33.3)	17 (56.7)	11 (36.7)	11 (36.7)
	Once in a year	14 (46.6)	8 (26.7)	12 (40)	13 (43.3)
	Once in 2 years	6 (20)	5 (16.7)	7 (23.33)	6 (20)
BP monitoring (pre and post)	Weekly	2 (6.6)	4 (13.33)	5 (16.7)	5 (16.7)
	Fortnightly	6 (20)	8 (26.7)	7 (23.33)	7 (23.33)
	Once a month	8 (26.7)	14 (46.6)	10 (33.3)	10 (33.3)
	Once in 2 months	11 (36.6)	2 (6.7)	6 (20)	6 (20)
	Once in 3 months	3 (10)	2 (6.7)	2 (6.7)	2 (6.7)

Figures in parenthesis indicate percentage

TABLE 4.69: ATTITUDE AND PRACTICE OF TYPE 2 DIABETES PATIENTS WITH NAFLD (N, %)

Questions	Responses	Experimental group		Control group	
		Pre	Post	Pre	Post
Lipid profile estimation (pre and post)	Once in 6 months	15 (50)	16 (53.3)	6 (20)	12 (40)
	Once in a year	10 (33.3)	14 (46.7)	19 (63.3)	14 (46.7)
	Once in 2 years	5 (16.7)	0 (0)	5 (16.7)	4 (13.33)
Kidney profile estimation (pre and post)	Once in 6 months	15 (50)	16 (53.3)	6 (20)	12 (40)
	Once in a year	10 (33.3)	14 (46.7)	19 (63.3)	14 (46.7)
	Once in 2 years	5 (16.7)	0 (0)	5 (16.7)	4 (13.33)
Eye examination frequency (pre and post)	Once in a year	15 (50)	24 (80)	17 (56.5)	17 (56.5)
	Once in 2 years	10 (33.3)	2 (6.7)	8 (26.7)	8 (26.7)
	Never	5 (16.7)	4 (13.33)	5 (16.7)	5 (16.7)
Liver ultrasound (pre and post)	Yes	1 (3.3)	25 (83.3)	1 (3.3)	10 (33.3)
	No	29 (96.7)	5 (16.7)	29 (96.7)	20 (66.7)

Figures in parenthesis indicate percentage

TABLE 4.70: IMPACT OF NUTRITION COUNSELLING ON ATTITUDE AND PRACTICES SCORES ON TYPE 2 DIABETES AND NAFLD (MEAN ± SD)

Variables	Score	Stage	Control Group (N=30)	Experimental Group (N=30)	P Value
Satisfaction with exercise regime	0-1	Pre	0.43 ± 0.5	0.40 ± 0.49	0.79
		Post	0.5 ± 0.5	0.73 ± 0.44	0.06
		Paired t	0.16	**0.0006*** **	
Dietary restrictions on eating	0-3	Pre	1.83 ± 0.79	1.9 ± 0.84	0.75
		Post	2.03 ± 0.76	2.53 ± 0.77	**0.014***
		Paired t	**0.031***	**9.21E*** **	
Regularity in taking medicines	0-1	Pre	0.73 ± 0.44	0.76 ± 0.43	0.77
		Post	0.83 ± 0.37	0.9 ± 0.3	0.45
		Paired t	0.08	**0.043***	
Health check up frequency	0-1	Pre	0.16 ± 0.37	0.13 ± 0.34	0.72
		Post	0.16 ± 0.37	0.26 ± 0.44	0.35
		Paired t	1	**0.043***	
FBS estimation frequency	0-1	Pre	0.43 ± 0.5	0.53 ± 0.50	0.44
		Post	0.46 ± 0.5	0.73 ± 0.44	**0.035***
		Paired t	0.32	**0.011***	
HbA1c estimation frequency	0-1	Pre	0.36 ± 0.49	0.33 ± 0.47	0.79
		Post	0.36 ± 0.49	0.56 ± 0.5	0.12
		Paired t	1	**0.0059****	
BP measurement frequency	0-1	Pre	0.73 ± 0.44	0.53 ± 0.5	0.11
		Post	0.73 ± 0.44	0.86 ± 0.34	0.20
		Paired t	1	**0.0006*** **	
Lipid profile estimation frequency	0-1	Pre	0.83 ± 0.37	0.83 ± 0.37	1
		Post	0.86 ± 0.34	1.0 ± 0.0	**0.043***
		Paired t	0.74	**0.022***	
Renal profile estimation frequency	0-1	Pre	0.83 ± 0.37	0.83 ± 0.37	1
		Post	0.86 ± 0.34	1.0 ± 0.0	**0.043***
		Paired t	0.74	**0.022***	

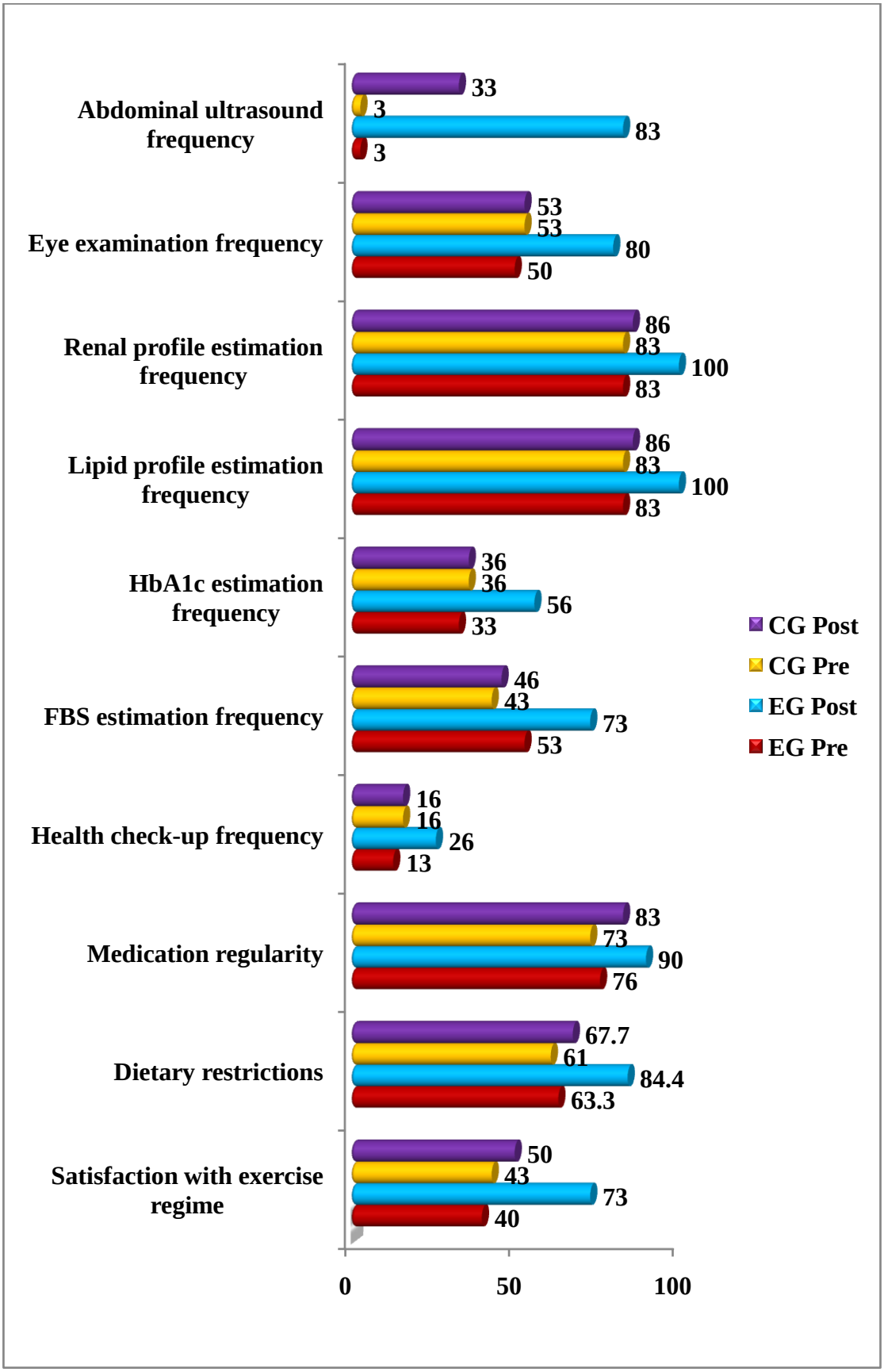
P<0.05*, P<0.01**, P<0.001***

TABLE 4.70: IMPACT OF NUTRITION COUNSELLING ON ATTITUDE AND PRACTICES SCORES ON TYPE 2 DIABETES AND NAFLD (MEAN ± SD)

Variables	Score	Stage	Control Group (N=30)	Experimental Group (N=30)	P Value
Eye examination frequency	0-1	Pre	0.53 ± 0.5	0.5 ± 0.5	0.80
		Post	0.53 ± 0.5	0.8 ± 0.4	**0.028***
		Paired t	1	**0.0014****	
Abdominal ultrasound frequency	0-1	Pre	0.03 ± 0.18	0.03 ± 0.18	1
		Post	0.33 ± 0.47	0.83 ± 0.37	**3.81E*****
		Paired t	**0.004****	**1.19E*****	

P<0.05*, P<0.01**, P<0.001***

FIG 4.36: IMPROVEMENT IN ATTITUDES AND PRACTICES OF TYPE 2 DIABETICS WITH NAFLD (%)



Impact of Nutrition Counselling on Attitude and Practices Scores of Type 2 Diabetics with NAFLD

At baseline, both the groups had average scores on attitudes and practices regarding type 2 diabetes and NAFLD (table 4.71). With the intervention, scores improved on all the aspects in the experimental arm (6.8 to 10.2, P 5.99E) and shifted to good category. Among the controls, although the scores improved significantly (6.9 to 7.7, P 0.006) it remained in the average category only. Thus, the score of the intervention arm subjects was significantly better than the controls (10.2 vs. 7.7, P 5.59E). With the intervention, the prevalence of low score became nil in both the arms. The prevalence of average score reduced significantly in the intervention arm (53.3% to 23.3%, P 0.017) and was significantly lower from the prevalence (50%) among controls (P 0.033). Consequently, the prevalence of good scores increased significantly in the intervention subjects (36.6% to 76.6%, P 0.0019) and was also significantly higher from the controls (76.6% vs. 50%, P 0.033) (fig 4.37).

IMPACT OF NUTRITION COUNSELLING ON KNOWLEDGE ATTITUDE AND PRACTICES SCORES OF TYPE 2 DIABETES AND NAFLD

The total KAP score was falling in the poor category at baseline for both the arms. The nutrition counselling intervention brought about increase in score (16.6 to 44.03, P 1.8E) shifting the mean score to average category (table 4.72). The controls also had improvement in KAP score (16.5 to 23.7, P 1.05E). However, the score remained in the poor category and consequently, the intervention arm subjects had significantly higher KAP score than the controls (P 44.03 vs. 23.7, P 8.72E). The prevalence of low score declined significantly in the intervention arm subjects (93.3% to 0%, P 0.000) and among controls declined from 96.6% to 70% (P 0.005). Consequently, the prevalence of poor score was present only among controls (P 0.000). The prevalence of average score increased from 6.6% to 80% (P 0.000) in the intervention arm and also increased among controls (3.3% to 30%, P 0.005), but was significantly lower from the intervention arm subjects (P 0.0001). None of the subjects had good KAP score in control arm whereas, one fifth of the intervention arm subjects moved to the good category of KAP score (P 0.023) and was therefore significantly higher from controls (P 0.023) (fig 4.38).

TABLE 4.71: IMPACT OF NUTRITION COUNSELLING ON THE ATTITUDE AND PRACTICES OF TYPE 2 DIABETES AND NAFLD (MEAN ± SD)

Variables	Score	Stage	Control Group (N=30)	Experimental Group (N=30)	P Value
Attitude & practice scores on T2DM and NAFLD	0-13	Pre	6.9 ± 1.9	6.8 ± 2.5	0.82
		Post	7.7 ± 1.8	10.2 ± 2.5	**5.59E*** **
		Paired t	**0.006****	**5.99E*** **	

P<0.05*, P<0.01**, P<0.001***

FIG 4.37: IMPACT OF NUTRITION COUNSELLING ON ATTITUDES AND PRACTICES OF TYPE 2 DIABETICS WITH NAFLD (%)

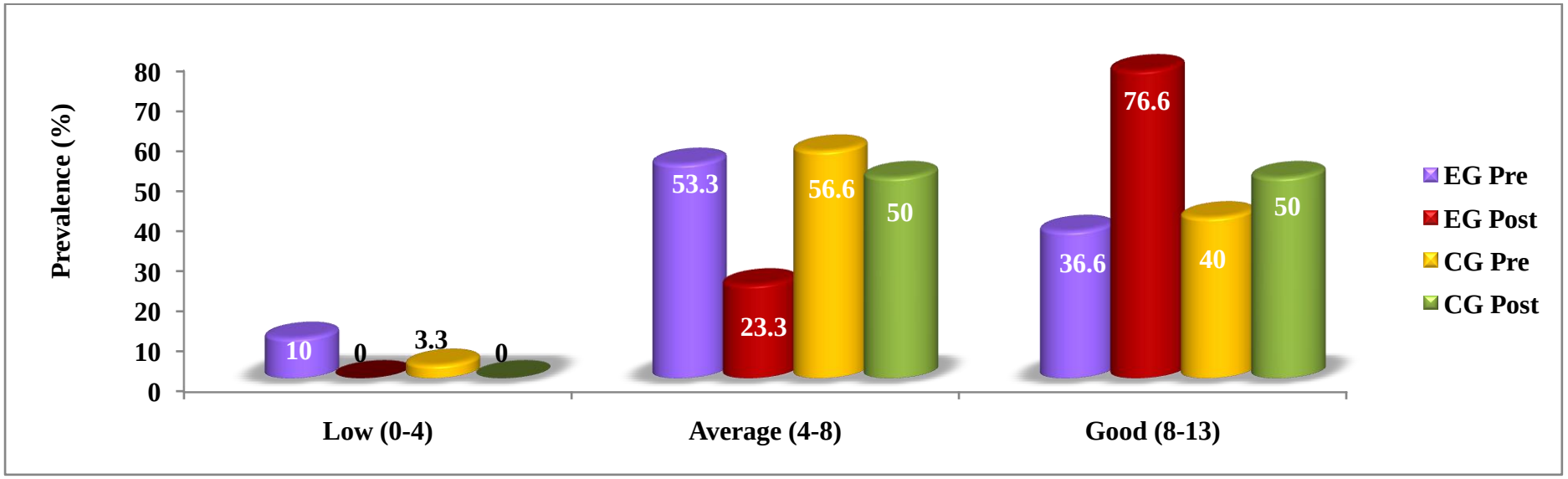
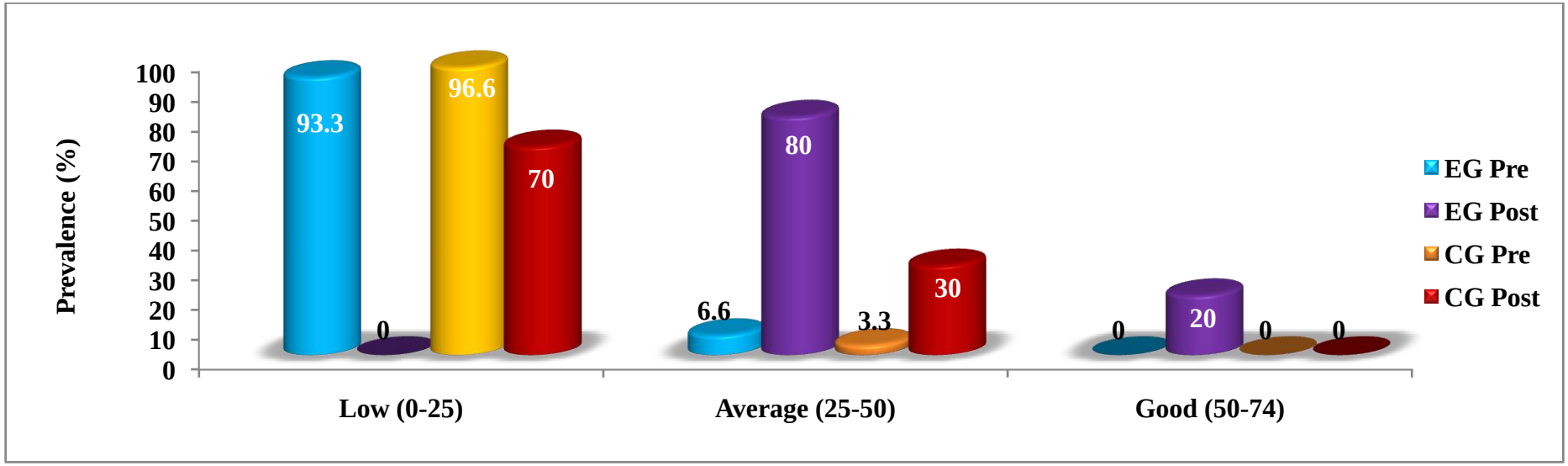


TABLE 4.72: IMPACT OF NUTRITION COUNSELLING ON THE KNOWLEDGE ATTITUDE AND PRACTICE SCORES OF TYPE 2 DIABETES AND NAFLD (MEAN ± SD)

Variable	Score	Stage	Control Group (N=30)	Experimental Group (N=30)	P Value
KAP SCORE	0-74	Pre	16.5 ± 5.4	16.6 ± 5.2	0.94
		Post	23.7 ± 7.09	44.03 ± 6.3	**8.72E*** **
		Paired t	**1.05E*** **	**1.8E*** **	

P<0.05*, P<0.01**, P<0.001***

FIG 4.38: IMPROVEMENT IN KAP SCORE AMONG TYPE 2 DIABETICS WITH NAFLD (%)



PHASE II (C): IMPACT OF LIFESTYLE MODIFICATION THERAPY IN THE MANAGEMENT OF NON-ALCOHOLIC FATTY LIVER DISEASE (NAFLD) IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

General Profile

Age and the duration of diabetes were similar for the experimental group and the control group (table 4.73). Most of the subjects in the experimental (43.3%) and the control arm (43.3%) were in the 50-60 years age category, followed by those in the 60-70 years age bracket.

An almost similar prevalence of type 2 diabetes in the family history was observed for the experimental (66.7%) and the control arm (63.3%). Those in the experimental arm had a higher prevalence of hypertension and cancer in their family and cardiac events in the family were more prevalent amongst those in the control arm.

Disease and Drug Profile

Non-significantly the prevalence of hypertension (60% vs. 53.3%) and hypothyroidism (26.7% vs. 16.7%) were more prevalent amongst those in the experimental arm and the prevalence of gout was similar in both the arms. But, thalassemia and rheumatoid arthritis were prevalent only amongst the controls and depression and asthma only amongst the experimental arm subjects (fig 4.39).

Most of the type 2 diabetic subjects with NAFLD were on OHAs, 90% in experimental arm and 86.6% in control arm. Based on the prevalent clinical conditions, the type 2 diabetic subjects with NAFLD were prescribed an array of medications ranging from dyslipidemic agents, beta blockers to thyroid hormones (table 4.74). Half of the experimental arm subjects and 46.6% controls were on various dyslipidemic agents. The intervention arm subjects were prescribed beta blockers significantly higher than the controls (40% vs. 13.3%, P 0.02).

Supplement Usage and Addiction Patterns

Supplements such as vitamin B complex, hypoglycaemic ayurvedic powders, calcium and vitamin D supplements were primarily being consumed more by the intervention arm subjects. Protein, iron and omega 3 fatty acids were only being taken as supplements exclusively by the experimental arm subjects. However, the subjects were asked to discontinue the supplements that would hinder with establishing the efficacy of lifestyle modification. In terms of addictions, only 6.7% of the controls were habituated to consuming tobacco products (table 4.74).

Oil Consumption Patterns

Cottonseed oil which comprises of 50% polyunsaturated fatty acids (PUFAs) and 25% saturated fatty acids (SFAs) was the most commonly consumed oil amongst the experimental group subjects (50%) and the controls (33.3%), followed by corn oil, groundnut oil and sunflower oil (table 4.75).

Dietary habits

Marginally higher proportions of subjects in the experimental arm were vegetarians than in the controls (83.3% vs. 76.6%). Likewise, marginally higher proportion subjects had non-vegetarian habits in the control arm than the experimental arm (20% vs. 16.6%) (table 4.76).

TABLE 4.73: GENERAL PROFILE OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (MEAN ± SD)

Variables	Control Group (N=30)	Experimental Group (N=30)	P value
Age (years)	56.23 ± 8.9	56.03 ± 8.7	0.93
30-40	0 (0)	1 (3.3)	1
40-50	6 (20)	5 (16.7)	0.74
50-60	13 (43.3)	13 (43.3)	1
60-70	7 (23.3)	9 (30)	0.56
>70	3 (10)	2 (6.7)	1
Duration of diabetes (years)	7 ± 6.1	6.9 ± 6.2	0.95

P<0.05*, P<0.01**, P<0.001***

FIG 4.39: DISEASE PROFILE OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (%)

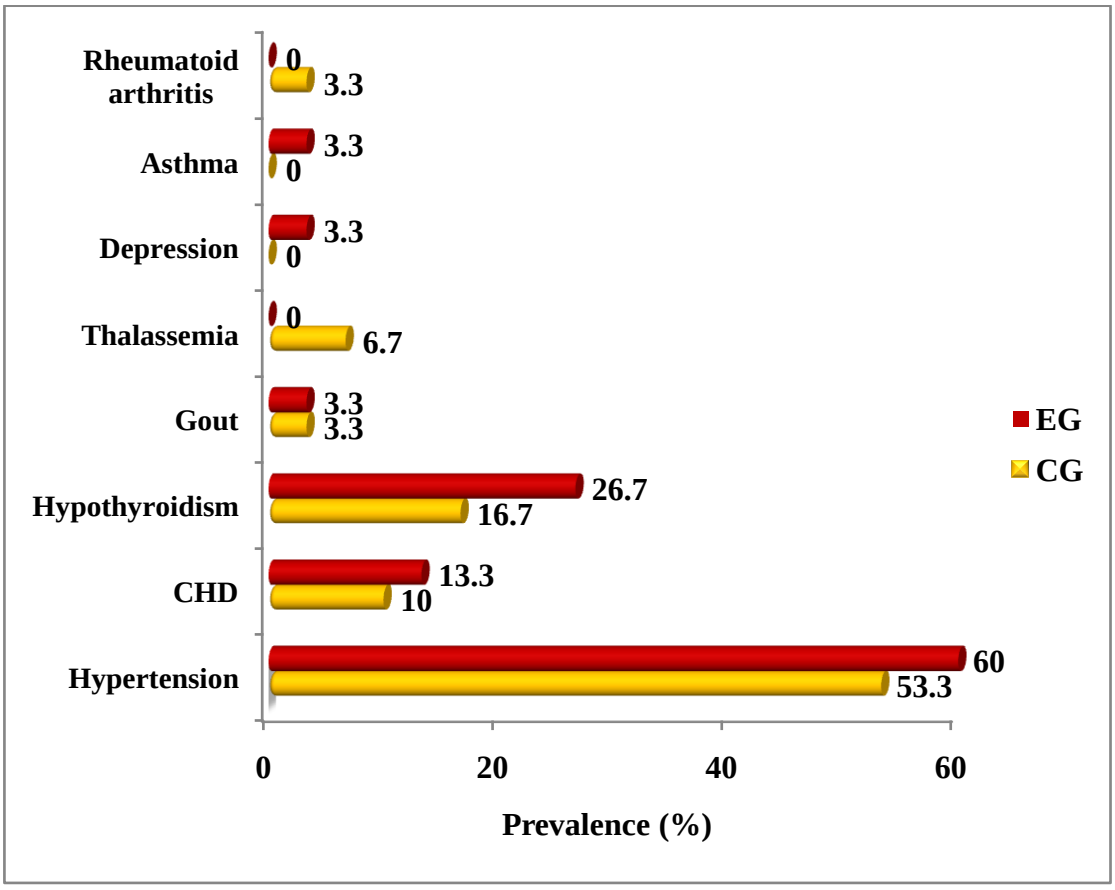


TABLE 4.74: DRUG PROFILE, SUPPLEMENT USAGE AND ADDICTIONS OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (N, %)

Drugs	Control Group (N=30)	Experimental Group (N=30)	P value
OHA	26 (86.7)	27 (90)	1
OHA + Insulin	4 (13.3)	3 (10)	1
Dyslipidemic agents	14 (46.7)	15 (50)	0.79
Anti-anginal agents	6 (20)	3 (10)	0.47
Anti-platelet agents	6 (20)	6 (20)	1
ACE inhibitor agents	1 (3.3)	1 (3.3)	1
Thyroid hormones	5 (16.7)	8 (26.7)	0.35
Angiotensin II antagonist agents	7 (23.3)	8 (26.7)	0.76
Beta blocker agents	4 (13.3)	12 (40)	**0.02***
NSAID agents	7 (23.3)	3 (10)	0.16
Anti-anemic agents	2 (6.7)	0 (0)	0.49
Anti-gout agents	1 (3.3)	1 (3.3)	1
Diuretic agents	0 (0)	3 (10)	0.23
Anti-depressant agents	0 (0)	1 (3.3)	1
Anti-asthmatic agents	0 (0)	1 (3.3)	1
Vitamin B complex	5 (16.7)	14 (46.7)	**0.013***
Hypoglycemic powder	1 (3.3)	6 (20)	0.10
Protein	0 (0)	1 (3.3)	1
Calcium and Vitamin D	8 (26.7)	10 (33.3)	0.57
Iron	0 (0)	2 (6.7)	0.49
Omega 3 fatty acids	0 (0)	1 (3.3)	1
Tobacco users	2 (6.7)	0 (0)	0.49
Smokers	0 (0)	0 (0)	-

Values in parenthesis indicate percentage

TABLE 4.75: OIL CONSUMPTION PATTERNS OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (N, %)

Oils	Control Group (N=30)	Experimental Group (N=30)	P value
Cottonseed	10 (33.3)	15 (50)	0.19
Corndrop	3 (10)	4 (13.3)	1
Groundnut	5 (16.7)	3 (10)	0.70
Sunflower	5 (16.7)	3 (10)	0.70
Safflower	1 (3.3)	0 (0)	1
Soyabean	0 (0)	1 (3.3)	1
Rice bran	1 (3.3)	0 (0)	1
Corndrop + sunflower	0 (0)	1 (3.3)	1
Corndrop + Safflower	1 (3.3)	0 (0)	1
Sunflower + Mustard	1 (3.3)	0 (0)	1
Rice bran + Mustard	2 (6.7)	0 (0)	0.49
Olive + Sunflower	0 (0)	1 (3.3)	1
Olive+Mustard+Sunflower	0 (0)	1 (3.3)	1
Rice bran+Sunflower+ Mustard	1 (3.3)	0 (0)	1
Sunflower + Corndrop + Rice bran + Mustard	0 (0)	1 (3.3)	1

Values in parenthesis indicate percentage

TABLE 4.76: DIETARY HABITS OF TYPE 2 DIABETES PATIENTS WITH NAFLD (N, %)

Dietary habits	Control group (N=30)	Experimental group (N=30)	P value
Vegetarian	23 (76.66)	25 (83.33)	0.52
Ovo-vegetarian	1 (3.33)	0 (0)	1
Non-vegetarian	6 (20)	5 (16.66)	0.74

Values in parenthesis indicate percentage

IMPACT OF LIFESTYLE MODIFICATION THERAPY IN THE MANAGEMENT OF NAFLD IN SUBJECTS WITH TYPE 2 DIABETES MELLITUS

Impact on Anthropometric and Blood Pressure Profile

At baseline, all the anthropometric measurements of the experimental arm and the control arm were similar. With the intervention, the weight declined non-significantly by 8.1% in the experimental arm (table 4.77). At the termination of the study, the weight of the experimental arm (65.8kg) became significantly lower (P 0.05) than the control arm (72.1kg). Owing to weight loss in the experimental arm, the BMI (7.9%), WC (5.2%) and WSR (6.2%) declined non-significantly and there was only a negligible decline in the control arm. At the point of termination, within the groups as well, there was no significant difference. The SBP was significantly higher (P 0.0001) in the experimental arm than the control arm at baseline (table 4.78). SBP was significantly higher than the controls at 1st month (P 0.0002) and in the 3rd month (P 0.048). At the end of the study, the SBP of the intervention arm subjects reduced significantly (145.2 to 128.1mmHg, P 3.23E). The SBP declined significantly from baseline to 1st month (P 0.03), 2nd month (P 0.004), 3rd month (P 0.0003) and at the 4th month (P 4.52E) (table 4.12). The SBP also declined significantly from 1st month to 3rd month (P 0.016) and 4th month (P 2.82E). SBP also significantly declined from 2nd month to 4th month (P 0.028) (table 4.79). No alterations in DBP were observed either between or within groups.

Impact on Nutritional Status

After the intervention, the proportion of normal BMI increased in experimental arm as well as in control group by 10%. The prevalence of overweight declined from 20% to 13.3% in the control group and the figures remained static in the experimental arm. Prevalence of obesity was similar in both the groups at baseline. After the intervention, there was a 10% drop in the experimental arm and 3.3% in controls. A 13.3% decline in elevated WC was witnessed in the experimental arm as against 3.3% decline in the control arm. Similarly, elevated WSR dropped by 16.7% in the experimental group and 6.7% in the controls (fig 4.40).

Impact on Weight Alterations Based on ≥7% Weight Loss

Weight loss ≥7% was significantly evident in subjects of the intervention arm compared to controls (60% vs. 13.3%, P 0.0001). Similarly, weight loss <7% was pronounced in controls than the intervention arm subjects (66.7% vs. 40%, P 0.040). Weight gain was observed only in the subjects of the control arm, wherein 16.7% gained <7% weight and 3.3% gained ≥7% weight (fig 4.41).

Impact on Nutrient Intake

Subjects of the intervention arm though had a non-significant decline in energy, carbohydrate, fat intake and a non-significant increase in protein, crude fibre, total dietary fibre, insoluble dietary fibre intake, the changes were more profound than the controls (table 4.80). Fat intake was significantly lower in intervention arm subjects than the controls at the 1st month (45.8g vs. 52.6g, P 0.029). The soluble fibre intake increased significantly with the intervention (P 0.041) (fig 4.42). It was significantly higher in the 3rd month compared to baseline (3.7g vs. 2.9g, P 0.042) and 2nd month (3.3g vs. 2.9g, P 0.020). However, the soluble fibre content was the highest in the 4th month which was significantly higher from the soluble fibre content of the 2nd month (3.8g vs. 2.9g, P 0.028) (table 4.81).

The proportion of energy coming from carbohydrates remained more or less the same, ranging from 53.1% to 56.7%, from protein increased from 11.2% to 12.4% and that of fat reduced from 32.8% to 29.6% (table 4.82). The proportion of fat was significantly lower in the intervention arm compared to the controls at baseline (32.8% vs. 35.4%, P 0.011) and at 1st month (29.05% vs. 33.1%, P 0.028).

Impact on Frequency of Eating Out

Prevalence of weekly eating out declined more in the intervention arm than the controls. The proportion of those eating out ‘rarely’; increased in the experimental arm and remained static in the control arm at the end of the study. The percentage also increased amongst those who went to eat out on a fortnightly basis in both the arms at the termination of the study. Those eating out once a month also declined in both the arms (fig 4.43).

Impact on Iron Profile

At baseline, the mean iron, TIBC, transferrin saturation and ferritin had a non-significant difference. The TIBC reduced significantly (P 0.03) from 355.4 to 347.4mcg/dl in the experimental arm post intervention. Ferritin declined significantly (P 0.025) from 67.1 to 50.7ng/ml in the experimental arm after the intervention. Although the control arm also had a decline, it was not significant. The other parameters of iron status remained unaltered (table 4.83).

Impact on Renal Profile

The parameters of renal function were similar in both the groups at baseline. However, the calcium levels of the control arm were significantly lower than that of the experimental arm at the beginning of the study (9.5 vs. 9.7 mg/dl, P 0.009). At the termination of the study, a significant decline in the calcium levels (9.7 to 9.6mg/dl, P 0.012) was observed in the experimental arm after the intervention but it had no significant difference compared to the post value of the control arm. The other parameters remained unaltered (table 4.84).

Impact on Thyroid Profile

Thyroid profile was similar at baseline in both the groups. However, T3 declined significantly (105.4 to 98.2ng/dl, P 0.0015) in the experimental arm (table 4.85).

TABLE 4.77: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON ANTHROPOMETRIC PROFILE OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (MEAN ± SD)

Variable	Timeline (Months)	Control Group (N=30)	Experimental Group (N=30)	P value
Weight (kg)	0	73.9 ± 11.4	71.6 ± 12.9	0.47
	1	73.5 ± 11.7	70.4 ± 12.6	0.31
	2	73 ± 11.7	69 ± 12.3	0.20
	3	72.3 ± 11.9	67.5 ± 12.3	0.13
	4	72.1 ± 12	65.8 ± 12.3	**0.05***
	F value	0.96 (↓2.4%)	0.39 (↓8.1%)	
BMI (kg/m^2)	0	28.6 ± 5.3	28.8 ± 5.5	0.9
	1	28.5 ± 5.3	28.3 ± 5.4	0.88
	2	28.3 ± 5.3	27.8 ± 5.3	0.72
	3	28.1 ± 5.4	27.2 ± 5.3	0.56
	4	28 ± 5.5	26.5 ± 5.3	0.3
	F value	0.98 (↓2.09%)	0.5 (↓7.9%)	
WC (cm)	0	98.7 ± 10.9	101.2 ± 9.03	0.34
	1	98.6 ± 10.9	100.3 ± 8.8	0.49
	2	98.1 ± 10.9	98.9 ± 8.3	0.73
	3	97.7 ± 11	97.6 ± 8.3	0.95
	4	97.5 ± 11	95.9 ± 9	0.54
	F value	0.99 (↓1.2%)	0.14 (↓5.2%)	
WSR	0	0.61 ± 0.08	0.64 ± 0.06	0.15
	1	0.61 ± 0.08	0.63 ± 0.06	0.22
	2	0.61 ± 0.08	0.62 ± 0.06	0.35
	3	0.6 ± 0.08	0.62 ± 0.06	0.55
	4	0.6 ± 0.08	0.6 ± 0.06	0.91
	F value	0.99 (↓1.6%)	0.31 (↓6.2%)	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis depict proportion

TABLE 4.78: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON BLOOD PRESSURE OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (MEAN ± SD)

Variable	Timeline (Months)	Control Group (N=30)	Experimental Group (N=30)	P value
SBP (mmHg)	0	129.9 ± 12.3	145.2 ± 16.4	**0.0001*** **
	1	129.1 ± 7.9	137.7 ± 8.1	**0.0002*** **
	2	130.2 ± 6.7	133.9 ± 12.7	0.16
	3	127.8 ± 6.9	132.1 ± 9.3	**0.048***
	4	129.4 ± 6.6	128.1 ± 6	0.41
	F value	0.83 (↓0.38%)	**3.23E*** (↓11.7%)**	
DBP (mmHg)	0	83.3 ± 8.2	87.6 ± 8.8	0.056
	1	84.9 ± 8.1	85.2 ± 9.4	0.9
	2	85.7 ± 4.7	84.8 ± 12.1	0.66
	3	84 ± 6.4	84.6 ± 7.9	0.73
	4	86.1 ± 6.1	85.5 ± 5.7	0.69
	F value	0.48 (↑3.3%)	0.68 (↓2.4%)	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis depict proportion

TABLE 4.79: DIFFERENCE IN SBP IN EXPERIMENTAL GROUP

Groups	P value SBP
Baseline vs. 1st month	**0.03***
Baseline vs. 2nd month	**0.004****
Baseline vs. 3rd month	**0.0003*****
Baseline vs. 4th month	**4.52E*****
1st month vs. 2nd month	0.17
1st month vs. 3rd month	**0.016***
1st month vs. 4th month	**2.82E*****
2nd month vs. 3rd month	0.54
2nd month vs. 4th month	**0.028***
3rd month vs. 4th month	0.051

P<0.05*, P<0.01**, P<0.001***

FIG 4.40: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON NUTRITIONAL STATUS OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (%)

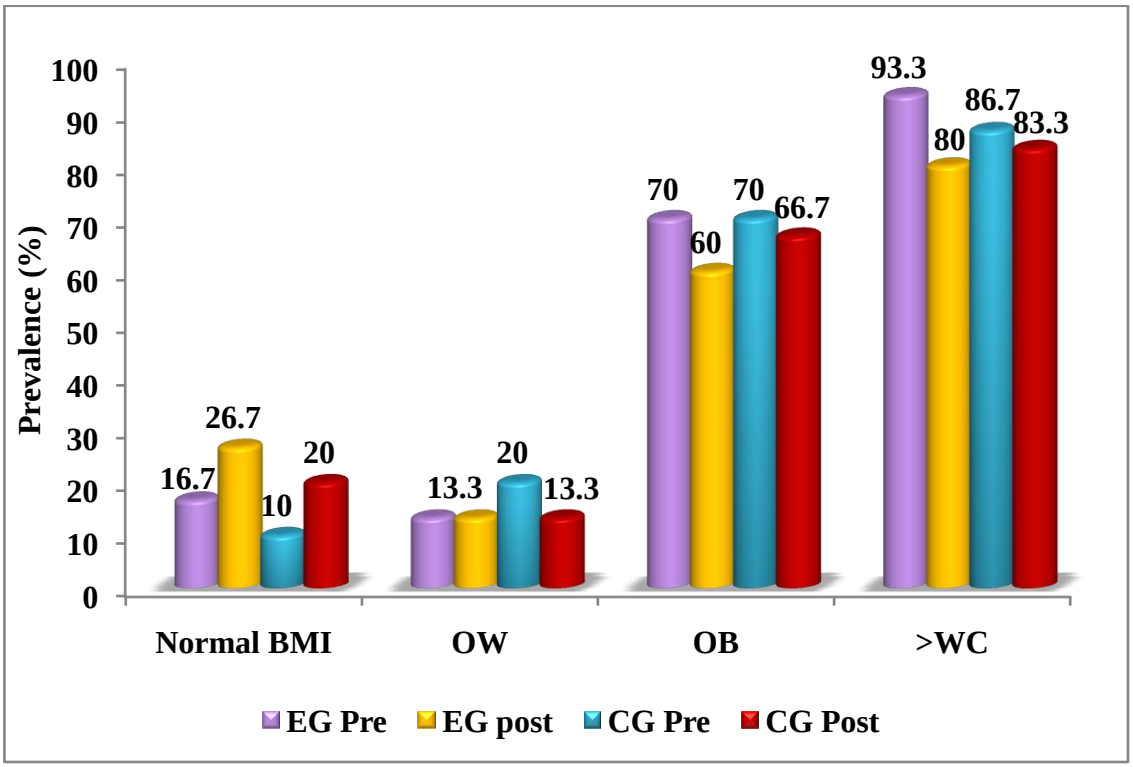


FIG 4.41: WEIGHT ALTERATIONS BASED ON ≥7% OF TYPE 2 DIABETES PATIENTS WITH NAFLD (%)

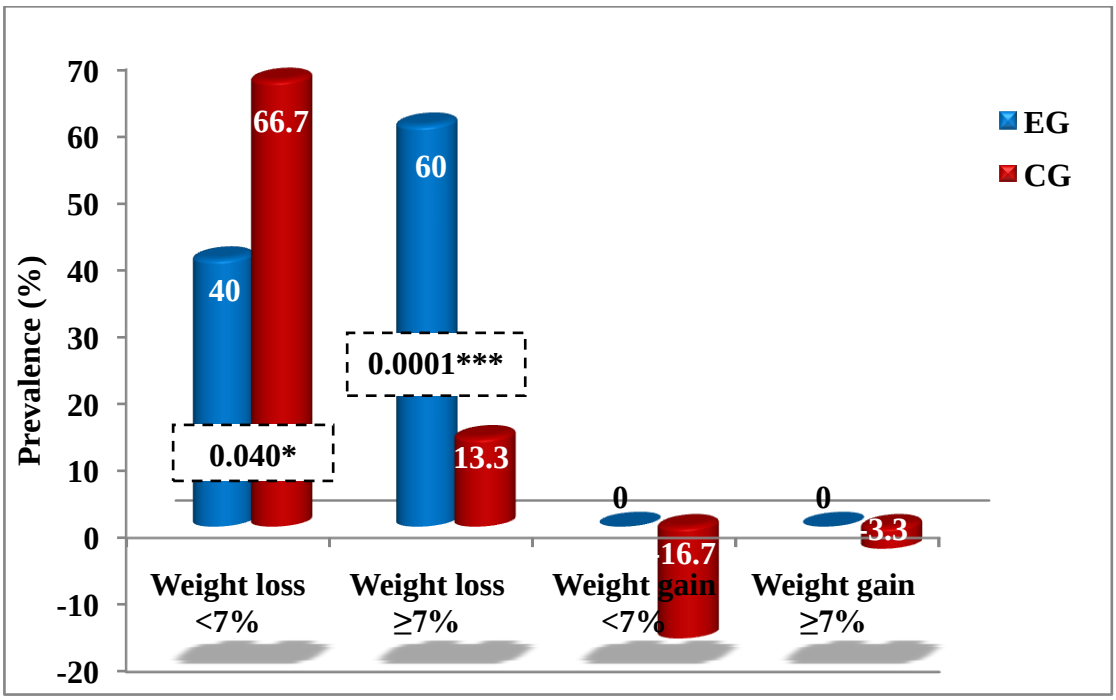


TABLE 4.80: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON THE NUTRIENT INTAKE OF TYPE 2 DIABETES PATIENTS WITH NAFLD (MEAN±SD)

Variable	Timeline (Months)	Control Group (N=30)	Experimental Group (N=30)	P value
Energy (kcal)	0	1464 ± 197	1483 ± 185	0.51
	1	1441 ± 205	1432 ± 160	0.86
	2	1450 ± 212	1391 ± 118	0.19
	3	1478 ± 204	1424 ± 100	0.20
	4	1474 ± 231	1436 ± 111	0.42
	F value	0.95	0.06	
Carbohydrates (g)	0	187.6 ± 36.5	197.3 ± 35.8	0.12
	1	192.5 ± 38.6	203.8 ± 39.9	0.26
	2	191.9 ± 40.9	193.1 ± 34.8	0.90
	3	197.5 ± 42.1	194.5 ± 30.8	0.75
	4	189.7 ± 45.3	193.7 ± 35.1	0.70
	F value	0.91	0.64	
Fat (g)	0	57.1 ± 10.3	53.8 ± 10.2	0.062
	1	52.6 ± 13.5	45.8 ± 9.5	**0.029***
	2	51.4 ± 11.5	46.1 ± 11.06	0.076
	3	51.5 ± 10.4	46.6 ± 11.7	0.097
	4	51.4 ± 13.1	48.8 ± 10.9	0.42
	F value	0.28	0.10	
Protein (g)	0	40.1 ± 8.7	41.7 ± 7.7	0.46
	1	42 ± 9.7	43.7 ± 8.68	0.48
	2	41.6 ± 8.5	42.2 ± 8.37	0.80
	3	42.9 ± 8.9	42.8 ± 8.64	0.41
	4	42.9 ± 7.5	46.4 ± 8.4	0.07
	F value	0.71	0.17	

P<0.05*, P<0.01**, P<0.001***

TABLE 4.80: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON THE NUTRIENT INTAKE OF TYPE 2 DIABETES PATIENTS WITH NAFLD (MEAN±SD)

Variable	Timeline (Months)	Control Group (N=30)	Experimental Group (N=30)	P value
Crude Fibre (g)	0	5.4 ± 1.5	5.8 ± 1.9	0.37
	1	5.6 ± 1.4	6.2 ± 2.1	0.29
	2	5.4 ± 1.8	5.3 ± 1.6	0.75
	3	5.6 ± 1.7	6.2 ± 1.9	0.15
	4	5.6 ± 2.1	6.3 ± 1.9	0.16
	F value	0.96	0.20	
Calcium (mg)	0	643.9 ± 272	622.1 ± 255.1	0.69
	1	580.7 ± 244.8	566.9 ± 231.8	0.82
	2	554.4 ± 215.4	613.8 ± 235.2	0.31
	3	580.4 ± 210.1	620.1 ± 260.3	0.51
	4	552.4 ± 186.8	696.1 ± 265.6	0.11
	F value	0.53	0.39	
Iron (mg)	0	12.3 ± 5.6	12.1 ± 4.2	0.99
	1	13.6 ± 6.2	12.5 ± 3.1	0.39
	2	11.9 ± 3.8	12.2 ± 4.8	0.73
	3	12.6 ± 3.7	13.2 ± 3.9	0.51
	4	12.2 ± 3.4	13.1 ± 3.7	0.28
	F value	0.64	0.78	
Vitamin A (Mcg)	0	119.9 ± 56.4	111.2 ± 57.1	0.49
	1	128.1 ± 56.5	108.5 ± 59.4	0.077
	2	135 ± 60.3	103.1 ± 53.2	**0.033***
	3	124.3 ± 58.5	101.9 ± 54.7	0.30
	4	120.5 ± 61.9	119.2 ± 52.5	0.92
	F value	0.85	0.76	

P<0.05*, P<0.01**, P<0.001***

TABLE 4.80: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON THE NUTRIENT INTAKE OF TYPE 2 DIABETES PATIENTS WITH NAFLD (MEAN±SD)

Variable	Timeline (Months)	Control Group (N=30)	Experimental Group (N=30)	P value
Vitamin C (mg)	0	69.8 ± 52.5	66.9 ± 58.6	0.96
	1	72.9 ± 58	65.1 ± 57.8	0.46
	2	76.3 ± 59.2	65.2 ± 49.1	0.59
	3	51.2 ± 47.5	78.8 ± 73.1	0.73
	4	58.3 ± 40.2	71.6 ± 67.6	0.18
	F value	0.29	0.91	
Total dietary fibre (g)	0	12.6 ± 4.5	12.9 ± 4.9	0.37
	1	12.4 ± 5.3	14.3 ± 5.5	0.17
	2	11.9 ± 4.4	13.08 ± 3.8	0.30
	3	12.3 ± 6.1	15.05 ± 5.9	0.08
	4	12.4 ± 5.7	15.6 ± 7.3	0.063
	F value	0.99	0.35	
Insoluble dietary fibre (g)	0	9.4 ± 4.1	9.9 ± 4	0.33
	1	9.3 ± 3.3	11.03 ± 4.4	0.067
	2	9.1 ± 3.8	10.1 ± 3.2	0.33
	3	9.1 ± 4.2	11.3 ± 4.4	0.056
	4	9.4 ± 4.5	11.8 ± 5.5	0.067
	F value	0.99	0.55	
Soluble dietary fibre (g)	0	2.7 ± 1.2	2.9 ± 1.1	0.26
	1	3.1 ± 1.3	3.3 ± 1.2	0.52
	2	2.7 ± 1	2.9 ± 0.8	0.47
	3	2.8 ± 1.1	3.7 ± 1.5	**0.009****
	4	2.8 ± 1	3.8 ± 1.8	**0.014***
	F value	0.77	**0.041***	

P<0.05*, P<0.01**, P<0.001***

FIG 4.42: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON SOLUBLE DIETARY FIBRE INTAKE OF TYPE 2 DIABETES PATIENTS WITH NAFLD (MEAN±SD)

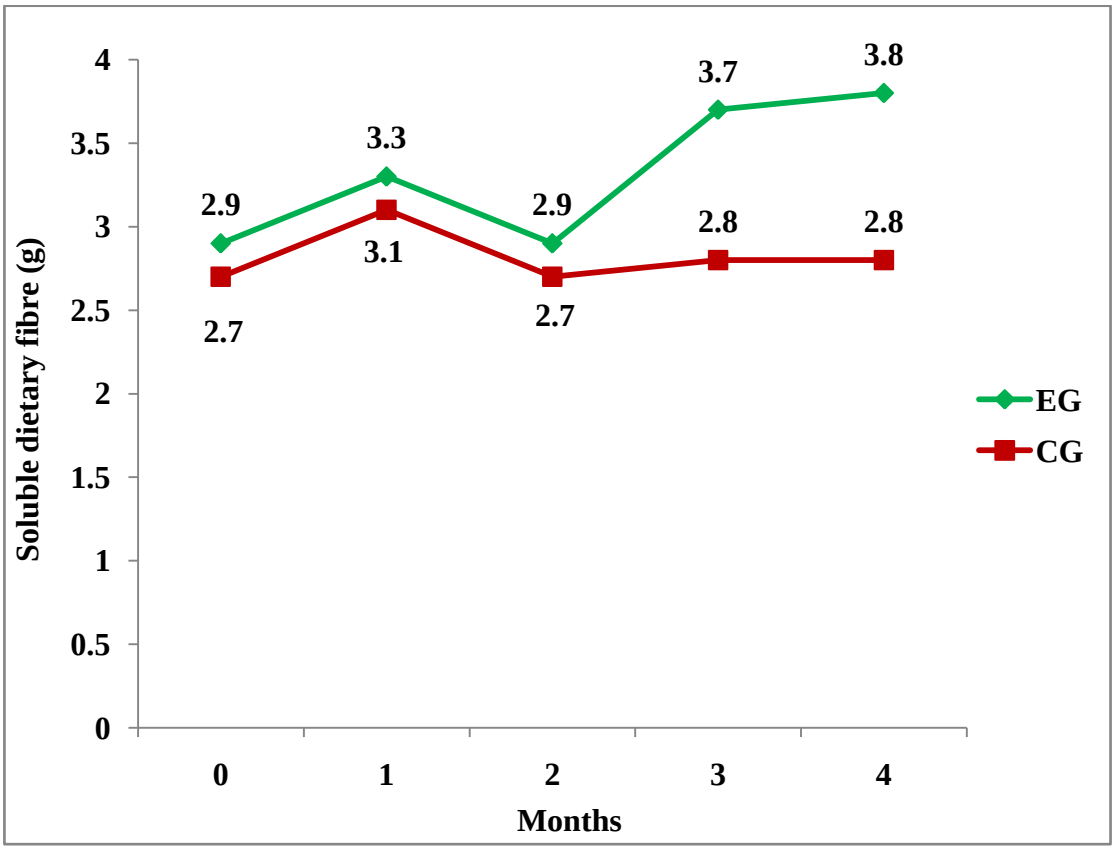


TABLE 4.81: DIFFERENCE IN SOLUBLE FIBRE INTAKE IN EXPERIMENTAL ARM SUBJECTS

Groups	P value Soluble fibre
Baseline vs. 1st month	0.38
Baseline vs. 2nd month	0.72
Baseline vs. 3rd month	**0.042***
Baseline vs. 4th month	0.051
1st month vs. 2nd month	0.22
1st month vs. 3rd month	0.21
1st month vs. 4th month	0.20
2nd month vs. 3rd month	**0.020***
2nd month vs. 4th month	**0.028***
3rd month vs. 4th month	0.87

P<0.05*, P<0.01**, P<0.001***

TABLE 4.82: PERCENT DISTRIBUTION OF MACRONUTRIENTS OF TYPE 2 DIABETES PATIENTS WITH NAFLD (MEAN±SD)

Variable	Timeline (Months)	Control Group (N=30)	Experimental Group (N=30)	P value
Carbohydrates (%)	0	51.1 ± 5.6	53.1 ± 5.6	0.06
	1	53.4 ± 7.3	56.7 ± 7.4	0.085
	2	52.9 ± 9.2	55.3 ± 7.6	0.27
	3	53.2 ± 7.3	54.6 ± 7.9	0.46
	4	51.3 ± 8.2	53.8 ± 8.2	0.22
	F value	0.63	0.54	
Protein (%)	0	10.8 ± 1.4	11.2 ± 1.3	0.14
	1	11.6 ± 2.3	12.1 ± 1.7	0.35
	2	11.5 ± 1.7	12.07 ± 1.8	0.21
	3	11.5 ± 1.4	12.1 ± 3.1	0.36
	4	11.7 ± 2.2	12.4 ± 2.9	0.31
	F value	0.32	0.44	
Fat (%)	0	35.4 ± 5.7	32.8 ± 5.4	**0.011***
	1	33.1 ± 10.3	29.05 ± 6.3	**0.028***
	2	32.2 ± 6.7	30.05 ± 7.3	0.25
	3	31.8 ± 6.5	28.5 ± 9.07	0.12
	4	31.9 ± 7.4	29.6 ± 8.7	0.35
	F value	0.19	0.56	

P<0.05*, P<0.01**, P<0.001***

FIG 4.43: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON EATING OUT AMONG TYPE 2 DIABETES SUBJECTS WITH NAFLD (%)

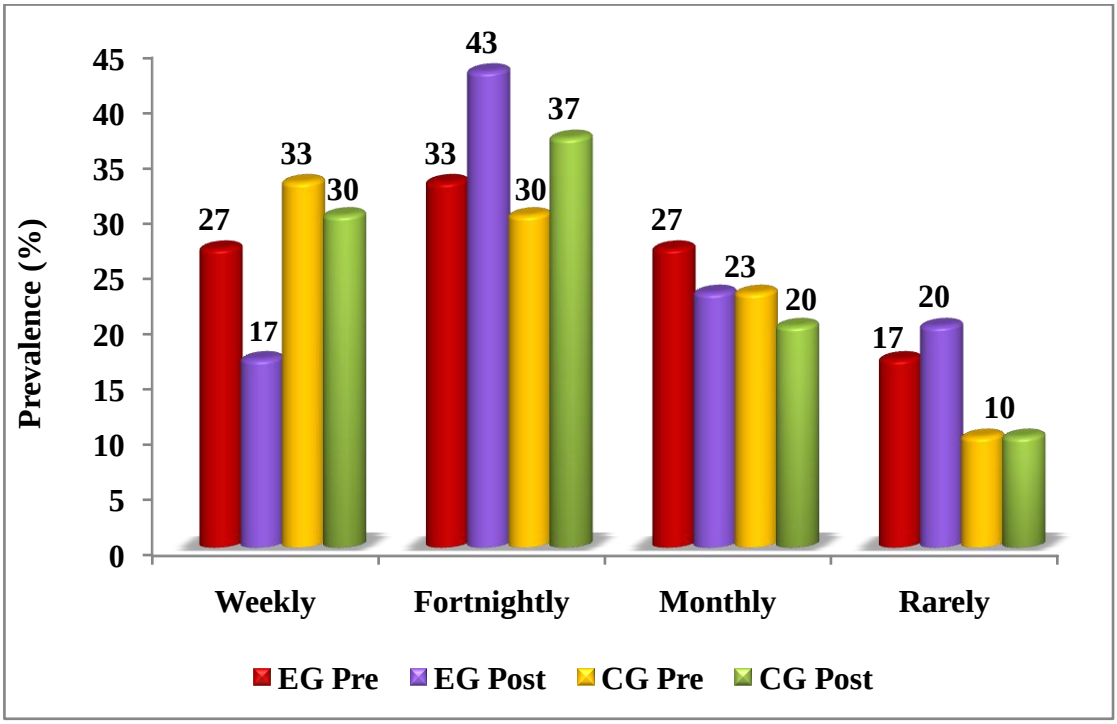


TABLE 4.83: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON IRON PROFILE OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (MEAN±SD)

Variables	Stage	Control Group (N=30)	Experimental Group (N=30)	P value
Iron (mcg/dl)	Pre	64.6 ± 18.4	74.7 ± 33.1	0.15
	Post	71.1 ± 23.5	75.4 ± 34.7	0.58
	Paired t	0.09 (↑10%)	0.89 (↑0.93%)	
TIBC (mcg/dl)	Pre	355.8 ± 49.3	355.4 ± 44.03	0.97
	Post	362.6 ± 48.4	347.4 ± 38.4	0.18
	Paired t	0.37 (↑1.9%)	**0.03* (↓2.3%)**	
Transferrin saturation (%)	Pre	18.4 ± 5.4	21.2 ± 9.07	0.14
	Post	20 ± 6.7	22.2 ± 10.6	0.33
	Paired t	0.14 (↑8.69%)	0.46 (↑4.7%)	
Ferritin (ng/ml)	Pre	63.7 ± 63.4	67.1 ± 68.6	0.84
	Post	53.9 ± 54.8	50.7 ± 41.7	0.8
	Paired t	0.31 (↓15.3%)	**0.025* (↓24.4%)**	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis depict proportion

TABLE 4.84: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON RENAL PROFILE OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (MEAN ± SD)

Variables	Stage	Control Group (N=30)	Experimental Group (N=30)	P value
BUN (mg/dl)	Pre	10.1 ± 3.4	10.9 ± 2.8	0.37
	Post	10.3 ± 3	10.7 ± 2.7	0.54
	Paired t	0.76 (↑1.9%)	0.76 (↓1.8%)	
Creatinine (mg/dl)	Pre	0.65 ± 0.11	0.7 ± 0.22	0.21
	Post	0.66 ± 0.12	0.7 ± 0.2	0.28
	Paired t	0.82 (↑1.5%)	0.89 (0%)	
Uric acid (mg/dl)	Pre	5.3 ± 1.2	5.5 ± 1.5	0.49
	Post	5.4 ± 1.4	5.5 ± 1.2	0.6
	Paired t	0.79 (↑1.8%)	0.98 (0%)	
Calcium (mg/dl)	Pre	9.5 ± 0.3	9.7 ± 0.28	**0.009****
	Post	9.5 ± 0.3	9.6 ± 0.3	0.33
	Paired t	0.97 (0%)	**0.012* (↓1.03%)**	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis depict proportion

TABLE 4.85: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON THYROID PROFILE OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (MEAN ± SD)

Variables	Stage	Control Group (N=30)	Experimental Group (N=30)	P value
T3 (ng/dl)	Pre	103.4 ± 15.8	105.4 ± 18.5	0.65
	Post	100.7 ± 15.5	98.2 ± 14.9	0.53
	Paired t	0.27 (↓2.6%)	**0.0015** (↓6.8%)**	
T4 (mcg/dl)	Pre	9.4 ± 1.7	9.3 ± 1.5	0.86
	Post	9.4 ± 1.8	9.3 ± 1.5	0.84
	Paired t	0.98 (0%)	0.94 (0%)	
TSH (microIU/ml)	Pre	3.3 ± 1.9	5.02 ± 5.4	0.13
	Post	3.5 ± 2.1	3.8 ± 2.8	0.63
	Paired t	0.66 (↑6.06%)	0.15 (↓24.3%)	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis depict proportion

Impact on Lipid Profile

The baseline value of TC and LDL-C of both the arms was similar. After the intervention, even though the experimental arm had a decline and the control arm saw a rise, the changes were not statistically significant either within or between the groups for both the variables (table 4.86). The HDL-C fraction was alike in both the groups at baseline. Post intervention, the HDL-C increased significantly from 47.2 to 52.2mg/dl (P 3.6E) for the subjects in the experimental arm and also became significantly higher (P 0.049) than the controls (46.7mg/dl) (fig 4.44). The triglycerides and VLDL-C at baseline did not differ significantly between the groups. However, at the termination of the study, triglycerides declined from 138.7 to 121.5mg/dl (P 0.031) and VLDL-C declined from 27.7 to 23.9mg/dl (P 0.021) in the experimental arm and even though the control arm also had a dip, it was not significant. Non-HDL-C declined non-significantly in the experimental arm and increased non-significantly in the control arm at the end of intervention.

Impact on Prevalence of Dyslipidemia

The prevalence of elevated LDL-C (>100mg/dl) declined significantly in the experimental arm from 63.3% to 36.7% (P 0.04) and the control arm had an increase in prevalence from 53.3% to 63.3% after the intervention period. Post intervention, the prevalence of high LDL-C became significantly lower in the experimental arm than the control arm (36.7% vs. 63.3%, P 0.04). After the intervention, the prevalence of hypercholesterolemia declined in the experimental arm from 26.7% to 20% and increased in the control arm from 30% to 33.3%. At the termination of the study, the prevalence of low HDL-C remained unaltered at 50% in the control arm but dropped from 46.7% to 20% in the experimental arm. An increase in the prevalence of hypertriglyceridemia was observed in experimental arm from 30% to 33.3% and in the control arm from 36.7% to 40% (fig 4.45).

Impact on Lipid Ratios

TG/H, AIP, TC/HDL, LDL/HDL, nonHDL/HDL were similar at baseline for the experimental arm and the control arm. At the termination of the study, elevated TG/H declined by 20.3% (P 0.0011), AIP>0.21 by 25% (P 0.0005), elevated TC/HDL by

7.7% (P 0.023), high LDL/HDL by 17.4% (P 0.012) and high nonHDL/HDL by 14.7% (P 0.0010) in the experimental arm, whereas there was no significant change observed in the control arm. The LDL/HDL (P 0.009) and nonHDL/HDL (P 0.022) of the experimental arm was also significantly lower than the control arm at the end of the study (table 4.87).

The prevalence of TG/H>3 declined in the experimental arm from 43.3% to 33.3% and from 46.7% to 40% in the control arm after the intervention. Likewise, the prevalence of elevated AIP declined from 90% to 70% in the experimental arm but increased from 80% to 83.3% in the control arm. The prevalence of elevated TC/HDL remained static at 16.7% pre and post intervention, but declined from 13.3% to 10% in the experimental arm. None of the experimental arm subjects had LDL/HDL >3.5 either prior to or after intervention, but the prevalence increased from 3.3% to 13.3% in the control arm (fig 4.46).

Impact on Hs-CRP

The hs-CRP at baseline did not differ significantly between the groups. However, at the termination of the study, it declined significantly in the experimental arm (P 0.024) from 4.6 to 3.4mg/l and even though the control arm also had a dip, it was not statistically significant (table 4.88). High risk CVD prevalence declined from 66.7% to 53.3% in the control arm and from 56.7% to 36.7% in the experimental arm after the intervention (fig 4.47). The prevalence of low risk CVD increased in the experimental arm from 13.3% to 26.7% and in the control arm from 6.7% to 20% after the intervention. However, the prevalence of medium risk CVD remained constant at 26.7% pre and post intervention, but increased in the experimental arm from 30% to 36.7%.

TABLE 4.86: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON LIPID PROFILE OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (MEAN ± SD)

Variables	Stage	Control Group (N=30)	Experimental Group (N=30)	P value
TC (mg/dl)	Pre	180.1 ± 41.4	179.1 ± 35.5	0.92
	Post	184.2 ± 35.8	175.8 ± 42.8	0.41
	Paired t	0.5 (↑2.3%)	0.56 (↓1.8%)	
LDL-C (mg/dl)	Pre	104.7 ± 38.4	104.2 ± 28.5	0.95
	Post	110.6 ± 31.8	96.7 ± 28.5	0.09
	Paired t	0.39 (↑5.6%)	0.14 (↓7.1%)	
Triglycerides (mg/dl)	Pre	143.4 ± 80.8	138.7 ± 50.8	0.78
	Post	138.2 ± 54.1	121.5 ± 48.8	0.21
	Paired t	0.64 (↓3.6%)	**0.031* (↓22.2%)**	
VLDL – C (mg/dl)	Pre	28.6 ± 16.1	27.7 ± 10.3	0.78
	Post	27.8 ± 11.02	23.9 ± 10.1	0.16
	Paired t	0.69 (↓2.8%)	**0.021* (↓13.7%)**	
Non-HDL-C (mg/dl)	Pre	133.4 ± 40.3	132.2 ± 32.7	0.90
	Post	137.5 ± 32.9	123.6 ± 42.2	0.16
	Paired t	0.54 (↑3.07%)	0.12 (↓6.5%)	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis depict proportion

FIG 4.44: IMPACT ON HDL-C OF TYPE 2 DIABETICS WITH NAFLD (MEAN ± SD)

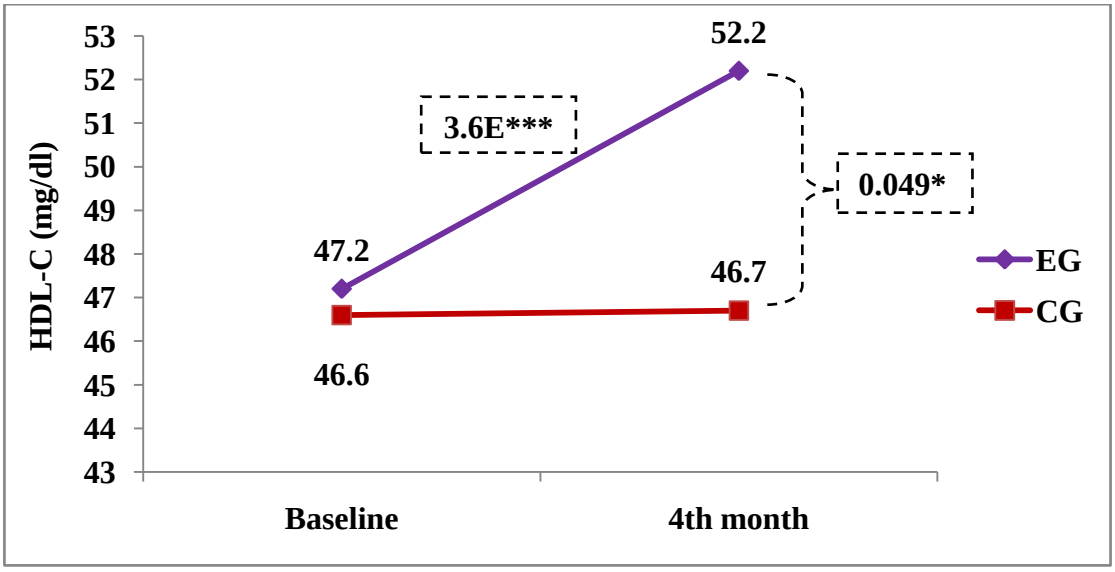


FIG 4.45: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON PREVALENCE OF DYSLIPIDEMIA AMONG TYPE 2 DIABETES SUBJECTS WITH NAFLD (N, %)

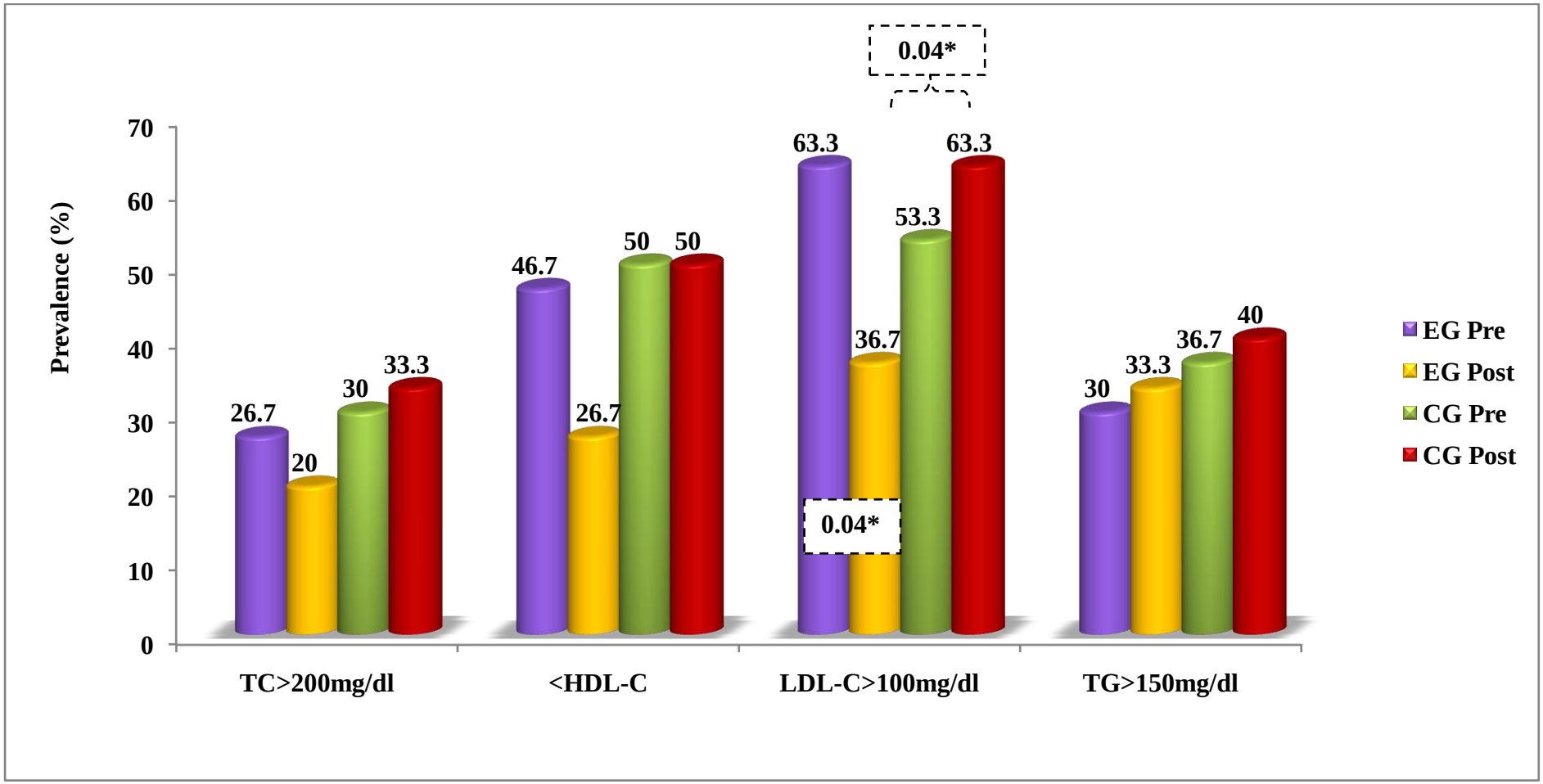


TABLE 4.87: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON LIPID RATIOS OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (MEAN±SD)

Variables	Stage	Control Group (N=30)	Experimental Group (N=30)	P value
TG/H	Pre	3.4 ± 2.6	3.14 ± 1.56	0.63
	Post	3.2 ± 1.6	2.5 ± 1.32	0.09
	Paired t	0.5 (↓5.9%)	**0.0011** (↓20.3%)**	
AIP	Pre	0.44 ± 0.26	0.44 ± 0.21	0.94
	Post	0.44 ± 0.20	0.33 ± 0.24	0.065
	Paired t	0.93 (0%)	**0.0005*** (↓25%)**	
TC/HDL	Pre	3.9 ± 1	3.9 ± 0.87	0.92
	Post	4 ± 0.88	3.6 ± 1.0	0.057
	Paired t	0.4 (↑2.5%)	**0.023* (↓7.7%)**	
LDL/HDL	Pre	2.1 ± 0.72	2.3 ± 0.62	0.42
	Post	2.4 ± 0.82	1.9 ± 0.67	**0.009****
	Paired t	0.08 (↑14.3%)	**0.012* (↓17.4%)**	
Non HDL-C/HDL-C	Pre	2.98 ± 1.07	2.91 ± 0.87	0.77
	Post	3.05 ± 0.88	2.48 ± 1.0	**0.022***
	Paired t	0.71 (↑2.3%)	**0.0010** (↓14.7%)**	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis depict proportion

FIG 4.46: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON ELEVATED LIPID RATIOS OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (%)

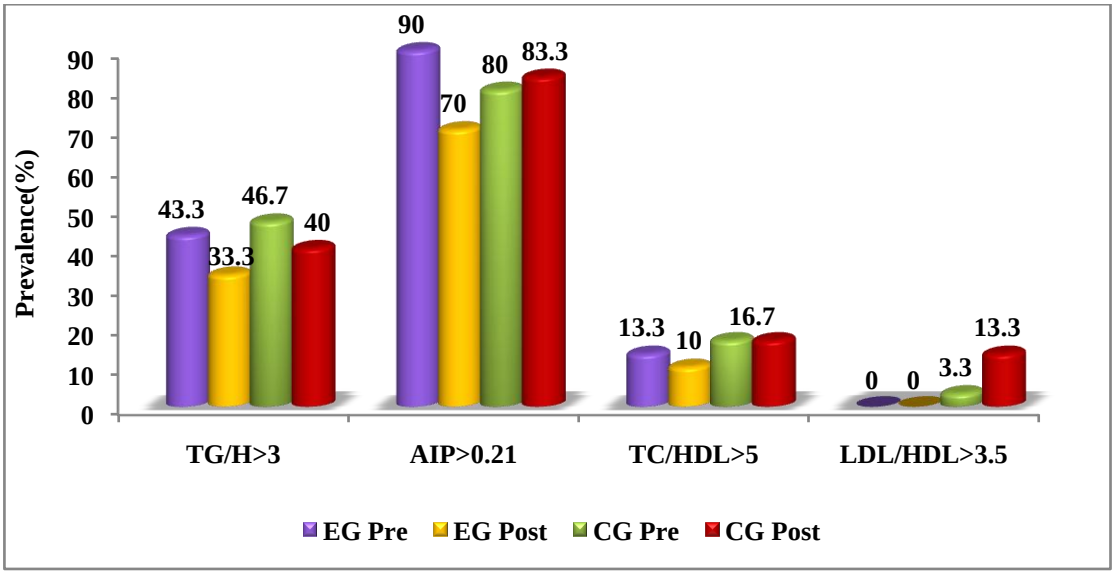
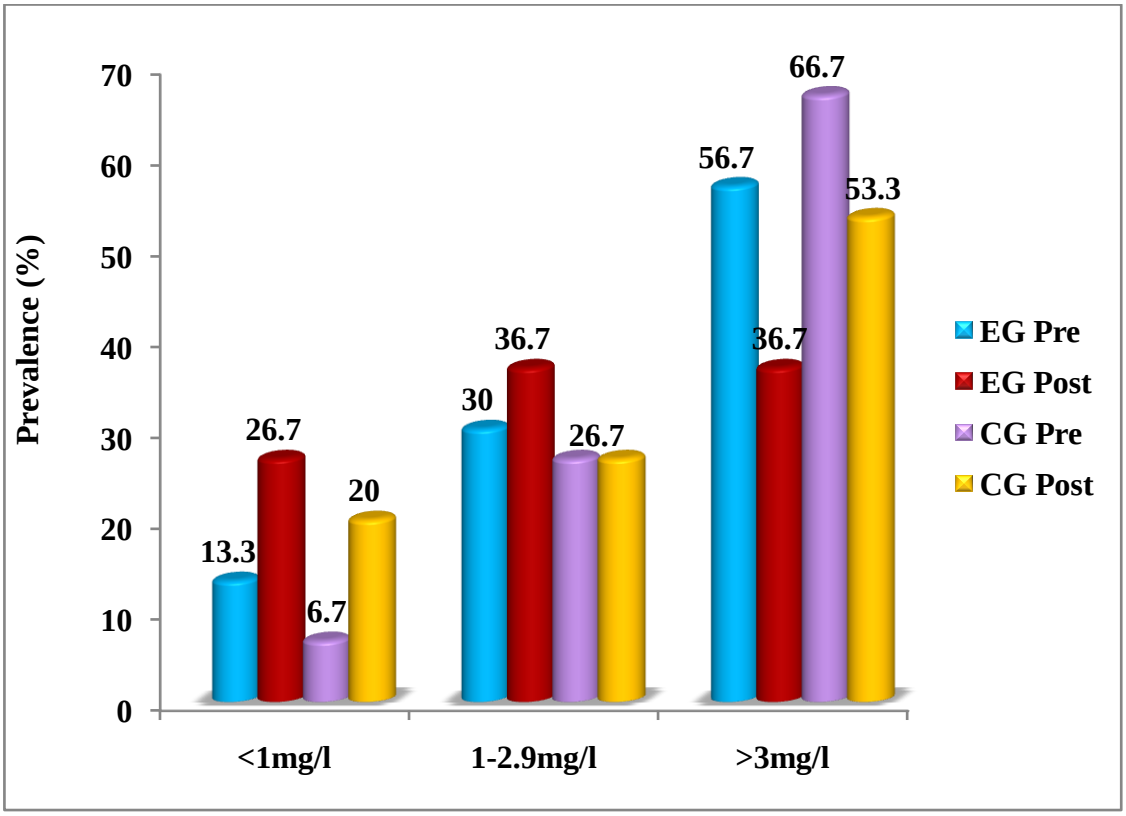


TABLE 4.88: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON Hs-CRP OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (MEAN ± SD)

Variable	Stage	Control Group (N=30)	Experimental Group (N=30)	P value
Hs-CRP (mg/l)	Pre	5.5 ± 3.6	4.6 ± 3.3	0.3
	Post	4.5 ± 4	3.4 ± 3.3	0.24
	Paired t	0.13 (↓18.1%)	**0.024* (↓26.08%)**	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis indicate percentage

FIG 4.47: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON Hs-CRP PROFILE OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (%)



Impact on Glycemic Profile

At baseline, the HbA1c of the subjects in both arms was similar. At termination, though the HbA1c in the experimental arm (8.1 to 7.6%) declined more than the controls (8.06 to 7.8%) within the group, yet it was not significant between the groups (table 4.89).

An excellent glycemic control (HbA1c <6%) was similarly prevalent in both the arms (3.3%) at baseline. At the end of the study, the figure remained unaltered for the controls but there was a rise by 10% in the experimental arm. The prevalence of good glycemia was similar at baseline (23.3%) for both the arms. However, at the termination, both the arms had a similar increase in prevalence (36.7%). An average glycemic control was prevalent in 33.3% of the intervention subjects and 36.7% controls at baseline. At termination, the prevalence came down to 10% in the experimental arm subjects (P 0.029) and to 20% in the controls. The prevalence of poor glycemic control (HbA1c >8%) remained static at 40% for the experimental arm subjects and increased from 36.7% to 40% for the controls (fig 4.48).

Impact on Hepatic Profile

At baseline, the biomarkers of hepatic status were similar in both the arms. Only total protein was significantly higher in the experimental arm than the controls (7.6g/dl vs. 7.3g/dl, P 0.014) prior to intervention. Though alkaline phosphatase declined in both the arms, only the difference in the experimental arm (90.5 to 81.2 U/L, P 0.005) was significant. GGT (32.4 to 26.3U/L, P 0.0016) declined significantly in the experimental arm. SGOT and albumin remained almost similar and there was no difference between the groups and within the groups at the termination of the study. SGPT declined significantly from 26.05 to 20.7U/L (P 0.03) in the experimental arm and also became significantly lower (P 0.014) than the control arm (27.8U/L) (table 4.90). The prevalence of elevated SGPT came down from 13.3% to 3.3%, that of SGOT from 10% to 6.7% and of GGT>35U/L from 33.3% to 23.3% in the experimental arm. In the control arm, the prevalence of elevated SGPT reduced from 16.7% to 13.3%, that of elevated GGT from 23.3% to 20% and the prevalence of elevated SGOT increased from 6.7% to 10%.

TABLE 4.89: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON GLYCEMIC PROFILE OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (MEAN ± SD)

Variables	Stage	Control Group (N=30)	Experimental Group (N=30)	P value
HbA1c (%)	Pre	8.06 ± 1.6	8.1 ± 1.8	0.89
	Post	7.8 ± 1.6	7.6 ± 1.6	0.59
	Paired t	0.33 (↓3.2%)	0.052 (↓6.2%)	
ABG (mg/dl)	Pre	184.7 ± 48.1	189.02 ± 60.2	0.51
	Post	178.2 ± 47.9	169.9 ± 50.7	0.51
	Paired t	0.35 (↓3.5%)	0.07 (↓10.1%)	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis depict proportion

FIG 4.48: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON GLYCATED HEMOGLOBIN OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (%)

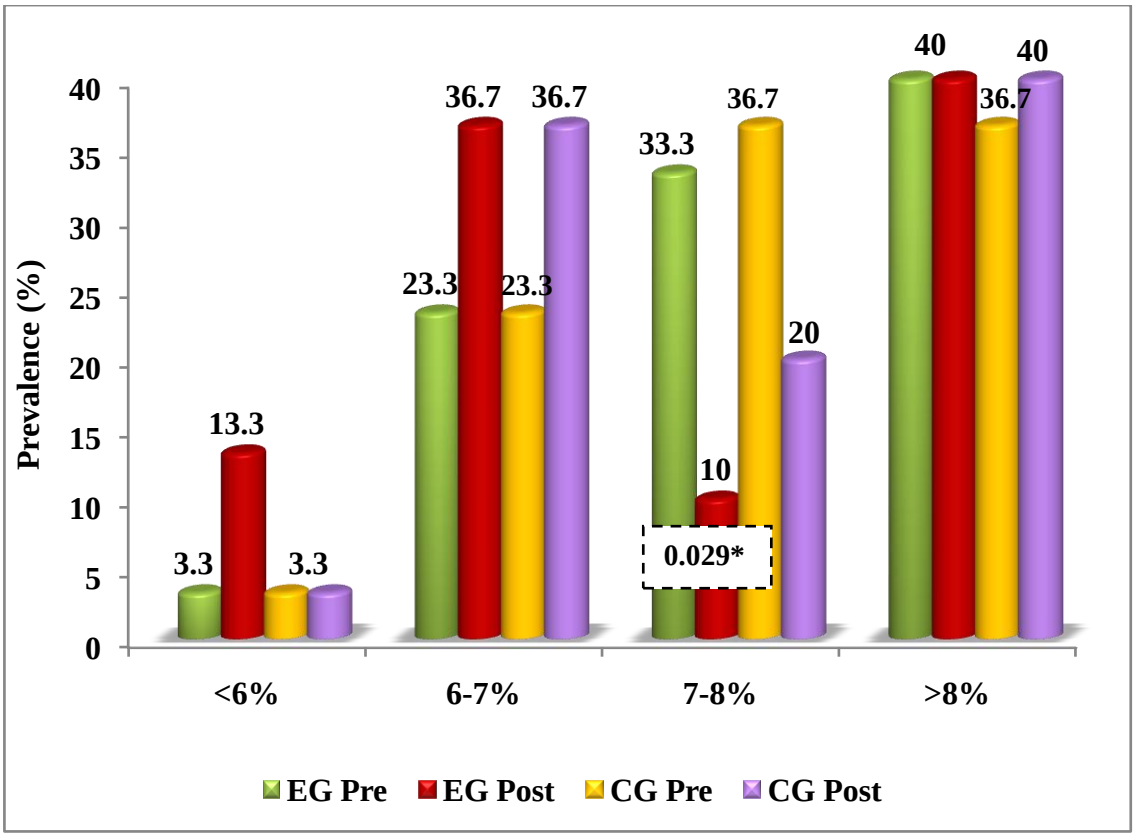


TABLE 4.90: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON HEPATIC PROFILE OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (MEAN ± SD)

Variables	Stage	Control Group (N=30)	Experimental Group (N=30)	P value
Alkaline phosphatase (U/L)	Pre	96.4 ± 19.8	90.5 ± 23.6	0.29
	Post	91.7 ± 20.7	81.2 ± 20.6	0.054
	Paired t	0.1 (↓4.9%)	**0.005** (↓10.3%)**	
Bilirubin direct (mg/dl)	Pre	0.18 ± 0.05	0.2 ± 0.05	0.16
	Post	0.18 ± 0.05	0.2 ± 0.05	0.16
	Paired t	0.58 (0%)	0.59 (0%)	
Bilirubin total (mg/dl)	Pre	0.61 ± 0.19	0.69 ± 0.23	0.12
	Post	0.63 ± 0.27	0.72 ± 0.22	0.17
	Paired t	0.56 (↑3.3%)	0.43 (↑4.3%)	
Bilirubin indirect (mg/dl)	Pre	0.43 ± 0.15	0.49 ± 0.18	0.14
	Post	0.46 ± 0.22	0.51 ± 0.18	0.34
	Paired t	0.4 (↑7%)	0.54 (↑4%)	
GGT (U/L)	Pre	25.3 ± 10.1	32.4 ± 17.3	0.058
	Post	26.01 ± 11.1	26.3 ± 12.7	0.91
	Paired t	0.74 (↑2.8%)	**0.0016** (↓18.8%)**	
SGOT (U/L)	Pre	22.8 ± 10.2	22.6 ± 10.05	0.91
	Post	23.8 ± 10.2	21.4 ± 9.3	0.33
	Paired t	0.41 (↑4.4%)	0.45 (↓5.3%)	
SGPT (U/L)	Pre	25.4 ± 11.1	26.05 ± 15.5	0.87
	Post	27.8 ± 12.1	20.7 ± 9.5	**0.014***
	Paired t	0.23 (↑9.4%)	**0.03* (↓20.5%)**	
Total protein (g/dl)	Pre	7.3 ± 0.32	7.6 ± 0.48	**0.012***
	Post	7.3 ± 0.39	7.5 ± 0.4	0.06
	Paired t	0.93 (0%)	0.68 (↓1.3%)	
Albumin (g/dl)	Pre	4.1 ± 0.31	4.3 ± 0.25	0.054
	Post	4.2 ± 0.25	4.3 ± 0.2	0.57
	Paired t	0.22 (↑2.4%)	0.51 (0%)	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis depict proportion

Impact on Prevalence of Metabolic Syndrome

Since all the enrolled subjects were confirmed cases of type 2 diabetes mellitus, all of them met the criteria of fasting glucose to be counted as a feature of MS. At baseline, more experimental arm subjects had elevated blood pressure than the controls (63.3% vs. 26.7%, P 0.0046). However, with the intervention, the proportion dropped significantly in the experimental arm to 36.7% (P 0.04) and increased in the control arm and the proportion of subjects with BP >130/85mmHg became similar in both the groups. Hypertriglyceridemia or its specific medication was more prevalent at baseline in the experimental arm than the controls. However, at the end of the study, the proportion declined by 13.3% in the experimental arm and increased by 10% in the control arm. The prevalence of low HDL-C remained static in the control arm at the pre and the post stage of the study. But, the prevalence declined by 20% in the experimental arm at the termination of the study. Abdominal obesity was much prevalent in the experimental (93.3%) and the control arm (86.7%) at baseline. After the intervention, the prevalence of abdominal obesity declined to 80% in the experimental arm and the control arm also had a minor drop in the prevalence. MS defined as the presence of abdominal obesity plus two or more of the above mentioned risk factors, was prevalent in 76.7% of the experimental arm subjects and 73.3% of the controls at the beginning of the study. Owing to the intervention, the prevalence declined to 53.3% in the experimental arm and the controls also had a minor drop in the prevalence to 70% (fig 4.49).

Impact on Features of Metabolic Syndrome

Two features of MS were present more in the control arm at baseline. However, at the termination phase, the presence of two features of MS declined in the control arm (20%) and increased in the experimental arm (33.3%). Three features of MS were more prevalent at baseline in the control arm than the intervention arm (50% vs. 23.3%, P 0.03). The figure remained static in experimental arm but the controls had a decline (43.3%) in three features of MS. Four features of MS were more prevalent in the experimental arm (30%) at baseline. After the intervention, the proportion declined in the experimental arm (23.3%) and increased in the control arm (26.7%). Five features of MS were prevalent more in the experimental arm at baseline than the controls (26.7% vs. 6.7%, P 0.03). However, at termination, the experimental arm saw

a decline to 13.3% and the control arm saw a rise to 10% in the presence of five features of MS (table 4.91). The average numbers of features of MS were significantly higher in the experimental arm than the control arm at baseline (3.66 vs. 3.1, P 0.037). However, post intervention, the average no. of features of MS declined significantly in the experimental arm (P 0.0008) from 3.66 to 3.03 and the controls had a non-significant increase in the average number of features of MS from 3.1 to 3.26 (table 4.92).

Impact on Physical Activity Profile

At baseline, the experimental and the control arm reflected a medium physical activity profile. However, after the commencement of the intervention, the experimental arm saw a steady but a non-significant rise in total METminutes/week. The control arm also had a rise first and then a drop in total METminutes/week. But, at the end of the study, the experimental arm (1301.08 total MET minutes/week) had non-significantly higher total MET minutes/week than the control arm (941.7 total METminutes/week) (table 4.93).

Impact on Physical Activity Status

At baseline, subjects had a similar physical activity status in both the groups. There were only 3.3% experimental arm subjects who had a high physical activity profile. After intervention, the figure increased to 6.6% amongst the experimental subjects. With regard to the controls there were none in the high activity category at the inception as well as at the termination of the study (fig 4.50). However, at the end of the study, the prevalence of subjects with medium physical activity increased from 63.3% to 70% in controls and from 56.7% to 63.3% in the experimental arm. The proportion of those with a low profile started dropping at the inception of the intervention and although the controls also saw a decline initially, the figures again took a rise at the point of termination of the study. The prevalence of low physical activity declined from 36.7% to 30% in the control arm and reduced from 40% to 30% in the experimental arm.

FIG 4.49: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON PREVALENCE OF METABOLIC SYNDROME AMONG TYPE 2 DIABETES SUBJECTS WITH NAFLD (%)

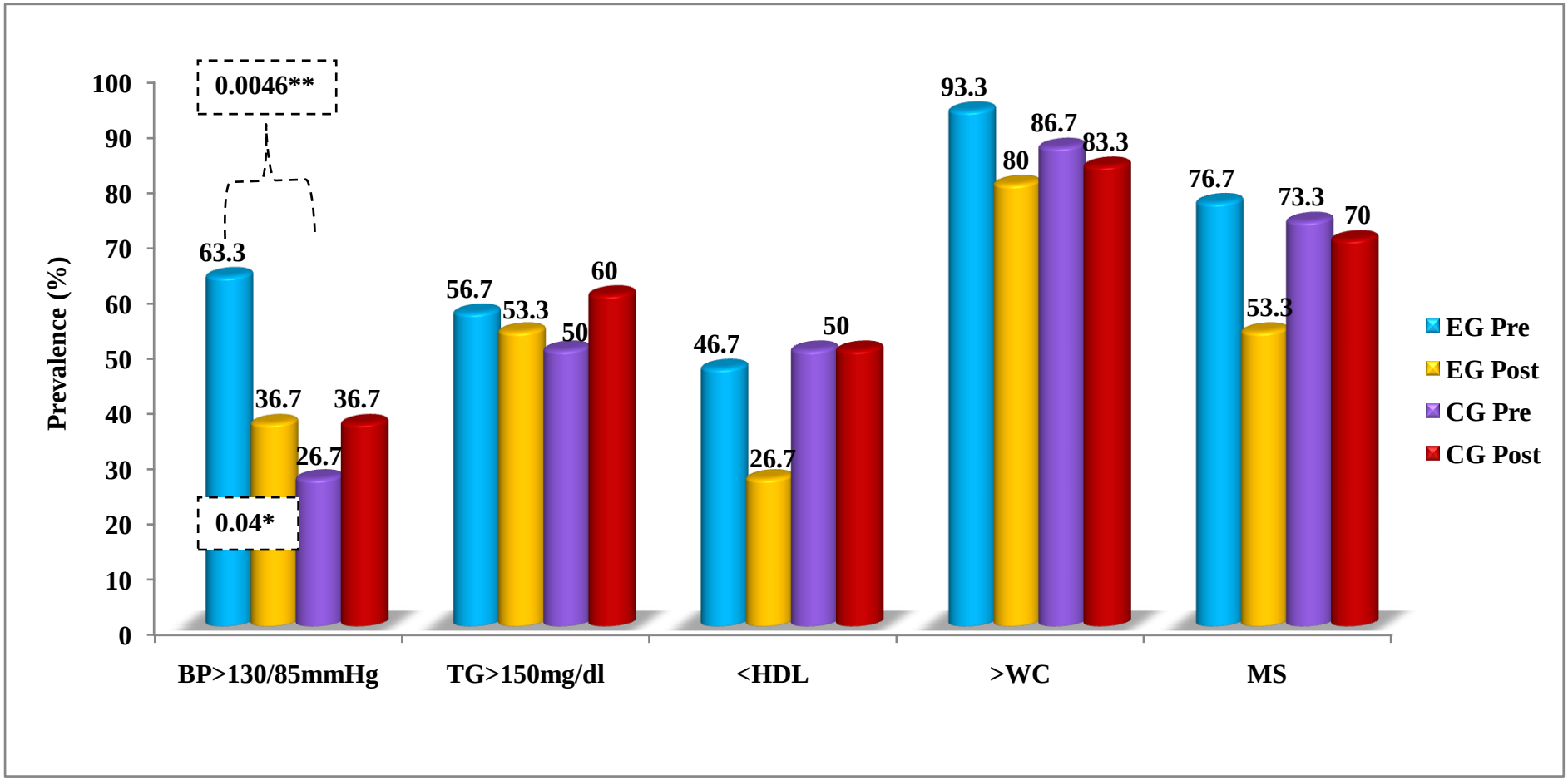


TABLE 4.91: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON NUMBER OF FEATURES OF METABOLIC SYNDROME AMONG TYPE 2 DIABETES SUBJECTS WITH NAFLD (N, %)

Variables	Stage	Control Group (N=30)	Experimental Group (N=30)	χ^2
One	Pre	0 (0)	1 (3.3)	1
	Post	0 (0)	2 (6.7)	0.49
	χ^2	-	1	
Two	Pre	7 (23.3)	5 (16.7)	0.52
	Post	6 (20)	10 (33.3)	0.24
	χ^2	0.75	0.13	
Three	Pre	15 (50)	7 (23.3)	**0.03***
	Post	13 (43.3)	7 (23.3)	0.10
	χ^2	0.60	1	
Four	Pre	6 (20)	9 (30)	0.37
	Post	8 (26.7)	7 (23.3)	0.76
	χ^2	0.54	0.56	
Five	Pre	2 (6.7)	8 (26.7)	**0.03***
	Post	3 (10)	4 (13.3)	1
	χ^2	1	0.20	

Values in parenthesis indicate percentage

TABLE 4.92: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON NUMBER OF FEATURES OF METABOLIC SYNDROME AMONG TYPE 2 DIABETES SUBJECTS WITH NAFLD (MEAN ± SD)

Variables	Stage	Control Group (N=30)	Experimental Group (N=30)	P value
Features of MS	Pre	3.1 ± 0.84	3.66 ± 1.18	**0.037***
	Post	3.26 ± 0.9	3.03 ± 1.18	0.39
	Paired t	0.2 (↑5.2%)	**0.0008*** (↓17.2%)**	

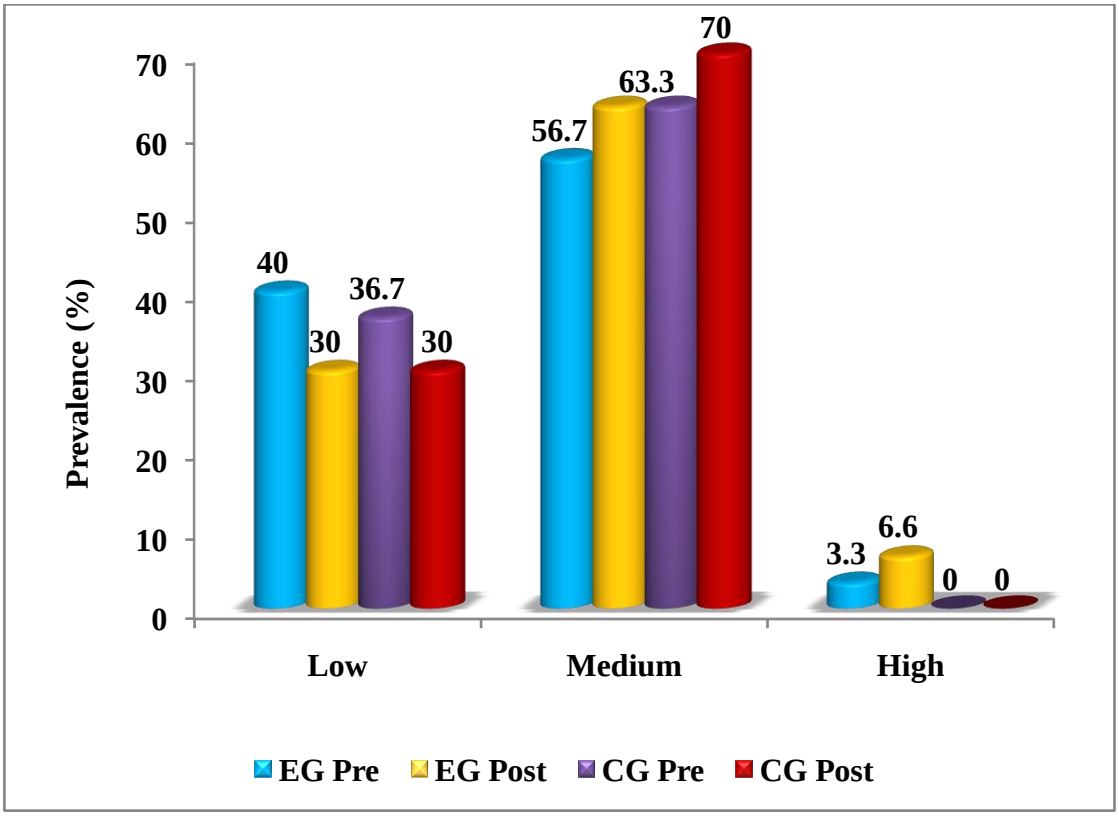
P<0.05*, P<0.01**, P<0.001***, values in parenthesis depict proportion

TABLE 4.93: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON PHYSICAL ACTIVITY PROFILE OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (MEAN ± SD)

Variables	Stage	Control Group (N=30)	Experimental Group (N=30)	P value
Total METminutes /week	0	800.6 ± 550	914.05 ± 820.5	0.53
	1	995.1 ± 549.4	1169.6 ± 892.8	0.36
	2	991.2 ± 549.5	1192.4 ± 895.9	0.29
	3	957.1 ± 578.1	1196.3 ± 892.1	0.22
	4	941.7 ± 583.6	1301.08 ± 1086	0.11
	F value	0.66 (↑17.6%)	0.57 (↑42.3%)	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis depict proportion

FIG 4.50: PHYSICAL ACTIVITY STATUS OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (%)



Impact on Liver Span

The liver span of the subjects in the experimental and the control arm was similar at baseline. After the intervention, a reduction in the liver span was observed in both the arms, but the decline was significant (P 0.037) only for the experimental arm subjects (173.5 to 166.4mm). However, the difference in the liver span of both the arms was not significant at the termination of the study (table 4.94).

At baseline, 66.7% of the experimental arm subjects and 60% of the controls had liver span above 160 mm. After intervention, the prevalence declined from 66.7% to 60% in the experimental arm and from 60% to 44.6% in controls (fig 4.51).

Impact on Liver Status

The prevalence of NAFLD declined significantly in the experimental arm from 100% to 63.3% (P 0.0002) and became significantly lower from controls at the end of the study (63.3% vs. 96.7%, P 0.0013) (fig 4.52). The severity of steatosis also went down significantly with the intervention by 35.4% from 1.86 to 1.2 (P 0.00016) and remained unaltered in controls because of which the former had a significantly lower grade of hepatic steatosis than the latter (1.2 vs. 1.93, P 0.0003) (table 4.95).

At baseline, 23.3% of the NAFLD subjects in the experimental arm had a minimal fatty liver and it dropped to 6.7% after the intervention. Among controls, 10% had a minimal fatty liver at baseline and it went down to nil. More of control arm subjects than the experimental arm subjects (86.7% vs. 66.7%) had a fatty liver at baseline. After the intervention, the prevalence of fatty liver went down from 66.7% to 56.7% amongst the experimental group subjects and increased to 96.7% amongst the controls. The prevalence of fatty liver was found to be significantly higher in the control arm than the experimental arm at the end of the study (P 0.0002). At baseline, 10% of the experimental arm subjects and 3.3% of the controls had a gross fatty liver. At the end of the study, the proportion dropped to nil in both the arms (table 4.96 and fig 4.53).

Impact on Liver Status Shifts

A shift from minimal fatty liver to normal liver was observed in 16.7% of the experimental arm subjects (table 4.97). One fifth of those who were having a fatty

liver reversed to a normal liver after the intervention. A meagre 3.3% shifted to minimal fatty liver from a fatty liver. The 10% cases of gross fatty liver shifted to the stage of fatty liver at the termination of the study. Only 3.3% of the minimal fatty liver cases moved down to the stage of fatty liver at the end of the study. Also, 3.3% of the minimal fatty liver cases maintained the same liver status at the termination of the study. About 43.3% of fatty liver subjects in the experimental arm maintained status quo at the end of the study. With regard to the controls, only 3.3% of the fatty liver subjects reversed to a normal liver. Also, 3.3% of the gross fatty liver subjects moved down to the category of fatty liver. Amongst those who had a minimal fatty liver, 10% of them moved onto the next stage of fatty liver. However, 83.3% of the control arm subjects maintained their fatty liver status at the termination point of the study and it was significantly higher than the experimental arm (P 0.0014).

Association of Variables among Subjects with NAFLD in the Intervention Group

The 8.1% weight loss in the experimental arm (table 4.98) was associated with reduction in the inflammatory markers such as hs-CRP (r 0.503, p 0.005), ferritin (r 0.416, p 0.022) and uric acid (r 0.545, p 0.002). The weight loss was also associated with reductions in triglycerides (r 0.407, p 0.026), VLDL-C (r 0.448, p 0.013), TC/HDL (r 0.425, p 0.019), SGPT (r 0.400, p 0.029) and liver span (r 0.495, p 0.005).

The weight loss brought about 7.9% decline in BMI which was associated with reduction in hs-CRP (r 0.698, p 0.000) and uric acid (r 0.471, p 0.009).

WC declined by 5.2% which was associated with reductions in hs-CRP (r 0.453, p 0.012), uric acid (0.626, p 0.000), number of features of MS (r 0.434, p 0.017), liver span (r 0.563, p 0.001) and improvement in physical activity status (r -0.484, p 0.007).

The decline in the WC led to 6.2% reduction in WSR which was associated with reduction in hs-CRP (r 0.580, p 0.001), uric acid (r 0.500, p 0.005), alkaline phosphatase (r 0.366, p 0.046), liver span (r 0.563, p 0.001) and improvement in physical activity status (r -0.472, p 0.008).

Association of Weight Alterations with Liver Status

Majority of the control arm subjects who lost <7% weight maintained their grade 2 NAFLD status and were comparably higher than the prevalence of <7% weight loss grade 2 NAFLD subjects of the intervention arm who maintained status quo (60% vs. 16.7%, P 0.0006). Two sub-sets of 6.7% each of the experimental arm who lost <7% weight, shifted positively from minimal fatty liver to normal liver and from fatty liver to normal liver, respectively vs. none in the control arm. Despite weight loss of ≥7%, a control arm subject shifted negatively from minimal fatty liver to fatty liver vs. none in the experimental arm. A single experimental arm subject shifted from fatty liver to minimal fatty liver with <7% weight loss. About 6.7% of the experimental arm subjects and 3.3% of the control arm subjects shifted one grade down from gross fatty liver to fatty liver with <7% weight loss.

About 10% of the subjects in the experimental arm who lost ≥7% weight shifted from minimal fatty liver to normal liver vs. none in the control arm. Additionally, 13.3% of the subjects of the experimental arm and 3.3% subjects of the control arm who lost ≥7% weight shifted from grade 2 fatty liver to normal liver. Majority of the experimental arm subjects and control arm subjects who lost ≥7% weight maintained their grade 2 fatty liver status (26.7% and 10%, P 0.09). ≥7% weight loss also brought an upward shift in 3.3% subjects of the experimental arm from minimal fatty liver to fatty liver. Another subject shifted downward from gross fatty liver to fatty liver and a single subject maintained minimal fatty liver status in the experimental arm.

Weight gain was only registered in the control arm. Of them, 13.3% subjects who gained <7% weight maintained their grade 2 fatty liver status and another 3.3% shifted negatively from minimal fatty liver to fatty liver. Similarly, 3.3% subjects who gained ≥7% weight, shifted from minimal fatty liver to fatty liver at the end of the study (table 4.99).

TABLE 4.94: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON LIVER SPAN OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (MEAN ± SD)

Variable	Stage	Control Group (N=30)	Experimental Group (N=30)	P value
Liver span (mm)	Pre	166.8 ± 21.9	173.5 ± 21.4	0.23
	Post	163.3 ± 26.3	166.4 ± 20	0.61
	Paired t	0.49 (↓2.09%)	**0.037* (↓4.09%)**	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis indicate percentage

FIG 4.51: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON LIVER SPAN OF TYPE 2 DIABETES SUBJECTS WITH NAFLD (%)

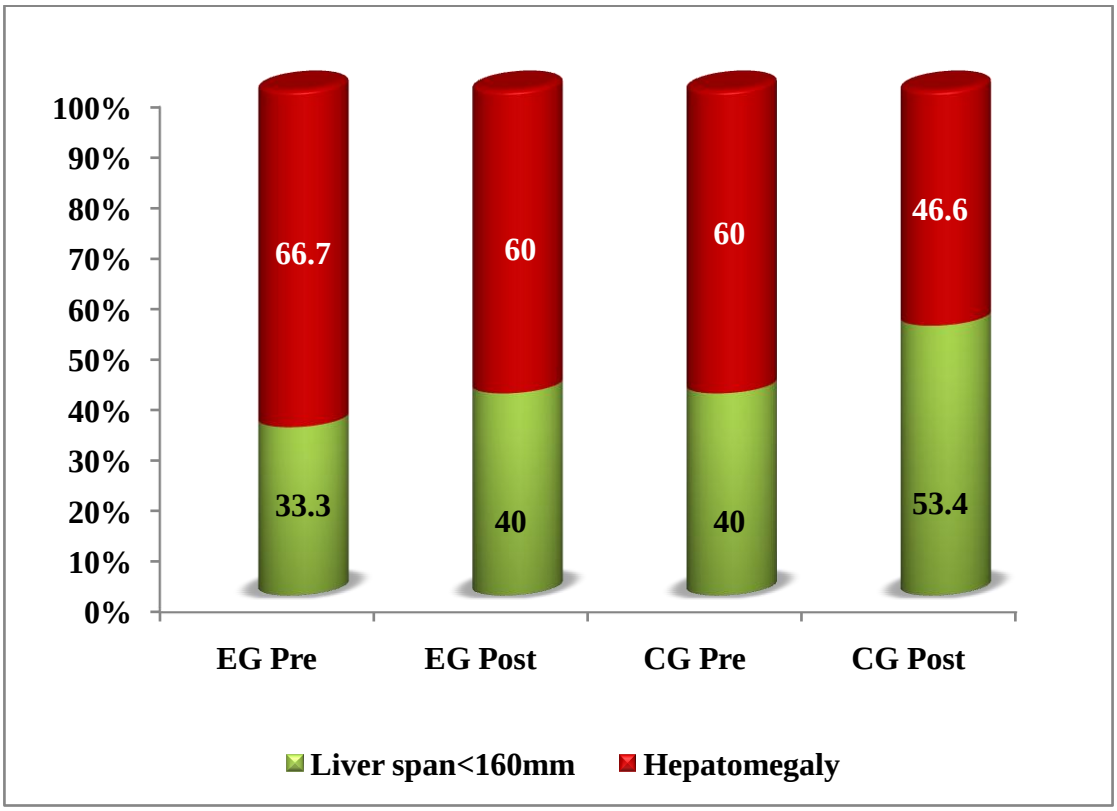


FIG 4.52: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON PREVALENCE OF NAFLD IN TYPE 2 DIABETICS (%)

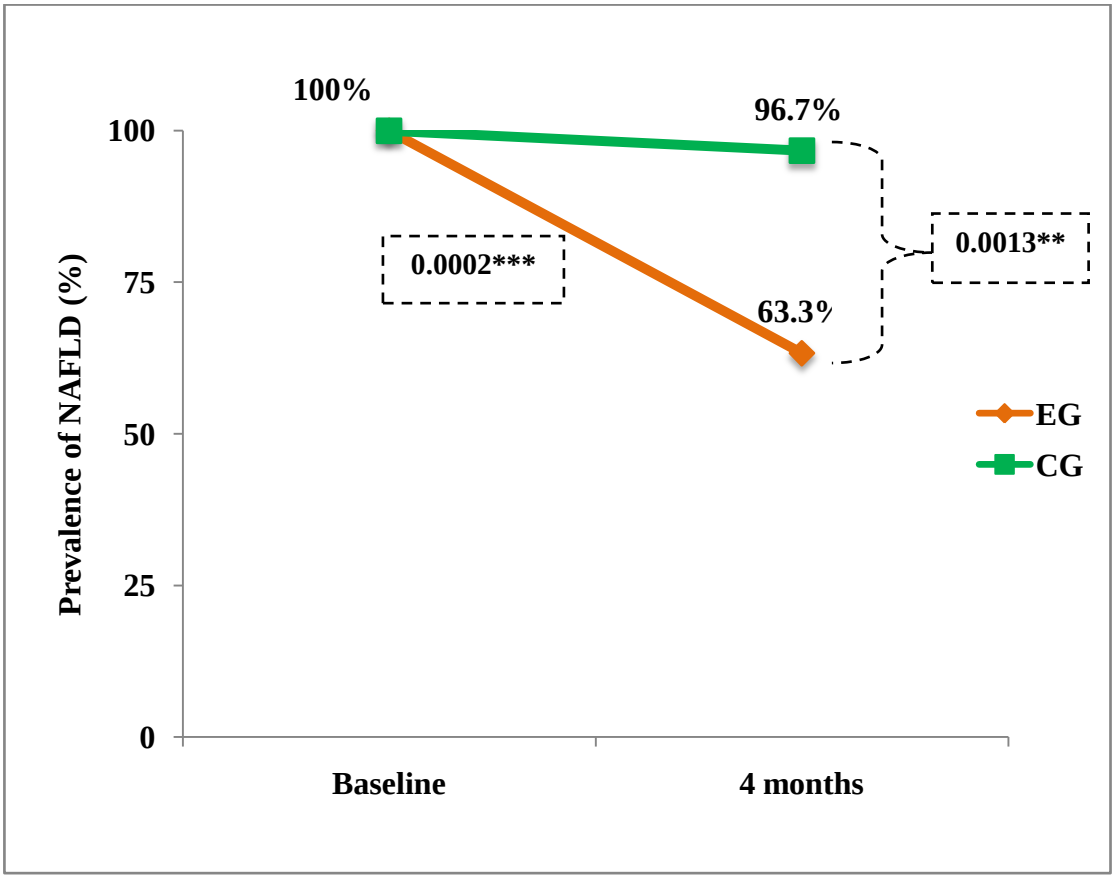


TABLE 4.95: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON SEVERITY OF USG STEATOSIS (MEAN ± SD)

Liver Status	Stage	Control Group (N=30)	Experimental Group (N=30)	P value
Severity of NAFLD (Grade)	Pre	1.93 ± 0.36	1.86 ± 0.57	0.59
	Post	1.93 ± 0.36	1.2 ± 0.96	**0.0003*** **
	Paired t	1 (0%)	**0.00016*** (↓35.4%)**	

TABLE 4.96: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON LIVER STATUS OF THE TYPE 2 DIABETES SUBJECTS WITH NAFLD (N,%)

Liver Status	Stage	Control Group (N=30)	Experimental Group (N=30)	P value
Normal Liver	Pre	0 (0)	0 (0)	-
	Post	1 (3.3)	11 (36.7)	**0.0013****
	χ^2	1	**0.0002*****	
Minimal fatty liver	Pre	3 (10)	7 (23.3)	0.16
	Post	0 (0)	2 (6.7)	0.49
	χ^2	0.078	0.073	
Fatty liver	Pre	26 (86.7)	20 (66.7)	0.069
	Post	29 (96.7)	17 (56.7)	**0.0002*****
	χ^2	0.35	0.42	
Gross fatty liver	Pre	1 (3.3)	3 (10)	.61
	Post	0 (0)	0 (0)	-
	χ^2	1	0.078	

Values in parenthesis indicate percentage, P<0.05*, P<0.01**, P<0.001***

FIG 4.53: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON LIVER STATUS OF THE TYPE 2 DIABETES SUBJECTS WITH NAFLD (%)

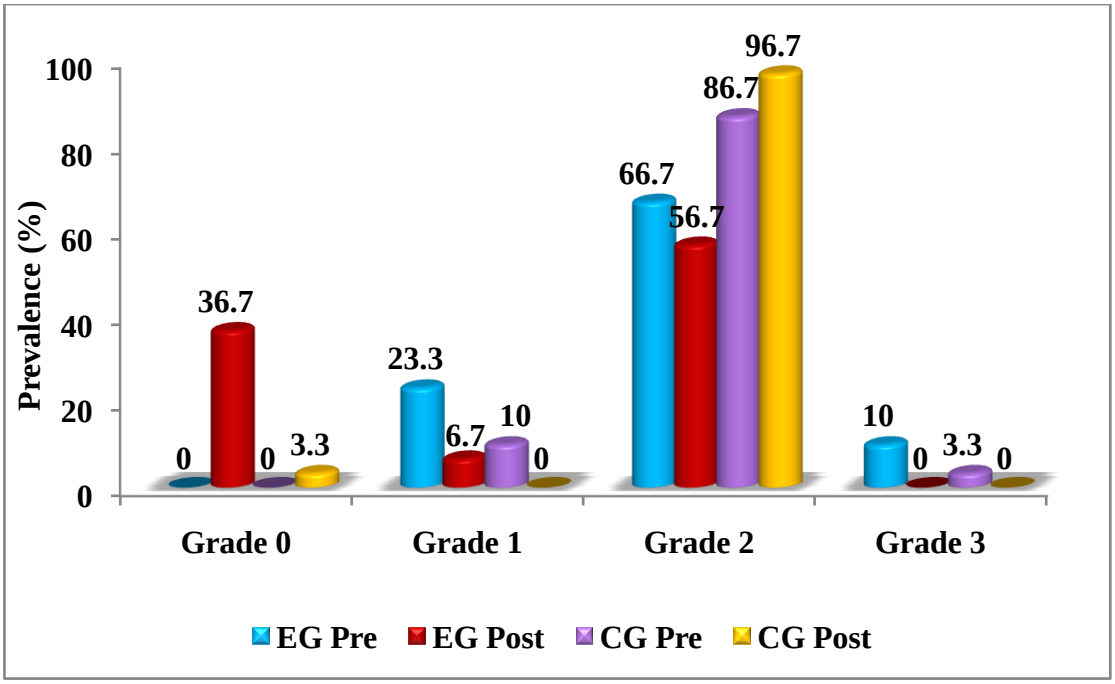


TABLE 4.97: IMPACT OF LIFESTYLE MODIFICATION THERAPY ON SHIFTS IN LIVER STATUS AMONG TYPE 2 DIABETES SUBJECTS WITH NAFLD (N, %)

Liver Status		**Shift**	**Control Group (N=30)**	**Experimental Group (N=30)**	χ^2
Pre	**Post**				
Minimal fatty liver	Normal liver	**Positive**	0 (0)	5 (16.7)	0.052
Fatty liver	Normal liver	**Positive**	1 (3.3)	6 (20)	0.10
Minimal fatty liver	Fatty liver	**Negative**	3 (10)	1 (3.3)	0.61
Fatty Liver	Minimal fatty liver	**Positive**	0 (0)	1 (3.3)	1
Gross fatty liver	Fatty Liver	**Positive**	1 (3.3)	3 (10)	0.61
Minimal fatty liver	Minimal fatty liver	**Status Quo**	0 (0)	1 (3.3)	1
Fatty liver	Fatty liver	**Status Quo**	25 (83.3)	13 (43.3)	**0.0014****

Values in parenthesis indicate percentage, p<0.05*, p<0.01**, p<0.001***

TABLE 4.98: CORRELATION OF VARIABLES IN THE EXPERIMENTAL ARM (N=30)

Variables	Weight	BMI	WC	WSR	SBP	DBP	Hs-CRP	GGT	SGPT	TG	HDL	HbA1c	Liver span
Hs-CRP	0.503	0.698	0.453	0.580	0.196	0.192	-	0.008	0.279	0.242	-0.076	0.201	0.174
	0.005**	**0.000*****	**0.012***	**0.001*****	0.299	0.310	-	0.968	0.136	0.198	0.691	0.287	0.356
Uric acid	0.545	0.471	0.626	0.500	0.146	0.262	0.270	-0.064	0.021	0.155	-0.087	0.220	0.628
	0.002**	**0.009****	**0.000*****	**0.005****	0.440	0.163	0.150	0.739	0.914	0.413	0.649	0.244	**0.000*****
TC	.260	0.173	0.172	0.071	-0.200	-0.207	0.218	0.006	0.089	0.331	0.191	-0.043	0.164
	.165	0.359	0.363	0.710	0.290	0.272	0.248	0.974	0.640	0.074	0.313	0.821	0.386
HDL	-.312	-0.206	-0.213	-0.115	-0.317	-0.121	-0.076	-0.173	-0.172	-0.429	-	-0.568	-0.327
	.093	0.274	0.258	0.544	0.088	0.523	0.691	0.359	0.363	**0.018***	-	**0.001****	0.078
LDL	-.024	-0.030	-0.168	-0.157	-0.289	-0.338	0.005	0.059	0.066	0.344	0.091	-0.135	-0.046
	0.901	0.875	0.375	0.409	0.122	0.067	0.981	0.758	0.728	0.063	0.632	0.478	0.809

Karl Pearson's coefficient of correlation significant at *r value*<0.05*, <0.01**, <0.001***

TABLE 4.98: CORRELATION OF VARIABLES IN THE EXPERIMENTAL ARM (N=30)

Variables	Weight	BMI	WC	WSR	SBP	DBP	Hs-CRP	GGT	SGPT	TG	HDL	HbA1c	Liver span
TG	0.407	0.310	0.230	0.137	0.162	0.119	0.242	0.425	0.210	-	-0.429	0.282	0.368
	0.026*	0.095	0.222	0.469	0.392	0.531	0.198	**0.019***	0.264	-	**0.018***	0.131	**0.046***
No. of features of MS	0.269	0.214	0.434	0.354	0.656	0.566	0.207	0.190	0.147	0.424	-0.500	0.406	0.084
	0.150	0.255	**0.017***	0.055	**0.000*****	**0.001****	0.271	0.315	0.440	**0.019***	**0.005****	**0.026***	0.658
VLDL	0.448	0.286	0.231	0.078	0.159	0.138	0.268	0.304	0.232	0.966	-0.436	0.408	0.411
	0.013*	0.126	0.219	0.681	0.401	0.467	0.152	0.103	0.218	**0.000*****	**0.016***	**0.025***	**0.024***
AP	0.212	0.356	0.268	0.366	0.157	0.345	0.497	0.133	0.277	0.385	0.038	0.322	0.067
	0.260	0.053	0.152	**0.046***	0.408	0.062	**0.005****	0.485	0.138	**0.036***	0.843	0.082	0.726
GGT	0.205	0.172	0.247	0.197	-0.090	0.012	0.008	-	0.541	0.425	-0.173	-0.085	0.042
	0.278	0.364	0.188	0.298	0.638	0.949	0.968	-	**0.002****	**0.019***	0.359	0.656	0.826

Karl Pearson's coefficient of correlation significant at *r value*<0.05*, <0.01**, <0.001***

TABLE 4.98: CORRELATION OF VARIABLES IN THE EXPERIMENTAL ARM (N=30)

Variables	Weight	BMI	WC	WSR	SBP	DBP	Hs-CRP	Ferritin	GGT	SGPT	TG	HDL	HbA1c	Liver span
SGPT	0.400	0.350	0.286	0.206	-0.024	0.231	0.279	0.428	0.541	-	0.210	-0.172	0.066	-0.028
	0.029*	0.058	0.126	0.275	0.900	0.219	0.136	**0.018***	**0.002****	-	0.264	0.363	0.730	0.881
HbA1c	0.148	-0.012	0.140	-0.020	0.256	0.329	0.201	-0.22	-0.085	0.066	0.282	-0.568	-	0.324
	0.434	0.950	0.459	-0.915	0.172	0.076	0.287	0.232	0.656	0.730	0.131	**0.001****	-	0.080
Liver span	0.495	0.288	0.563	0.326	0.099	0.118	0.174	0.035	0.042	-0.028	0.368	-0.327	0.324	-
	0.005**	0.123	**0.001****	0.079	0.604	0.534	0.356	0.854	0.826	0.881	**0.046***	0.078	0.080	-
PA Status	-0.286	-0.336	-0.484	-0.472	-0.309	-0.354	-0.446	-0.25	-0.162	-0.132	-0.081	0.035	-0.211	0.069
	0.125	0.070	**0.007****	**0.008****	0.096	0.055	**0.013***	0.175	0.392	0.487	0.669	0.856	0.264	0.719

Karl Pearson's coefficient of correlation significant at *r value*<0.05*, <0.01**, <0.001***

TABLE 4.99: ASSOCIATION OF WEIGHT ALTERATIONS WITH HEPATIC STATUS AMONG TYPE 2 DIABETES SUBJECTS WITH NAFLD (N, %)

Weight Status	Liver Status Pre	Liver Status Post	Control Group (N=30)	Experimental Group (N=30)	χ^2
Weight Loss <7%	Minimal fatty liver	Normal liver	0 (0)	2 (6.7)	0.49
	Fatty liver	Normal liver	0 (0)	2 (6.7)	0.49
	Minimal fatty liver	Fatty liver	1 (3.3)	0 (0)	1
	Fatty Liver	Minimal fatty liver	0 (0)	1 (3.3)	1
	Gross fatty liver	Fatty Liver	1 (3.3)	2 (6.7)	1
	Fatty liver	Fatty liver	18 (60)	5 (16.7)	**0.0006*** **
Weight Loss ≥7%	Minimal fatty liver	Normal liver	0 (0)	3 (10)	0.078
	Fatty liver	Normal liver	1 (3.3)	4 (13.3)	0.35
	Minimal fatty liver	Fatty liver	0 (0)	1 (3.3)	1
	Gross fatty liver	Fatty Liver	0 (0)	1 (3.3)	1
	Minimal fatty liver	Minimal fatty liver	0 (0)	1 (3.3)	1
	Fatty liver	Fatty liver	3 (10)	8 (26.7)	0.09
Weight Gain <7%	Minimal fatty liver	Fatty liver	1 (3.3)	0 (0)	1
	Fatty liver	Fatty liver	4 (13.3)	0 (0)	0.11
Weight Gain ≥7%	Minimal fatty liver	Fatty liver	1 (3.3)	0 (0)	1

Values in parenthesis indicate percentage, P<0.05*, P<0.01**, P<0.001***

IMPACT OF ≥7% WEIGHT LOSS ON THE ANTHROPOMETRIC, BIO-PHYSICAL, BIOCHEMICAL, DIETARY, METABOLIC SYNDROME AND LIVER STATUS OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY

General profile

The age and the duration of diabetes did not differ significantly between subjects who lost < or ≥7% weight (table 4.100). Majority of the subjects who lost ≥7% weight were in the 50-60 years age bracket. All the subjects who lost ≥7% weight were on OHAs unlike the subjects who lost <7% wherein three fourths were on OHAs and the remaining were on OHA plus insulin (table 4.101).

Impact on Anthropometric and Blood Pressure Profile

At baseline, all the anthropometric indices and blood pressure was similar for the subjects who lost < and ≥7% weight. The intervention resulted in significant decline within the arms, but the changes were more prominent in the subjects who lost ≥7% weight; BMI (9.9% vs. 4.8%), WC (6.4% vs. 3.4%), WSR (6.3% vs. 3.2%) and SBP (12.7% vs. 10.5%), respectively. However, the changes did not differ significantly between the two groups post intervention (table 4.102).

Impact on Nutritional Status

There was a marginal increase in prevalence of normal BMI (16.7% to 22.2%) in subjects who lost ≥7% weight. Prevalence of obesity remained unaltered at 58.3% in subjects who lost <7% weight and declined (77.7% to 61.1%) in subjects who lost ≥7% weight and consequently the prevalence of overweight increased (5.5% to 16.7%). A change in the prevalence of overweight occurred from 25% to 8.3% as those with normal BMI increased to 33.3% from 16.7% in subjects who lost <7% weight. Elevated WC came down to 75% from 91.6% in subjects who lost <7% weight and reduced from 94.4% to 83.3% in subjects who lost ≥7% weight. Post intervention, the prevalence of elevated WSR came down from 88.8% to 66.7% in subjects with ≥7% weight loss. Those who lost <7% weight had a marginal decline to 91.6% (fig 4.54).

TABLE 4.100: AGE AND DURATION OF DIABETES OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (MEAN ± SD)

Variable	Weight Loss		P value
	<7% (N=12)	≥7% (N=18)	
Age (years)	56.6 ± 11.7	55.6 ± 6.4	0.77
30-40 years	0 (0)	1 (5.6)	1
40-50 years	4 (33.3)	1 (5.6)	0.12
50-60 years	3 (25)	10 (55.6)	0.10
60-70 years	3 (25)	6 (33.3)	0.70
>70 years	2 (16.7)	0 (0)	0.15
DOD (years)	8.6 ± 6.3	5.8 ± 6.1	0.24

P<0.05*, P<0.01**, P<0.001***

TABLE 4.101: DRUG PROFILE OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (N, %)

Drugs	Weight Loss		P value
	<7% (N=12)	≥7% (N=18)	
OHA	9 (75)	18 (100)	0.054
OHA + Insulin	3 (25)	0 (0)	0.054
Dyslipidemic agents	6 (50)	9 (50)	1
Anti-anginal agents	1 (8.33)	2 (11.1)	1
Anti-platelet agents	3 (25)	3 (16.7)	0.65
ACE inhibitor agents	0 (0)	1 (5.6)	1
Thyroid hormones	4 (33.3)	4 (22.2)	0.67
Angiotensin II antagonist agents	3 (25)	5 (27.7)	1
Beta blocker agents	6 (50)	6 (33.3)	0.45
NSAID agents	2 (16.7)	1 (5.6)	0.54
Anti-gout agents	0 (0)	1 (5.6)	1
Diuretic agents	1 (8.33)	2 (11.1)	1

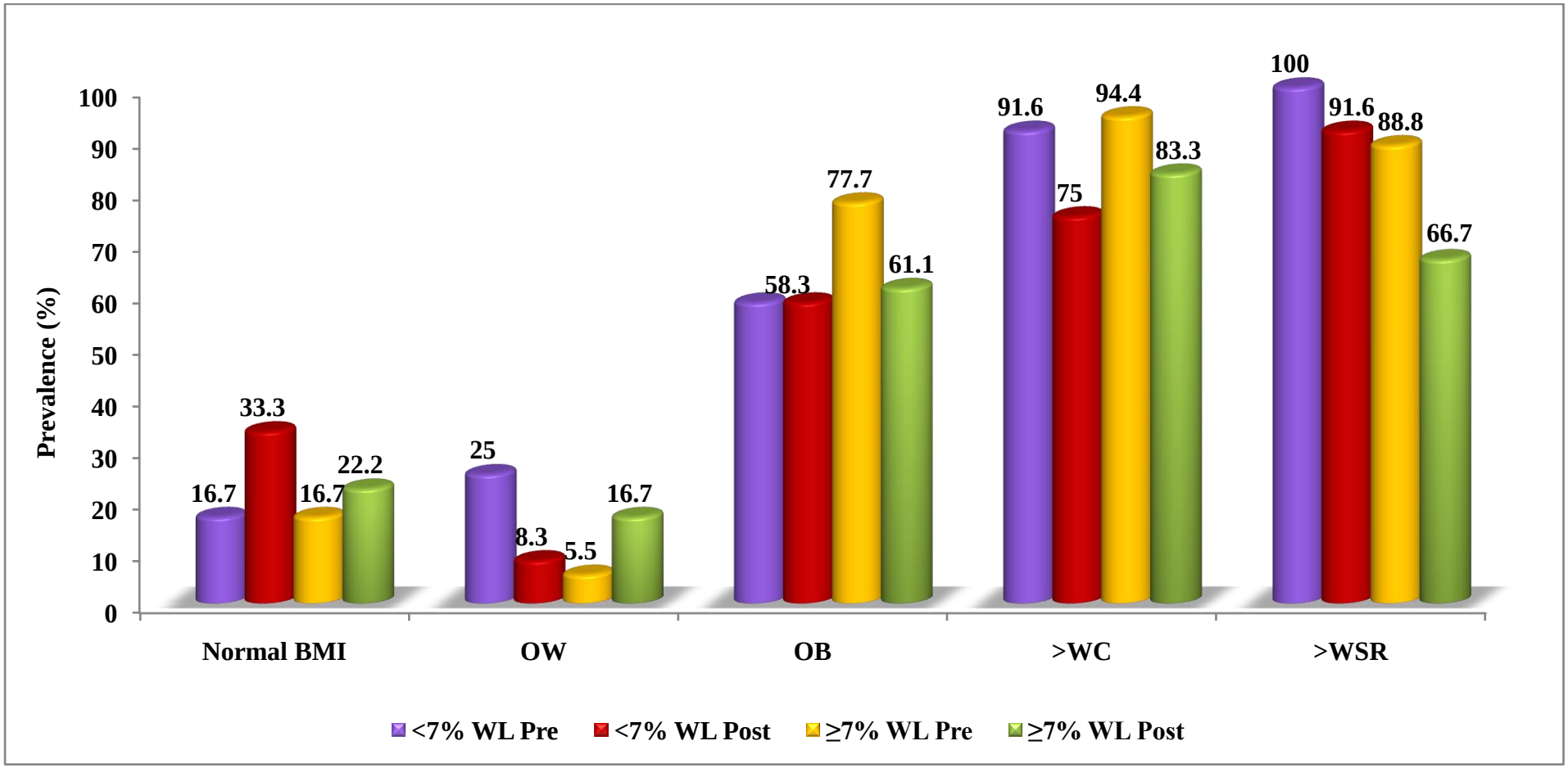
Values in parenthesis indicate percentage

TABLE 4.102: IMPACT ON THE ANTHROPOMETRIC AND BLOOD PRESSURE PROFILE OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (MEAN ± SD)

Variable	Stage	Weight Loss		P value
		<7% (N=12)	**≥7% (N=18)**	
BMI (kg/m^2)	Pre	28.7 ± 7.3	28.9 ± 4.3	0.93
	Post	27.3 ± 7.1	26.03 ± 3.9	0.56
	Paired t	**1.59E*** (↓4.8%)**	**2.63E*** (↓9.9%)**	
WC (cm)	Pre	100.5 ± 10.9	101.6 ± 7.8	0.75
	Post	97.1 ± 10.9	95.1 ± 7.7	0.56
	Paired t	**1.39E*** (↓3.4%)**	**1.02E*** (↓6.4%)**	
WSR	Pre	0.63 ± 0.07	0.64 ± 0.06	0.71
	Post	0.61 ± 0.07	0.60 ± 0.06	0.70
	Paired t	**2.02E*** (↓3.2%)**	**1.14E*** (↓6.3%)**	
SBP (mmHg)	Pre	144.08 ± 18.2	146.05 ± 16.6	0.75
	Post	129 ± 6.6	127.5 ± 5.6	0.51
	Paired t	**0.004** (↓10.5%)**	**3.76E*** (↓12.7%)**	
DBP (mmHg)	Pre	86.9 ± 10.9	88.1 ± 7.8	0.73
	Post	85.7 ± 4.9	85.3 ± 6.3	0.84
	Paired t	0.72 (↓1.4%)	0.11 (↓3.2%)	

P<0.05*, P<0.01**, P<0.001***

FIG 4.54: IMPACT ON THE NUTRITIONAL STATUS OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (%)



Dietary Choices and Impact on Nutrient Intake

About 88.9% of the subjects who lost ≥7% weight were vegetarians along with three fourths of those who lost <7% weight. Subjects who lost ≥7% weight had a non-significant but more profound decline in carbohydrate and fat intake than the subjects who lost <7% and had a non-significant increase in protein, crude fibre, iron, vitamin A, vitamin C, total dietary fibre and insoluble fibre intake which was of greater intensity than the subjects who lost <7% weight (table 4.103). But, subjects with ≥7% weight loss had a significant increase in soluble fibre intake (P 0.017). The soluble fibre intake in the 3rd (4.1g vs. 3.3g, P 0.039) and the 4th month (4.3g vs. 3.3g, P 0.031) was significantly different from baseline in subjects with ≥7% weight loss and soluble fibre intake in the 3rd month (4.1g vs. 3.1g, P 0.011) and the 4th month (4.3g vs. 3.1g, P 0.011) was also significantly higher from the intake in the 2nd month in subjects with ≥7% weight loss (table 4.104).

In subjects who had ≥7% weight loss, their proportion of protein intake increased significantly (P 0.034) (table 4.105). It was significantly higher at the 3rd month (12.4% vs. 11.04%, P 0.040) and 4th month (13% vs. 11.04%, P 0.0017) compared to baseline. The proportion of CHO intake declined similarly in subjects with ≥7% weight loss and with <7% weight loss, whereas that of fat declined more non-significantly in subjects with ≥7% weight loss (table 4.106).

Impact on Frequency of Eating Out

Weekly eating out declined non-significantly from 27.7% to 11.1% in subjects who lost ≥7% weight (fig 4.55) and no alterations were seen in subjects who lost <7% weight (25%). Fortnightly eating out declined marginally in subjects who lost <7% weight and increased non-significantly from 27.7% to 50% in subjects who lost ≥7% weight. Eating out on a monthly basis, the prevalence was significantly higher in subjects who lost ≥7% weight compared to subjects who lost <7% weight (55.5% vs. 41.6%, P 0.025). About 27.7% at baseline and 33.3% post intervention subjects with ≥7% weight loss reported eating out only rarely. The prevalence of rarely eating out was only predominant in subjects who lost ≥7% weight and hence was significantly higher than the subjects who lost <7% weight, post intervention (33.3% vs. 0%, P 0.019).

TABLE 4.103: IMPACT ON NUTRIENT INTAKE OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (MEAN ± SD)

Variable	Timeline (Months)	Weight Loss		P value
		<7% (N=12)	≥7% (N=18)	
Energy (kcal)	0	1540 ± 182	1471 ± 206	0.34
	1	1396 ± 197	1457 ± 131	0.35
	2	1383 ± 130	1397 ± 113	0.76
	3	1458 ± 119	1402 ± 80	0.17
	4	1443 ± 139	1432 ± 92	0.81
	F value	0.12	0.36	
Carbohydrates (g)	0	212.6 ± 38.5	196 ± 39.5	0.26
	1	206.2 ± 48.9	202.2 ± 34.1	0.80
	2	199.2 ± 39.7	188.9 ± 31.7	0.46
	3	201.8 ± 27.5	189.6 ± 32.7	0.28
	4	203 ± 35.6	187.6 ± 34.5	0.24
	F value	0.92	0.68	
Fat (g)	0	50.4 ± 8.7	53.5 ± 9.4	0.37
	1	43.1 ± 9	47.6 ± 9.6	0.21
	2	43.8 ± 12.5	47.6 ± 10.3	0.35
	3	45.2 ± 11.7	47.5 ± 11.9	0.61
	4	46.7 ± 8.9	50.1 ± 12.1	0.38
	F value	0.44	0.37	
Protein (g)	0	44.4 ± 6.3	39.9 ± 8.3	0.12
	1	43.2 ± 9.1	44.1 ± 8.6	0.79
	2	42.9 ± 9	41.7 ± 8.1	0.70
	3	46.7 ± 6.8	43.6 ± 9.6	0.32
	4	46 ± 6.6	46.6 ± 8.1	0.81
	F value	0.70	0.18	

P<0.05*, P<0.01**, P<0.001***

TABLE 4.103: IMPACT ON NUTRIENT INTAKE OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (MEAN ± SD)

Variable	Timeline (Months)	Weight Loss		P value
		<7% (N=12)	≥7% (N=18)	
Crude Fibre (g)	0	5.6 ± 2.1	5.9 ± 1.9	0.60
	1	5.8 ± 2.5	6.3 ± 1.8	0.58
	2	5.4 ± 1.9	5.2 ± 1.5	0.71
	3	5.9 ± 1.3	6.4 ± 2.2	0.45
	4	6.1 ± 1.8	6.5 ± 2.1	0.54
	F value	0.93	0.21	
Iron (mg)	0	13.9 ± 4.5	11.2 ± 3.7	0.08
	1	12.1 ± 3.4	12.8 ± 2.9	0.54
	2	12.3 ± 6.1	12.2 ± 3.9	0.94
	3	12.9 ± 3.2	13.4 ± 4.3	0.74
	4	13.8 ± 3.6	12.7 ± 4.2	0.46
	F value	0.77	0.48	
Vitamin C (mg)	0	55.1 ± 48.7	78.4 ± 69.6	0.28
	1	49.6 ± 36.7	75.4 ± 67.4	0.18
	2	48.4 ± 42.1	76.2 ± 65.1	0.16
	3	53.3 ± 49.6	95.8 ± 82.3	0.08
	4	60.5 ± 64.4	79.1 ± 70.5	0.46
	F value	0.97	0.90	
Total dietary fibre (g)	0	11.8 ± 5.07	14.4 ± 4.4	0.16
	1	13.1 ± 5.7	15.2 ± 5.3	0.31
	2	12.2 ± 3.8	13.6 ± 3.8	0.35
	3	12.6 ± 7.4	16.6 ± 4.2	0.11
	4	12.7 ± 7.6	17.6 ± 6.7	0.08
	F value	0.99	0.12	

P<0.05*, P<0.01**, P<0.001***

TABLE 4.103: IMPACT ON NUTRIENT INTAKE OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (MEAN ± SD)

Variable	Timeline (Months)	Weight Loss		P value
		<7% (N=12)	≥7% (N=18)	
Insoluble dietary fibre (g)	0	9.2 ± 4.1	11.1 ± 3.7	0.18
	1	10.1 ± 4.6	11.6 ± 4.3	0.37
	2	9.5 ± 3.2	10.5 ± 3.2	0.39
	3	9.4 ± 5.5	12.5 ± 3.2	0.09
	4	9.6 ± 5.6	13.3 ± 5.2	0.07
	F value	0.99	0.23	
Soluble dietary fibre (g)	0	2.6 ± 1.1	3.3 ± 0.8	0.10
	1	2.9 ± 1.2	3.5 ± 1.2	0.16
	2	2.7 ± 0.9	3.1 ± 0.8	0.35
	3	3.3 ± 1.8	4.1 ± 1.3	0.20
	4	3.1 ± 2.1	4.3 ± 1.6	0.12
	F value	0.86	**0.017***	

P<0.05*, P<0.01**, P<0.001***

TABLE 4.104: DIFFERENCE IN SOLUBLE DIETARY FIBRE INTAKE IN SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS

Groups	P value soluble dietary fibre
Baseline vs. 1st month	0.45
Baseline vs. 2nd month	0.50
Baseline vs. 3rd month	**0.039***
Baseline vs. 4th month	**0.031***
1st month vs. 2nd month	0.19
1st month vs. 3rd month	0.21
1st month vs. 4th month	0.13
2nd month vs. 3rd month	**0.011***
2nd month vs. 4th month	**0.011***
3rd month vs. 4th month	0.68

P<0.05*, P<0.01**, P<0.001***

TABLE 4.105: PERCENT DISTRIBUTION OF MACRONUTRIENTS OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (MEAN ± SD)

Nutrients	Timeline (Months)	Weight Loss		P value
		<7% (N=12)	≥7% (N=18)	
Carbohydrates (%)	0	55.1 ± 6.1	53.1 ± 5.3	0.35
	1	58.4 ± 7.1	55.5 ± 7.6	0.29
	2	57.4 ± 8.1	54 ± 7.2	0.24
	3	55.5 ± 7.6	54.1 ± 8.4	0.62
	4	56.1 ± 6.7	52.3 ± 8.9	0.23
	F value	0.77	0.77	
Protein (%)	0	11.8 ± 0.82	11.04 ± 1.4	0.052
	1	12.3 ± 1.7	12 ± 1.7	0.63
	2	12.4 ± 2.1	11.8 ± 1.7	0.52
	3	11.7 ± 4.1	12.4 ± 2.2	0.61
	4	11.6 ± 3.9	13 ± 2	0.30
	F value	0.95	**0.034***	
Fat (%)	0	29.6 ± 4.9	33 ± 5.5	0.09
	1	28.3 ± 7	29.5 ± 6.1	0.61
	2	28.8 ± 8.8	30.8 ± 6.6	0.47
	3	25.4 ± 10.2	30.6 ± 7.7	0.12
	4	26.8 ± 10.3	31.5 ± 7.3	0.15
	F value	0.76	0.62	

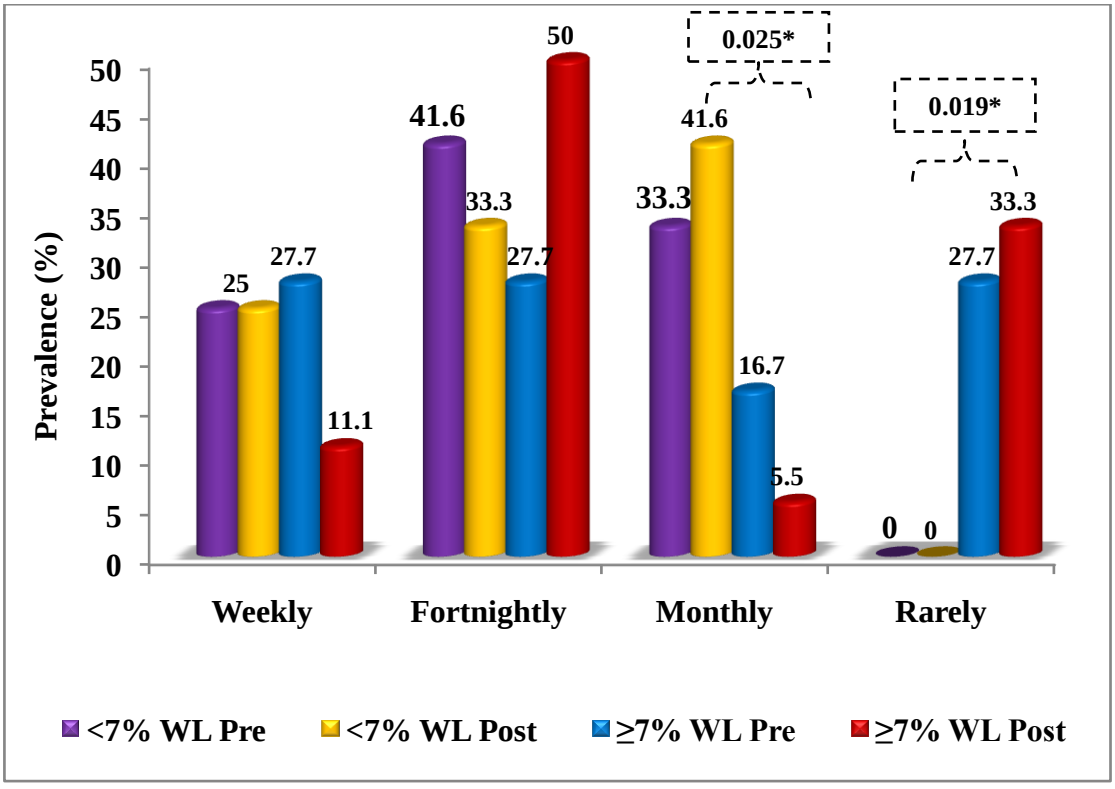
P<0.05*, P<0.01**, P<0.001***

TABLE 4.106: DIFFERENCE IN PERCENT DISTRIBUTION OF PROTEIN OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS

Groups	Protein percent distribution
Baseline vs. 1st month	0.069
Baseline vs. 2nd month	0.12
Baseline vs. 3rd month	**0.040***
Baseline vs. 4th month	**0.0017****
1st month vs. 2nd month	0.80
1st month vs. 3rd month	0.58
1st month vs. 4th month	0.12
2nd month vs. 3rd month	0.45
2nd month vs. 4th month	0.08
3rd month vs. 4th month	0.38

P<0.05*, P<0.01**, P<0.001***

FIG 4.55: IMPACT ON FREQUENCY OF EATING OUT AMONG NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (%)



Impact on Physical Activity Profile

At baseline, the total METminutes/week was non-significantly higher in subjects who lost ≥7% weight compared to subjects who lost <7% weight and the difference between the two became significant after intervention (1622.2 vs. 819.3, P 0.032). This was as a result of a significant increase in total METminutes/week of subjects who lost ≥7% weight, by 52.5% from 1063.2 to 1622.2 (P 0.012) compared to a marginal 18.8% increase in subjects who lost <7% weight (table 4.107).

In subjects who lost <7% weight, proportion of subjects with low physical activity was non-significantly higher at baseline as well as post intervention as more than half were in the said category compared to subjects who lost ≥7% weight wherein only 27.7% at baseline and 16.7% post intervention had low physical activity. The decline in proportion of subjects with low physical activity was adjusted for the rise in proportion of subjects with medium physical activity in both the groups, wherein subjects who lost ≥7% weight from baseline had non-significantly higher proportion of subjects at baseline as well as post intervention (fig 4.56).

Impact on Lipoproteins

TC and LDL-C had non-significant alterations in both groups. HDL-C increased significantly by 13.8% from 44.8mg/dl to 51mg/dl (P 0.0006) in subjects who lost <7% weight and also increased significantly by 10.2% from 48.2mg/dl to 53.1mg/dl (P 0.0007) in subjects who lost ≥7% weight. Triglycerides declined reaching near significance (P 0.06) from 129.9mg/dl to 107.4mg/dl in subjects who lost ≥7% weight and was non-significantly lower (P 0.06) from subjects who lost <7% weight (107.4mg/dl vs. 142.7mg/dl). There was a non-significant decline in VLDL-C in both the groups. Non-HDL-C declined profoundly from 131.5mg/dl to 115.2 mg/dl reaching near significance (P 0.052) in subjects who lost ≥7% weight whereas it increased marginally in subjects who lost <7% weight (table 4.108).

Impact on Prevalence of Dyslipidemia

The prevalence of hypercholesterolemia declined non-significantly in both the groups. However, the prevalence of hypertriglyceridemia increased from 33.3% to 58.3% in subjects who lost <7% weight and reduced from 27.7% to 16.7% in subjects who lost ≥7% weight. Consequently, the prevalence of hypertriglyceridemia became significantly lower in subjects who lost ≥7% weight compared to subjects who lost <7% weight (16.7% vs. 58.3%, P 0.045). Prevalence of low HDL-C was non-significantly higher at baseline in subjects who lost <7% weight compared to subjects who lost ≥7% weight (66.7% vs. 38.8%, P 0.14). After the intervention, the prevalence reduced to 41.6% from 66.7% in subjects who lost <7% weight and declined from 38.8% to 16.7% in subjects who lost ≥7% weight. The prevalence of elevated LDL-C declined significantly from 72.2% to 38.8% (P 0.047) in subjects who lost ≥7% weight and reduced non-significantly from 50% to 33.3% in subjects who lost <7% weight (fig 4.57).

Impact on Lipoprotein Ratios

Other than TC/HDL, all the lipid ratios reduced significantly, namely; TG/H by 22.2% from 2.7 to 2.1 (P 0.017), AIP by 31.7% from 0.41 to 0.28 (P 0.008), LDL/HDL by 21.7% from 2.3 to 1.8 (P 0.045) and nonHDL/HDL by 18.5% from 2.7 to 2.2 (P 0.04) in subjects who lost ≥7% weight (table 4.109). On the other hand, in subjects who lost <7% weight, had a relatively smaller decline in the mean lipid ratios and only TG/H (by 13.5% from 3.7 to 3.2, P 0.021) and AIP (by 16% from 0.50 to 0.42, P 0.006) reduced significantly. After the intervention, the prevalence of TG/H >3 was significantly lower in subjects who lost ≥7% weight compared to subjects who lost <7% weight (16.7% vs. 58.3%, P 0.045). The prevalence of AIP >0.21 declined significantly in subjects who lost ≥7% weight from 94.4% to 61.1% (P 0.040) (fig 4.58).

TABLE 4.107: PHYSICAL ACTIVITY PROFILE OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (MEAN ± SD)

Variables	Stage	Weight Loss		P value
		<7% (N=12)	≥7% (N=18)	
Total METminutes /week	Pre	689.62 ± 696.2	1063.66 ± 880.74	0.20
	Post	819.3 ± 810.26	1622. 22 ± 1146.37	**0.032***
	Paired t	0.49 (↑18.8%)	**0.012* (↑52.5%)**	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis depict proportion

FIG 4.56: PHYSICAL ACTIVITY STATUS OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (%)

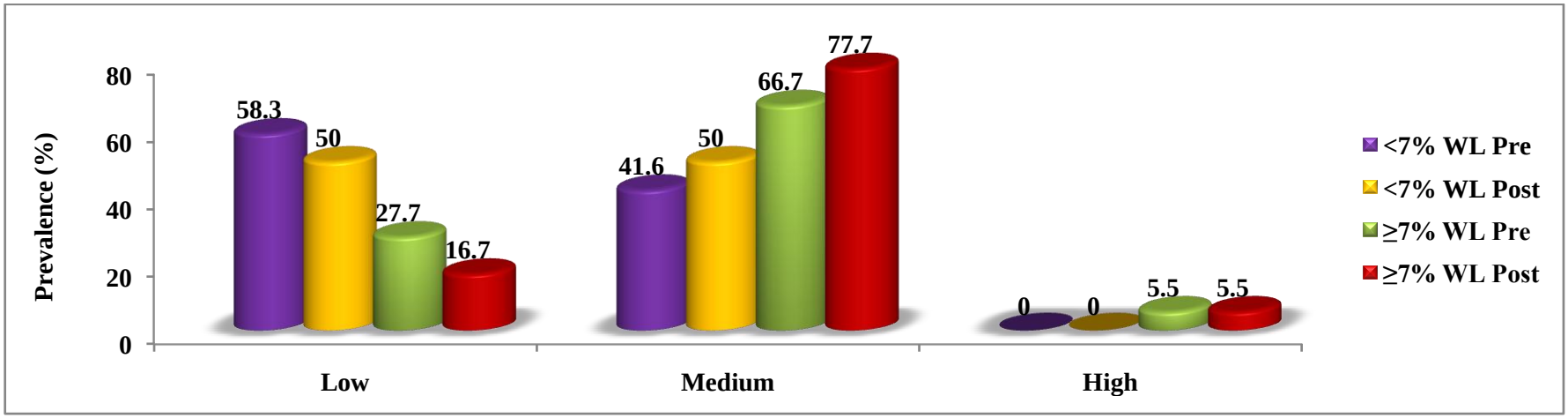


TABLE 4.108: IMPACT ON LIPOPROTEINS OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (MEAN ± SD)

Variable	Stage	Weight Loss		P value
		<7% (N=12)	**≥7% (N=18)**	
TC (mg/dl)	Pre	178.1 ± 25.08	179.7 ± 41.8	0.89
	Post	187.1 ± 35.7	168.3 ± 46.5	0.22
	Paired t	0.17 (↑5.1%)	0.16 (↓6.3%)	
HDL-C (mg/dl)	Pre	44.8 ± 9.9	48.2 ± 9.2	0.35
	Post	51 ± 13.8	53.1 ± 9.3	0.61
	Paired t	**0.0006*** (↑13.8%)**	**0.0007*** (↑10.2%)**	
LDL (mg/dl)	Pre	102.3 ± 20.5	105.6 ± 33.3	0.74
	Post	102.2 ± 21.9	93.1 ± 36.6	0.40
	Paired t	0.97 (↓0.09%)	0.12 (↓11.8%)	
Triglycerides (mg/dl)	Pre	151.8 ± 64.2	129.9 ± 39.2	0.30
	Post	142.7 ± 54.2	107.4 ± 40.5	0.06
	Paired t	0.28 (↓5.9%)	0.06 (↓17.3%)	
VLDL-C (mg/dl)	Pre	30.3 ± 12.8	25.9 ± 7.8	0.30
	Post	27.2 ± 12.5	21.7 ± 7.7	0.18
	Paired t	0.11 (↓10.2%)	0.08 (↓16.2%)	
Non HDL-C (mg/dl)	Pre	133.3 ± 25.8	131.5 ± 37.4	0.87
	Post	136.1 ± 37.1	115.2 ± 44.3	0.17
	Paired t	0.66 (↑2.1%)	0.052 (↓12.4%)	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis depict proportion

FIG 4.57: IMPACT ON PREVALENCE OF DYSLIPIDEMIA ON NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (%)

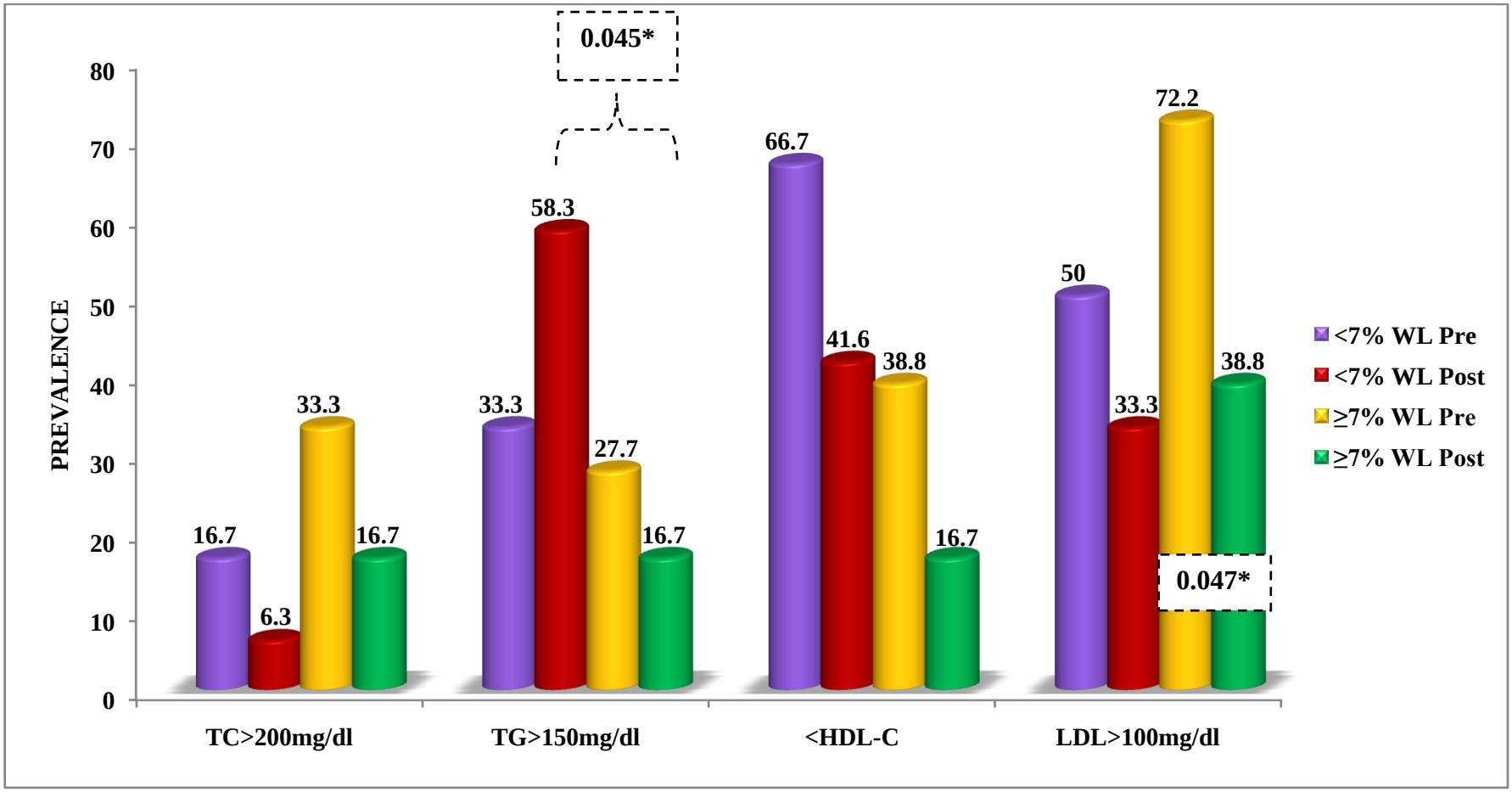
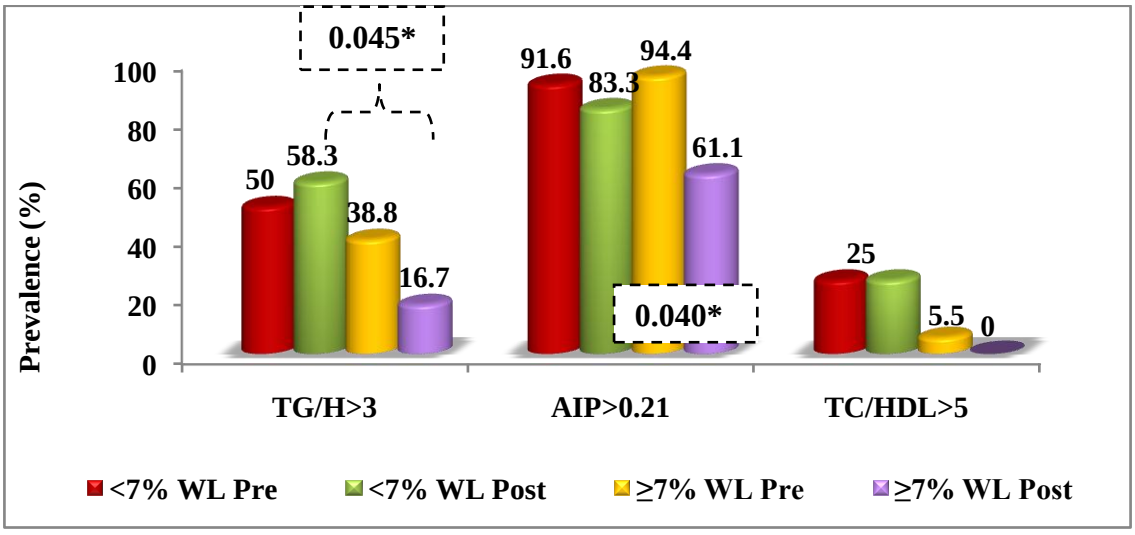


TABLE 4.109: IMPACT ON LIPID RATIOS OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (MEAN±SD)

Variables	Stage	Weight Loss		P value
		<7% (N=12)	≥7% (N=18)	
TG/H	Pre	3.7 ± 2.1	2.7 ± 0.96	0.16
	Post	3.2 ± 1.7	2.1 ± 0.80	0.056
	Paired t	**0.021* (↓13.5%)**	**0.017* (↓22.2%)**	
AIP	Pre	0.50 ± 0.26	0.41 ± 0.16	0.31
	Post	0.42 ± 0.29	0.28 ± 0.18	0.16
	Paired t	**0.006** (↓16%)**	**0.008** (↓31.7%)**	
TC/HDL	Pre	4.08 ± 0.96	3.77 ± 0.81	0.36
	Post	3.9 ± 1.16	3.36 ± 0.85	0.17
	Paired t	0.21 (↓4.4%)	0.058 (↓10.8%)	
LDL/HDL	Pre	2.33 ± 0.61	2.3 ± 0.64	0.90
	Post	2.25 ± 0.67	1.8 ± 0.65	0.08
	Paired t	0.59 (↓3.4%)	**0.045* (↓21.7%)**	
Non HDL-C/HDL-C	Pre	3.12 ± 0.94	2.7 ± 0.82	0.31
	Post	2.87 ± 1.12	2.2 ± 0.84	0.09
	Paired t	0.11 (↓8%)	**0.004** (↓18.5%)**	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis depict proportion

FIG 4.58: IMPACT ON PREVALENCE OF ELEVATED LIPID RATIOS OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (%)



Impact on Hs-CRP

Hs-CRP declined from 4.7mg/l to 3.04mg/l reaching near significance (P 0.052) in subjects who lost ≥7% weight and had non-significant decline in subjects who lost <7% weight (table 4.110). The prevalence of high risk of CVD declined non-significantly from 61.1% to 33.3% in subjects who lost ≥7% weight and reduced from 50% to 41.6% in subjects who lost <7% weight (fig 4.59).

Impact on Glycemic Profile

HbA1c was non-significantly higher at baseline in subjects who lost <7% weight compared to subjects who lost ≥7% weight. However, the difference between the two groups became nil as both had 7.6% as the post value after non-significant decline (table 4.111).

The prevalence of excellent glycemia increased from nil to 11.1%, that of good glycemia from 22.2% to 33.3%. Prevalence of average glycemia declined from 44.4% to 16.7% and that of poor glycemia increased from 33.3% to 38.8% in subjects who lost ≥7% weight. In subjects who lost <7% weight, prevalence of excellent glycemia increased from 8.3% to 16.7%, that of good glycemia increased from 25% to 41.6%, prevalence of average glycemia became nil from 16.7% and that of poor glycemia reduced from 50% to 41.6%. No significant difference in the prevalence was observed between the two groups (fig 4.60).

Impact on Hepatic Profile

Alkaline phosphatase declined significantly only in subjects who lost <7% weight, from 92.7U/L to 80.6U/L (P 0.027). GGT was non-significantly lower at baseline in subjects who lost ≥7% weight compared to subjects who lost <7% weight, however subjects with ≥7% weight loss had significantly lower GGT than the subjects who lost <7% weight after intervention (22.4U/L vs. 32.3U/L, P 0.033) as GGT declined significantly by 20% from 28U/L to 22.4U/L (P 0.007) in subjects who lost ≥7% weight. Prevalence of elevated GGT declined non-significantly in both the groups and was non-significantly higher in subjects who lost <7% weight post intervention.

SGOT had non-significant alterations although it declined by 14.8% in subjects who lost <7% weight and increased mildly in subjects who lost ≥7% weight. SGPT declined significantly only in subjects who lost <7%, from 29.8U/L to 22.4U/L (P 0.031), as it was non-significantly higher at baseline and post intervention in these subjects compared to those who lost ≥7% weight (table 4.112).

Impact on Prevalence of Metabolic Syndrome

The prevalence of MS was similar at baseline with three fourths of the subjects in both the groups having MS. The intervention brought along a significant decline in the prevalence of MS from 77.7% to 44.4% (P 0.043) in subjects who lost ≥7% weight from baseline (fig 4.61). This was owing to a significant decline in the number of features of MS by 24.4% from 3.6 to 2.7 (P 0.0006) (table 4.113). The subjects who lost <7% weight also had a decline in the prevalence of MS from 75% to 66.7% but of non-significant nature.

Impact on Liver Span

The liver span was non-significantly higher at baseline in subjects who lost ≥7% weight, however, post intervention it declined significantly within the group by 6.6% from 179mm to 167.3mm (P 0.004) (table 4.114). The prevalence of elevated liver span decreased from 77.7% to 61.1% in these subjects (fig 4.62).

Impact on Prevalence of NAFLD

As all the subjects enrolled were confirmed cases of NAFLD, the intervention brought about a significant reduction in the prevalence of NAFLD from 100% to 61.1% in subjects who lost ≥7% weight and reduced non-significantly from 100% to 66.7% in subjects who lost <7% weight (fig 4.63).

TABLE 4.110: IMPACT ON Hs-CRP OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (MEAN ± SD)

Variable	Stage	Weight Loss		P value
		<7% (N=12)	≥7% (N=18)	
Hs-CRP (mg/l)	Pre	4.5 ± 3.2	4.7 ± 3.6	0.85
	Post	3.8 ± 3.7	3.04 ± 3.03	0.52
	Paired t	0.28 (↓15.5%)	0.052 (↓35.3%)	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis indicate percentage

FIG 4.59: IMPACT ON Hs-CRP STATUS OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (%)

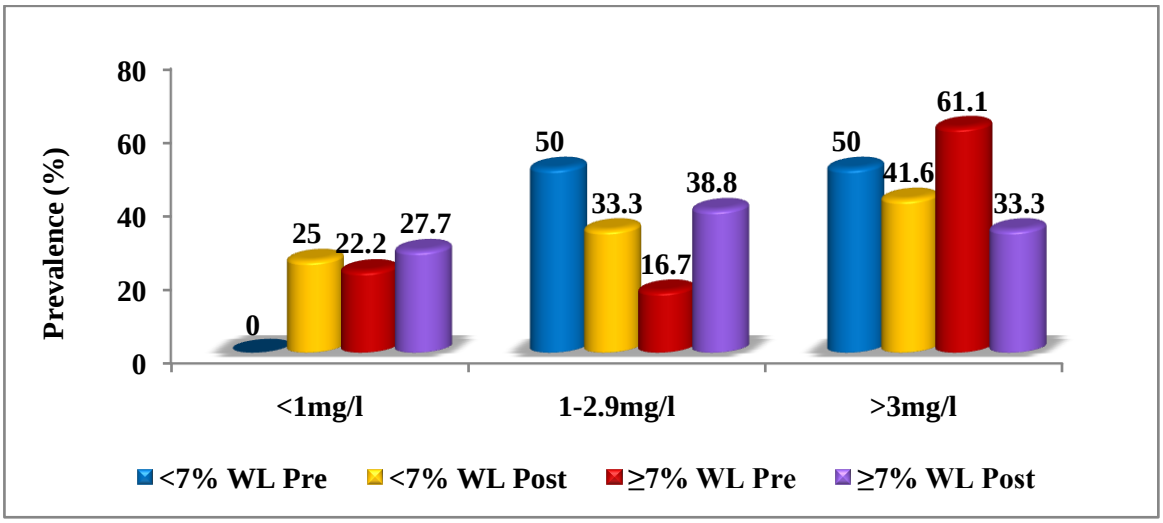


TABLE 4.111: IMPACT ON GLYCEMIC PROFILE OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (MEAN ± SD)

Variables	Stage	Weight Loss		P value
		<7% (N=12)	≥7% (N=18)	
HbA1c (%)	Pre	8.4 ± 2.4	7.9 ± 1.4	0.41
	Post	7.6 ± 1.9	7.6 ± 1.6	0.97
	Paired t	0.09 (↓9.5%)	0.32 (↓3.8%)	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis depict changes in proportion

FIG 4.60: GLYCATED HEMOGLOBIN STATUS OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (%)

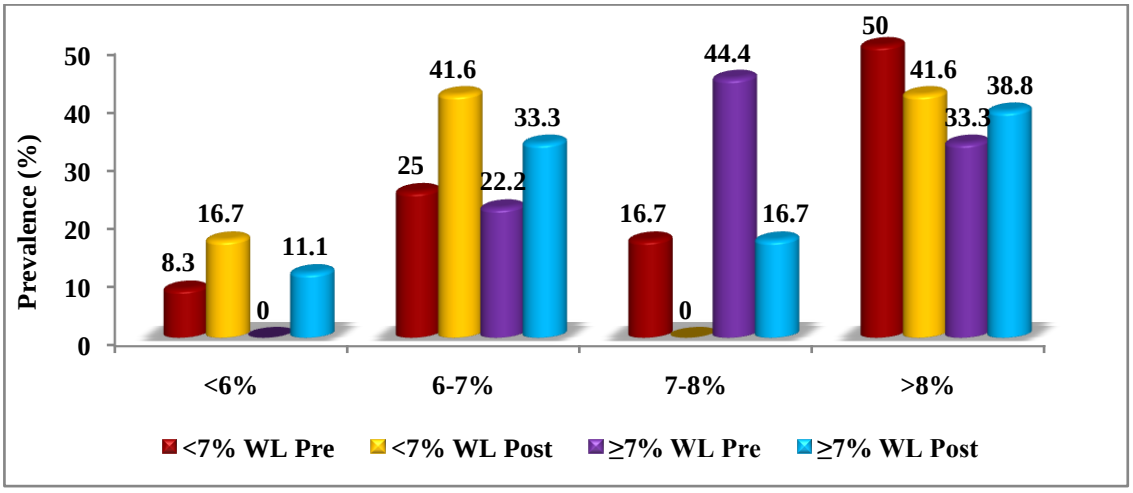


TABLE 4.112: IMPACT ON HEPATIC PROFILE OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (MEAN±SD)

Variables	Stage	Weight Loss		P value
		<7% (N=12)	≥7% (N=18)	
Alkaline phosphatase (U/L)	Pre	92.7 ± 22.3	89.07 ± 25.1	0.68
	Post	80.6 ± 21.4	81.6 ± 20.6	0.90
	Paired t	**0.027* (↓13.1%)**	0.08 (↓8.4%)	
SGOT (U/L)	Pre	26.3 ± 12.4	20.1 ± 7.4	0.13
	Post	22.4 ± 7.6	20.8 ± 10.5	0.63
	Paired t	0.078 (↓14.8%)	0.73 (↑3.5%)	
SGPT (U/L)	Pre	29.8 ± 17.1	23.5 ± 14.5	0.30
	Post	22.4 ± 9.6	19.6 ± 9.5	0.43
	Paired t	**0.031* (↓24.8%)**	0.28 (↓16.6%)	
GGT (U/L)	Pre	39.2 ± 19.9	28 ± 14.3	0.10
	Post	32.3 ± 13.3	22.4 ± 10.9	**0.033***
	Paired t	0.076 (↓17.6%)	**0.007** (↓20%)**	
GGT >35 (U/L)	Pre	6 (50)	4 (22.2)	0.13
	Post	4 (33.3)	3 (16.7)	0.39
	χ^2	0.41	1	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis depict proportion

FIG 4.61: IMPACT ON PREVALENCE OF METABOLIC SYNDROME AMONG SUBJECTS WITH NAFLD ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (%)

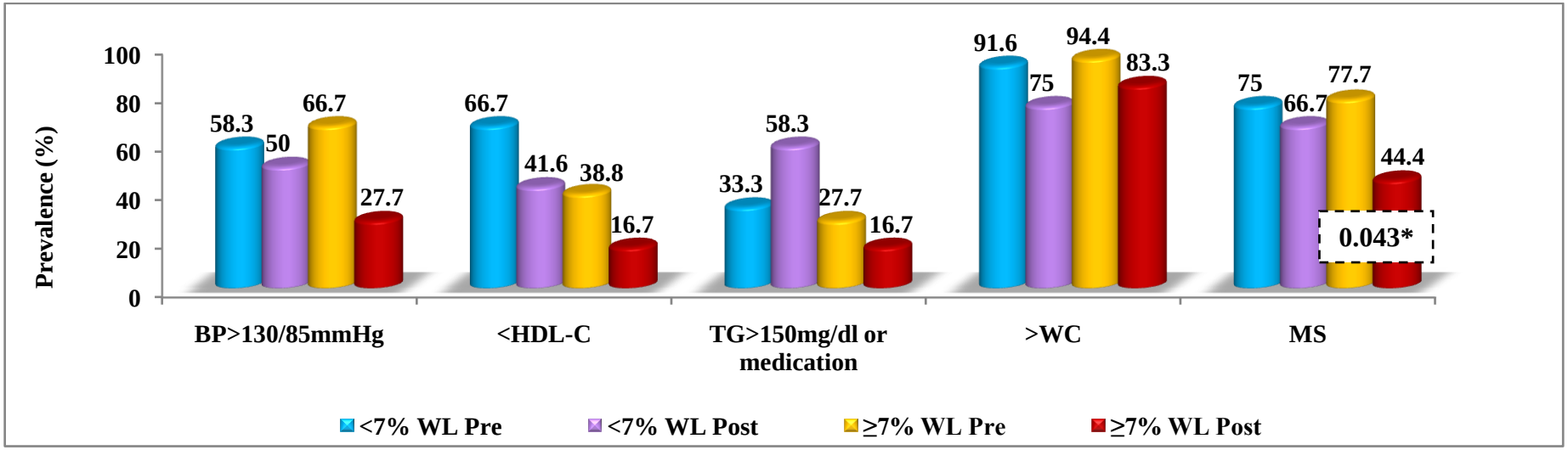


TABLE 4.113: IMPACT ON NUMBER OF FEATURES OF METABOLIC SYNDROME AMONG SUBJECTS WITH NAFLD ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (MEAN ± SD)

Variable	Stage	Weight Loss		P value
		<7% (N=12)	≥7% (N=18)	
No. of features of metabolic syndrome	Pre	3.75 ± 1.42	3.6 ± 1.03	0.77
	Post	3.5 ± 1.38	2.72 ± 0.95	0.10
	Paired t	0.33 (↓6.7%)	**0.0006*** (↓24.4%)**	

Values in parenthesis indicate percentage, P<0.05*, P<0.01**, P<0.001***

TABLE 4.114: IMPACT ON LIVER SPAN OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (MEAN ± SD)

Variable	Stage	Weight Loss		P value
		<7% (N=12)	≥7% (N=18)	
Liver span (mm)	Pre	165.2 ± 22.2	179.1 ± 19.6	0.08
	Post	164.9 ± 23.2	167.3 ± 18.2	0.74
	Paired t	0.95 (↓0.18%)	**0.004** (↓6.6%)**	

P<0.05*, P<0.01**, P<0.001***, values in parenthesis indicate percentage

FIG 4.62: IMPACT ON PREVALENCE OF HEPATOMEGALY OF NAFLD SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (%)

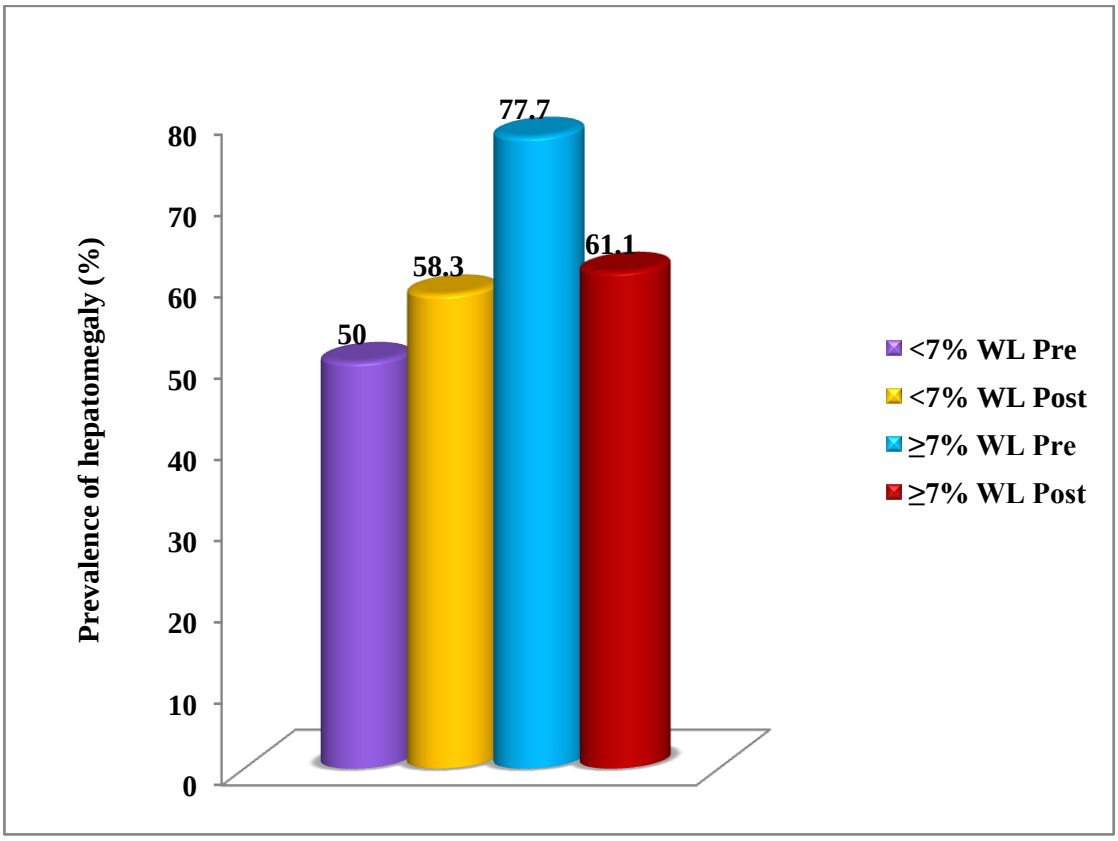
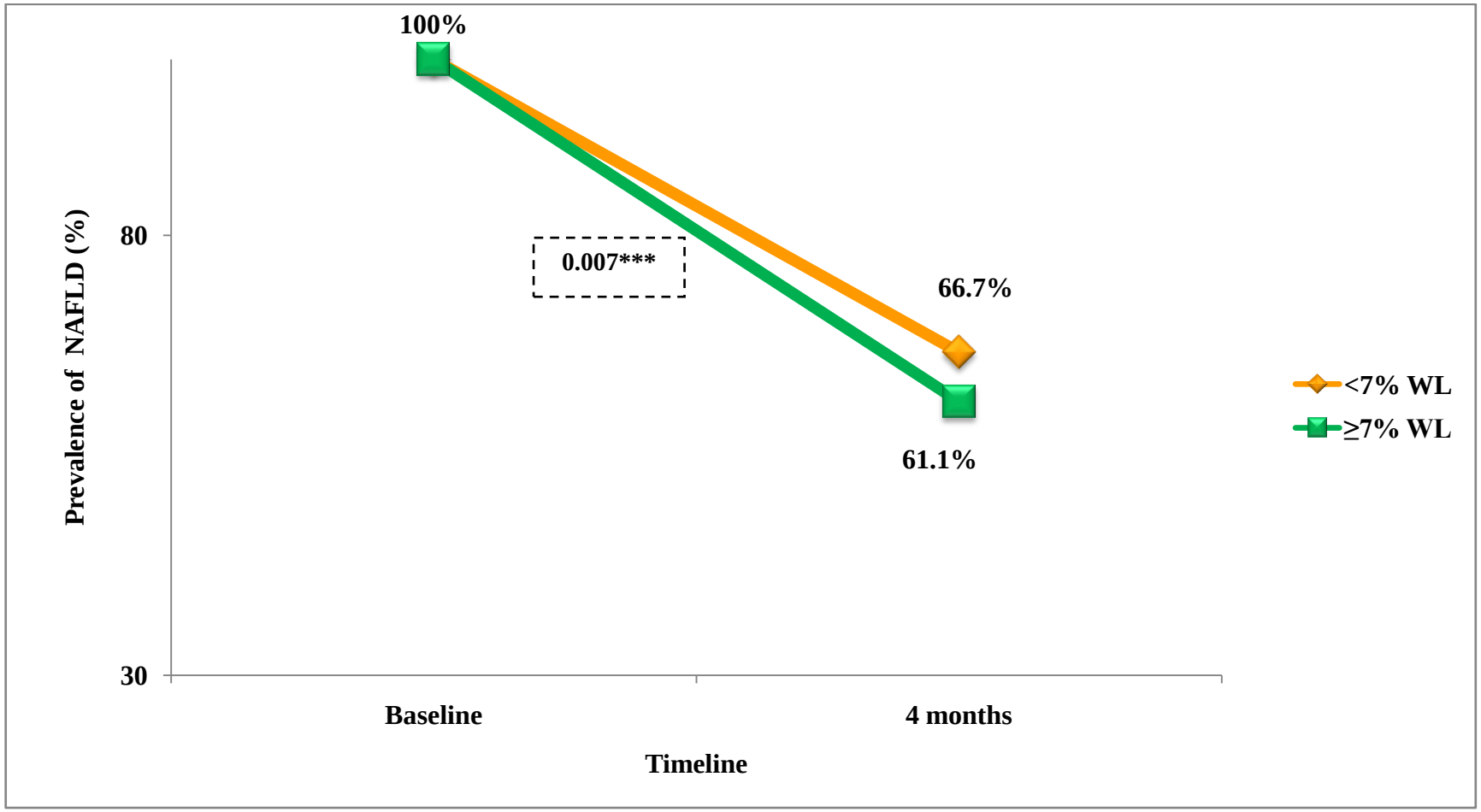


FIG 4.63: IMPACT ON PREVALENCE OF NAFLD IN SUBJECTS ON LIFESTYLE MODIFICATION THERAPY WITH ≥7% WEIGHT LOSS (%)



DISCUSSION

Background

There are no evidence based guidelines for the treatment of NAFLD. Management of the condition relies on correcting the underlying metabolic aberrations (Lewis and Mohanty, 2010). However, lifestyle modification is considered the cornerstone of the management of NAFLD (Bacchi et al., 2013) and is used as the first line therapy for the treatment of NAFLD (Schwenger and Allard, 2014; WGO, 2014), with the key elements weight loss, diet and physical activity (Byrne, 2012; Schwenger and Allard, 2014; Mavrogiannaki and Migdalis, 2013). It is known to increase muscle mass, peripheral insulin sensitivity, improve lipemic status (Colak et al., 2012), inflammation (Schwenger and Allard, 2014), liver histology and delay the progression of NAFLD to more advanced forms (McCarthy and Rinella, 2012).

There are sufficient studies documented on impact of lifestyle intervention on the various bio-chemical parameters, but very few have hepatic data as the end point (Conlon et al., 2013). Evidence on the efficiency of lifestyle modification in NAFLD through behavioural therapy has come from the West and data is lacking in the Indian context. Thus, keeping in mind the primary target of changing the lifestyle, strengthening the self monitoring through education (Shams et al., 2011), and the secondary target of reducing the liver fat content and avoiding disease progression (Shams et al., 2011; Schwenger and Allard, 2014), the research was framed with the objective of imparting nutrition counselling to NAFLD patients to adopt lifestyle modification. It was preluded by developing a booklet on lifestyle modification that was distributed as a ready reckoner to the subjects of the experimental arm. It was hypothesized that lifestyle modification in addition to standard care would aid in improving steatosis compared to type 2 diabetics with NAFLD on standard care alone.

From the first phase of the study, sixty confirmed cases of NAFLD with type 2 diabetes were enrolled on the basis of willingness to participate. They were randomly allocated into two groups; experimental arm and the control arm with thirty in each. They were investigated for knowledge attitude and practices regarding type 2 diabetes and NAFLD along with anthropometric, biochemical, dietary, physical activity assessment and hepatic ultrasonography. Based on the loopholes identified in KAP,

nutrition counselling advocating lifestyle modification was planned. The aim of nutrition counselling was to enhance their knowledge levels that would change their behaviours positively, empower them with doable solutions to modify their lifestyle through diet and physical activity ultimately leading to good practices to manage NAFLD effectively.

To each of the subjects in the experimental arm, inter-personal nutrition counselling was carried out once in a month for a period of four months, in addition to standard care. Knowing that attention spans last not more than forty minutes, each session was designed accordingly. After the session, twenty minutes were kept aside for the subjects to ask queries, if they had any and work out on the practically feasible solutions. Individual goal setting was done to achieve the desired goals. The controls were the recipients of standard care only. Standard care had to be tailor made for each of the subjects based on the underlying metabolic derangements, as there are no set protocols for the treatment of NAFLD.

All the subjects were evaluated monthly for anthropometric, blood pressure, physical activity and dietary assessment as process indicators. At the termination of the study, a reassessment of KAP regarding type 2 diabetes and NAFLD along with anthropometric, biochemical, dietary, physical activity assessment and hepatic ultrasonography was carried out to evaluate the outcome of the nutrition counselling intervention propagating lifestyle modification. A 7% decrease in body weight was selected as outcome measure considering that this value was also the chosen target in the Diabetes Prevention Program (Knowler et al., 2002), the Look AHEAD study (Pi-Sunyer et al., 2007), in the trial comparing cognitive behavioural therapy (CBT) with standard nutritional treatment in NASH (Promrat et al., 2010) and in a CBT study in NAFLD subjects (Moscatiello et al., 2011).

Impact on Knowledge Attitude and Practices

The subjects in both the arms had poor KAP scores about type 2 diabetes and NAFLD at the baseline level. The nutrition counselling intervention improved the scores significantly that led to majority of the experimental arm subjects to have average

KAP score, while a few attained good KAP scores. The controls also improved marginally on the scores as a result of concern over NAFLD as a diseased condition that led to gaining information from the physician and resorting to the internet, however, majority of the subjects remained in the low KAP score category. The fact that the total KAP score of the experimental arm subjects was significantly higher from the controls, establishes the utility of nutrition counselling as a doable technique to bring about significant changes in KAP scores.

In a general population study of 5000 people from the United States, 98% of the subjects stated that their physician had never discussed about NAFLD and 80% had never heard about cirrhosis of liver. About 84% of the subjects were unaware about the risk factors for NAFLD and 70% of them were of the opinion that it was genetic. As many as 93% had no clue as to how NAFLD is diagnosed and 95% felt that hepatic fat deposition would cause serious health problems (Ghevariya et al., 2014).

Medical adherence was achieved through patient education as nutrition counselling in the present study further enhanced the self monitoring aspect of weight loss, estimating fasting blood sugar, blood pressure and improved compliance to medication (Shams and Baraka, 2010; Devins et al., 2005). Importantly, the subjects in the present study also opined to go for regular follow ups, which are a crux component of making them understand the relevance of the same through patient education for effective disease management (Devins et al., 2005).

In a random telephonic survey to determine the awareness about NAFLD among the general population in Hong Kong, 83% of the subjects had never come across the term NAFLD. Respondents who had heard about NAFLD were interviewed in the second phase of the telephonic survey, 47% had no idea about the clinical presentation of the disease and an overwhelming 78% reported that blood tests could be used to diagnose NAFLD. About 46% of the subjects found their knowledge about NAFLD to be inadequate and 35% opined it as highly inadequate. The authors had concluded that the general population of Hong Kong had inadequate knowledge about NAFLD (Leung et al., 2009).

In a KAP study about NAFLD among the residents of Harbin that were selected rando mly, the general population and the NAFLD subjects displayed lack of knowledge reg

arding NAFLD. However, both the groups had a positive and a healthy attitude for N AFLD. But the practice among the NAFLD subjects was significantly lower from the subjects of the general population. In totality, the KAP pass rate was significantly low er in NAFLD subjects than the normal population. The authors concluded that there is a need for strong nutrition education advocacy measure in order to strengthen NAFL D related knowledge, especially among those afflicted with NAFLD **(**Xue-ying et al., 2014).

Studies have shown that the population afflicted with chronic liver disease, for example with cirrhosis (Volk et al., 2013) or hepatitis C (Stein et al., 2001) has a poor understanding about their diseased condition. This lack of knowledge further hinders with self care management. Patients benefit from education as a study showed an increase in understanding of the symptoms, transmission and treatment of the HCV (Kizer et al., 2006). In a study of cirrhotic patients, an educational intervention comprising of a booklet on cirrhosis and its relevance led to a 26% improvement in knowledge about the disease in these subjects (Volk et al., 2013).

It emerged from a study that the general practitioners have bare adequate knowledge about NAFLD and despite training their knowledge on diet composition and steatogenic drugs remained poor. However, the training improved practices regarding screening, NASH detection and managing NAFLD in chronic viral hepatitis. It was concluded that targeted training is required for general practitioners to improve their knowledge and practice regarding NAFLD (Grattagliano et al., 2008).

In a study concerning hepatitis C patients, 17% of the subjects said that the nurse specialist provided education to them regarding maintaining a healthy balanced diet and only a little above 10% said that other related aspects of management were touched upon (Grogan and Timmins, 2010). In another study on Hepatitis C patients, 71% subjects were of the opinion that there was insufficient education material on hepatitis C at the clinic, although many opined that their support person would be interested in receiving education material and participate in education sessions (Jennings et al., 2011). In a similar type of study, more than half of the hepatitis C subjects (52%) felt that their current knowledge regarding liver disease was inadequate and as many as 91% opined that receiving information regarding HCV

was important and very important (Balfour et al., 2004). In cirrhotic patients, about 65% of them cited the need for education on cure for cirrhosis, 45% on diagnosis and transmission of cirrhosis while a substantial chunk also wanted information about the physical and psychological aspects (Zandi et al., 2005).

In health care intervention, educating the patient is of paramount importance as it yields favourable results. Targeting the modifiable factors through dissemination of knowledge may have a substantial impact on liver disease progression and treatment outcomes. Moreover, knowledge leads to improved treatment adherence among the patients, facilitates effective decision-making, which in totality reduces the health care costs and improve the health outcomes in the long run (Valery et al., 2015).

One of the factors that usually hinders with the effective management of type 2 diabetes mellitus and its associated co-morbidities, is the poor knowledge levels among the patients. It further translates into poor attitude and simultaneously poor practices to manage the diseased condition. The idea behind nutrition counselling was to educate the NAFLD subjects about the nature of their liver disease (Wainwright, 2015) and empower them with the knowledge to manage NAFLD effectively. Therefore, KAP intervention through nutrition counselling and providing booklet as a ready reckoner for NAFLD management brought about favourable hepatic changes owing to significant improvement in total KAP score and prevented disease progression. The present study further corroborates the evidence that increasing knowledge in patients with liver disease has the potential to affect behavioural change favourably, enhance patient self efficacy and retard disease progression (Singal et al., 2011).

The authors of a study on lifestyle modification in NAFLD had concluded that an intervention program with only basic nutrition and exercise education is all that would be necessary to promote lifestyle modification (Eckard et al., 2013), the evidence for which comes from the present research. Weight loss through lifestyle modification was proven to be an effective strategy in the management of NAFLD (Papandreou and Andreou, 2015) in type 2 diabetics as well. It resulted in weight loss, increased physical activity, reduced liver enzymes, inflammation, and improved hepatic steatosis status, symbolizing the benefits of lifestyle modification (Schwenger and Allard, 2014).

To ensure long term effectiveness of the adopted lifestyle changes by the intervention arm subjects, an improvement in the weight, biochemical profile, and hepatic status acted as further reinforcement to continue with the improved lifestyle, as was also proposed in a recent review (Sattar et al., 2014).

Impact of Lifestyle Modification on Hepatic Status

The prevalence of NAFLD declined significantly in the experimental arm from 100% to 63.3% and reduced the severity of grade of USG based hepatic steatosis from 1.86 to 1.2 (P 0.00016). Importantly, the impact of nutrition counselling propagating lifestyle modification became even more evident as the prevalence of NAFLD became significantly lower from controls (63.3% vs. 96.7%, P 0.0013) and likewise did the severity of grade of USG based hepatic steatosis (1.2 vs. 1.92, P 0.0003). Hence, lifestyle modification for a period of four months was able to reverse the course of NAFLD, as cited in the literature (WGO, 2014), in 36.7% subjects, maintained grade 2 steatosis in 43.3% subjects and grade 1 steatosis in 3.3% subjects, reduced gross fatty liver to fatty liver in 10% subjects, fatty liver to minimal fatty liver in 3.3% subjects and had only a single subject progress from minimal fatty liver to fatty liver. It was a short term study yet it brought about profound changes in the liver status. A significant reduction in the prevalence of NAFLD was accompanied by decrease in the prevalence of minimal fatty liver, fatty liver and figures becoming nil in grade 3 category.

Liver Status in Relation to Metabolic Syndrome

The prevalence of MS declined in the intervention arm owing to a significant decline in the average number of features of MS signifying CVD risk reduction in these subjects. The intervention brought along a significant decline in the prevalence of MS (P 0.043) in subjects who lost ≥7% weight from baseline owing to a significant decline in the number of features of MS. Though literature points that NAFLD patients with MS are less likely to regress (Hamaguchi et al., 2005), nutrition counselling to adopt lifestyle changes for a period of four months brought about reversal of NAFLD in 36.7% subjects. Of these eleven subjects who reverted to normal liver, eight (72.7%) had MS at baseline and of them seven (63.6%) got

corrected for MS post intervention. The present finding strongly adds to the evidence that when components of MS and the BMI are addressed to, NAFLD can be reversed even when weight may not essentially normalise (Powell et al., 2005).

Liver Status in relation to Weight Loss and Weight Gain

Majority of the subjects in the intervention arm lost ≥7% weight and so did the subjects who reverted to normal liver. Importantly, no weight gain was observed in any of the subjects who were receiving nutrition counselling intervention. Hence, weight reduction indeed turned out to be an effective strategy to reverse the fatty deposition in the hepatocytes (Singh et al., 2015). As weight loss in most of the subjects who reverted to normal liver was ≥7%, it was bound to reverse NAFLD in them as the condition is reversible wherein atleast 3-5% of weight loss is documented (Weiß et al., 2014). Moreover, a weight loss of 7-10% is a mandate in obese subjects if reversal of NAFLD is sought (Bhatt and Smith, 2015).

The controls lost less than 7% weight in majority and majority of the subjects maintained grade 2 hepatic steatosis status. Weight gain was observed in a small chunk of them that turned to be detrimental as two of the subjects negatively progressed from grade 1 steatosis to grade 2 steatosis.

The non-significant 8.1% weight loss in the intervention group was associated with reduction in uric acid, hs-CRP, liver span, VLDL-C, TC/HDL, ferritin, triglycerides and SGPT. Weight loss was significant with a mean 8.7% drop in those who reversed to normal liver and slightly lesser in those who maintained NAFLD (6.3%). The control arm had only 2.4% weight loss and the weight of the intervention arm subjects was significantly lower than the controls by the end of the intervention. Majority of the subjects who reverted to normal liver lost ≥7% weight from baseline. Among the NAFLD subjects who reversed to a normal liver, majority of the obese subjects (66.7%) and normal BMI subjects (75%) lost ≥7% weight from baseline. The close association of weight loss with reduction in adiposity (Kim and Younossi, 2008; Conlon et al., 2013) would have also led to improvement in insulin sensitivity in these subjects, especially among those who reversed to normal liver. The subjects with a normal BMI are also known to benefit from weight loss that results in improvement in

NAFLD (Cho et al., 2014). The intervention brought about a significant reduction in the prevalence of NAFLD from 100% to 61.1% in subjects who lost ≥7% weight.

In a RCT involving obese NASH patients, a combination of diet (1000-1500 kcal/day), exercise (10,000 steps/day and 200 minutes/week of moderate physical activity) and behaviour modification brought about a significant improvement in NASH and reduced weight by 9.3% in the intervention arm. Amongst those having >7% weight loss, a significant improvement in steatosis was also observed. However, the study was hampered by a very high-attrition rate; 65 cases were recruited, 41 entered the study and only 28 completed the study (Promrat et al., 2010), unlike the present study wherein there was no drop-out. In another study, a weight loss of 8kgs in type 2 diabetics who had a poor control, reversed their hepatic steatosis along with normalization of fasting plasma glucose, rate of endogenous hepatic glucose production and the hepatic insulin responsiveness (Petersen et al., 2005).

In the fatty liver ancillary study, the intervention group had moderate calorie restriction (1200-1500 kcal/day), <30% fat of total energy, increased moderate physical activity (175 minutes/week) with 7% weight loss target and the control arm received only education. At the end of one year, the intervention group subjects lost more weight along with a greater decline in BMI, WC, steatosis and HbA1c than the controls. However, SGPT and SGOT had no significant change (Lazo et al., 2010).

Overweight and obese NAFLD patients who underwent three months of diet therapy (50% carbohydrates, 30% fat and 20% protein with a deficit of 500 calories), of the 23 enrolled, fifteen of them decreased one grade of fatty liver and eight others decreased by two grades. A significant correlation was observed between decrease in grade of fatty liver and a decrease in weight and BMI (Tahaei et al., 2010).

A study wherein lifestyle intervention with NAFLD patients comprised of 10 concealing sessions with a dietician and moderate intensity physical activity of 3 hours/week, a significant decrease in body fat and liver fat was observed along with an increase in fitness levels. The condition of NAFL resolved in 20 patients at the end of the intervention (Kantartzis et al., 2009).

A study evaluated 65 patients with NAFLD over a minimum of 3 months who were placed on an aerobic exercise regimen and a specific diet. The exercise regimen consisted of brisk walking; jogging or rhythmic aerobic exercises for a minimum of 45 min, 5 days per week, to achieve a target heart rate of 60–70% of their maximal heart rate. The dietary regimen was predicated on a total of 25 kcal/kg per day containing 60% carbohydrate, 20% fat, 20% protein and 200 mg of cholesterol. A total of 44 patients complied with the exercise programme and were included in the analysis. There was a significant improvement in BMI, WC, WHR and serum aminotransferases in the patients adherent to the diet and exercise regimen (Baba et al., 2006).

In a study involving CBT for weight loss and increasing physical activity in NAFLD subjects, CBT was associated with a higher probability of weight loss, normalization of liver enzymes, improvement in insulin sensitivity and reduction in the MS score. A weight loss ≥7% was found to be beneficial in improving liver biochemistry and histology (Moscatiello et al., 2011).

Impact on Anthropometrics

The reduction in the BMI of the intervention arm subjects was associated with lowering of inflammation as the hs-CRP and uric acid declined. It also brought about shifts in nutritional status, as the prevalence of normal BMI increased and that of obesity decreased. About 80% of the normal BMI subjects, 25% of the overweight subjects and 28.6% of the obese subjects of the experimental arm reverted to normal liver. A 8.4% reduction in BMI was associated with reversal of NAFLD. In a study it was found that atleast 5% BMI reduction is associated with a significant decrease in liver fat and volume in patients with biopsy-proven NASH (Patel et al., 2015).

A non-significant reduction in visceral fat was achieved as the WC (5.23%) and WSR (6.25%) declined in the intervention group, thus contributing to reduction in hepatic steatosis (Belfort et al., 2006; Gastaldelli et al., 2007). Reduction in WC and WSR was associated with reduction in uric acid, hs-CRP, liver span and improvement in physical activity. Importantly, WC reduction was also associated with reduction in the number of features of MS and WSR with reduction in alkaline phosphatase.

Prevalence of elevated WC and WSR declined significantly in the subjects who reverted to normal liver to the extent that it also became significantly lower from subjects who maintained their NAFLD status. A reduction in visceral adiposity means improvement in IR these subjects, leading to downregulation of adipose tissue lipolysis, therefore lesser flux of FFA into the liver (Huang et al., 2005; Day, 2006; Stefan et al., 2008; Tilg and Moschen, 2008). The liver fat content of these subjects would have also gone down owing to its correlation with visceral adiposity (Kotronen et al., 2008; Perseghin et al., 2000). This can be further substantiated by the fact that the liver span reduced significantly only in the intervention subjects; among those who reverted to normal liver and in subjects who lost ≥7% weight. This implies reduced risk of atherosclerosis owing to reduction in VAT (Day, 2006; Stefan et al., 2008; Tilg and Moschen, 2008).

Impact on Blood Pressure

The weight loss in the experimental arm also led to a significant drop in systolic blood pressure by 11.7% at the termination of the study, which was previously significantly higher than that of the control arm subjects at baseline, 1st month and 3rd month. Prevalence of elevated blood pressure dropped significantly in the intervention arm. Moreover, the subjects who maintained their NAFLD status in the experimental arm had a significantly higher prevalence of hypertension than those who reverted to normal liver.

Impact on Inflammatory Status

Ferritin declined significantly after the intervention and especially among subjects who lost ≥7% weight. Since liver is the key metabolic organ central to the regulation of systemic inflammation (Bhatia et al., 2012), it implies a reduction in systemic inflammation in these subjects and reduction in the pro-inflammatory cytokines (Smirnov et al., 1999; Kwak et al., 1995; Pham et al., 2004) as the ferritin reduced.

The intervention led to a significant decline in hs-CRP and consequently reduced the prevalence of high risk of CVD. Also, hs-CRP declined significantly only in subjects who reverted to normal liver (prevalence of elevated CRP also became nil) and their

post treatment value was also significantly lower from the subjects who maintained their NAFLD status. This depicts lower risk of future CVD in these subjects (Ridker, 2001) as decreased plasma concentrations of this acute phase protein resembles decreased inflammatory activity in the arterial wall (Pfützner and Forst, 2006) and significant decline in the presence of inflammation as well (Gohel and Chacko, 2013). The reduction in the hs-CRP was associated with improvement in physical activity status.

Impact on Lipemic Status

Triglycerides and VLDL-C declined significantly with intervention. It is indicative of effective suppression of VLDL-C secretion by insulin that must have decreased concentrations of TG and VLDL-C in circulation (Adiels et al., 2006). The prevalence of hypertriglyceridemia was significantly lower post intervention in subjects who lost ≥7% weight compared to subjects who lost <7% weight. Reduction of triglycerides was associated with reduction in GGT, alkaline phosphatase, liver span and improvement in HDL-C. The prevalence of elevated LDL-C declined significantly in subjects who lost ≥7% weight and reduced non-significantly in subjects who lost <7% weight.

HDL-C increased significantly with intervention and became significantly higher than that of the controls. Subjects with reversal of NAFLD had greater increase in HDL-C than the subjects who maintained their NAFLD status. HDL-C increased significantly in subjects who lost <7% weight and also increased significantly in subjects who lost ≥7% weight.

All the lipoprotein ratios; TG/H, AIP, TC/HDL and LDL/HDL declined significantly with the intervention. Since these ratios are better predictors of CVD risk than isolated lipoprotein fractions (Millán et al., 2009), a significant decrease implies CVD risk reduction in these subjects. Importantly, TG/H, AIP, LDL/HDL and nonHDL/HDL reduced significantly in subjects who lost ≥7% weight.

Impact on Hepatic Profile

Among the liver markers GGT, SGPT and ALP declined significantly in the experimental arm. Infact, post intervention, the experimental arm also had

significantly lower SGPT than the control arm, which is suggestive of IR reduction (Vozarova et al., 2002). An 8.1% weight loss significantly brought down the SGPT by 20.5%, this stands in tandem with the evidence that atleast 5% weight loss is sufficient in bringing down the ALT levels (Daly et al., 2006; Suzuki et al., 2005). Post intervention in subjects who reverted to a normal liver, alkaline phosphatase reduced significantly and was also significantly lower from subjects who maintained their NAFLD status. GGT declined more evidently in subjects who reversed to normal liver than subjects who maintained their NAFLD status. GGT declined significantly in subjects who lost ≥7% weight and was also significantly lower from subjects who lost <7% weight. A reduction in GGT implies CVD risk reduction and vascular oxidative stress reduction in these subjects, which otherwise is implicated to give rise to secondary diabetic complications (Musso et al., 2011; Gohel and Chacko, 2013). Since GGT elevation is associated with visceral obesity and hepatic IR (Gohel and Chacko, 2013), significant GGT decline demonstrates visceral fat reduction, which can be further corroborated by significant decline in WC of these subjects. As the WC declined, it brought down the SGPT as well because abdominal obesity is associated with elevated liver enzymes, particularly, SGPT (Thulstrup et al., 1999; Marchesini et al., 2001). To further support that, alkaline phosphatase, which may have generated from adipose tissue, also went down significantly symbolising reduction in adipose tissue mass owing to decline in WC (abdominal obesity). There are other studies as well which support that lifestyle modification has the potential to bring down the liver enzymes, improve steatosis and reduce hepatic fat content (Kugelmas et al., 2003; Hickman et al., 2004; Bellentani et al., 2008; Suzuki et al., 2005).

Reduction of liver enzymes in the present study correlated with reduction in ferritin and triglycerides and most importantly, with weight loss. A study wherein 152 NAFLD subjects with elevated transaminases were inducted for lifestyle modification counselling that targeted physical activity and nutrition behaviours, the resultant weight loss improved the liver function (St George et al., 2009). However, in another study wherein nutrition counselling improved liver histology, no changes were observed in SGPT or SGOT. But, both the enzymes reduced significantly among the responders (Huang et al., 2005).

Another reason to corroborate the improvement in liver enzymes of the intervention arm subjects is the close association of the former with improved physical activity in the present study. To support it, a 10 weeks moderate intensity physical activity in NAFLD subjects reduced SGPT, independent of the weight loss (St George et al., 2009). However, in the present study most of the subjects had normal transaminase levels, yet they improved on SGPT as a higher level of physical activity is associated with greater reduction in SGPT (St George et al., 2009). Moreover, weight loss or increased physical activity is capable of reducing liver enzymes (Schwenger and Allard, 2014). In two other NAFLD studies as well, liver enzymes were shown to have improved with increased physical activity and gradual weight loss (Ueno et al., 1997; Suzuki et al., 2005), as was also observed in the present study.

Impact on Physical Activity Status

The physical activity profile improved non-significantly for the experimental arm and was also non-significantly higher than the controls at the termination of the study. However, the subjects who reversed to normal liver had a significantly better and improved physical activity profile than the subjects who maintained their NAFLD status at pre and post stage of the study. Subjects with ≥7% weight loss had a significantly improved physical activity after the intervention to the extent that it was better than the subjects who lost <7% weight. It further corroborates that physical activity benefits NAFLD subjects than a completely sedentary lifestyle (Frith et al., 2010) and minor changes in fitness levels can confer major health benefits in NAFLD (Zelber-Sagi et al., 2011) and prevented further progression of NAFLD (Krasnoff et al., 2008). Importantly, these subjects would have benefited as higher levels of physical activity are associated with lower levels of intra-hepatic lipids (St George et al., 2009; Perseghin et al., 2007; Zelber-Sagi et al., 2008; Kantartzis et al., 2009). It is postulated that the reduction of hepatic steatosis is due to the activation of protein kinases activated by adenosine monophosphate (Zelber-Sagi et al., 2007). Physical activity up-regulates the insulin receptors in the muscle tissue which increases the delivery of glucose and insulin to the muscles (Goodyear and Kahn, 1998), thereby improves substrate utilization in the muscles (Zelber-Sagi et al., 2007). Especially in type 2 diabetics, it improves insulin sensitivity (Boule et al., 2001; Umpierre et al., 2011; Kelley and Mandarino, 2000; Kantartzis et al., 2009) by enhancing beta

oxidation which retards fat accumulation in the hepatocytes (Kelley and Mandarino, 2000) and enhancing whole-body lipid oxidation (Hannukainen et al., 2007) and reduces the prevalence of cardio-metabolic risk factors (Boule et al., 2001; Umpierre et al., 2011) as was also evident in the present study, wherein improvement in physical activity was significantly associated with reduction in WC, WSR and hs-CRP. The physical activity data is less likely to have been over-reported by the subjects as improvements in the same were triangulated by significant increase in HDL-C.

Self done physical activity has a higher rate of acceptance than group physical activity (Perri et al., 1997), because of which the former approach was chosen in the present study. Walking and yoga were the most commonly undertaken physical activities by the intervention subjects. Walking at a steady pace for 30-45 minutes or any form of physical activity improves glycemic (Cobo et al., 2008; Boule et al., 2001; Umpierre et al., 2011) and lipemic profile possibly through stimulation of beta oxidation of fatty acids and DNL inhibition in the liver through AMPK activation (Kaser et al., 2010).

In a RCT on sedentary type 2 diabetic NAFLD subjects revealed that four months of either aerobic or resistance training was equally effective in reducing the hepatic fat and one fourth of the subjects indulging in either of the two activities became free of steatosis. Both forms of exercise improved insulin sensitivity and reduced total body fat mass, VAT and superficial and deep subcutaneous fat and HbA1c (Bacchi et al., 2013). In a study on exclusive impact of four months of aerobic exercise, hepatic triglyceride content reduced by 21% even in the absence of weight loss (Johnson et al., 2009). In a study that involved intense psychological counselling for improving physical activity, physical fitness improved along with liver fat reduction, which was independent of weight loss (Montesi et al., 2014).

In another study on the impact of aerobic exercise in NAFLD, the subjects were advised regular aerobic exercise for 30 min/d, for at least 5 d/wk and moderate energy restricted diet for the obese was advised as well. The compliant subjects had a decrease in IR, BMI, WC and SGPT at 6 months. Of the very few subjects in the compliant group who underwent repeat liver biopsy, significant changes were observed in steatosis, necro-inflammation and the NASH score improved. The

improvement in insulin sensitivity correlated with decline in SGPT and liver histology (Bhat et al., 2012).

Impact on Nutrient Intake

The intervention brought about a non-significant decline in energy, carbohydrate and fat intake. Similar trends were observed in subjects who lost ≥7% weight. The intervention also non-significantly increased the intake of protein, crude fibre, total dietary fibre and insoluble dietary fibre. However, the soluble fibre intake increased significantly with the intervention, especially among subjects who lost ≥7% weight. Consumption of vegetables, whole grains (cereals and pulses) and citrus fruits was propagated to the intervention arm subjects. Whole grains help to decrease visceral fat, improve obesity, dyslipidemia and MS (McKeown et al., 2009; Katcher et al., 2008). Importantly, carbohydrates high in indigestible and fermentable fibre, low in GI aid in maintaining glucose concentrations, insulin and FFA (Zivkovic et al., 2007).

The subjects who lost ≥7% weight, their proportion of protein intake in diet increased significantly. Though it was not a very high protein intake and content, it could have possibly reduced intra-hepatocellular lipids (Bortolotti et al., 2009). Dietary protein may aid in the management of NAFLD as catabolism of amino acids requires energy and a high protein intake may trigger increased β oxidation of fatty acids via an increase in the energy expenditure of the hepatocytes (de Wit et al., 2012). A good protein intake aids in weight loss and improves glucose homeostasis and nullifies the impact of a high fat diet on the intra-hepatic lipids (Carvalhana et al., 2012; Mouzaki and Allard, 2012; McCarthy and Rinella, 2012; Bortolotti et al., 2009; Bortolotti et al., 2011; Tovar and Torres, 2010). It is postulated that high protein intake leads to a higher metabolic rate of amino acids in the liver and consumes large energy. The excess energy consumption increases lipid oxidation and hence aids in preventing fat accumulation in the hepatocytes (Leidy et al., 2007). Protein intake is essential for the regeneration of hepatocytes and supplies crucial amino acids that prevent excessive fat accumulation within hepatocytes (Leclercq and Horsmans, 2008).

The proportion of energy being derived from fat became significantly lower from controls at the 1st month, being less than 30%. The proportion of energy coming from protein also increased marginally. A low fat diet was recommended to the intervention

arm subjects without much emphasis on energy restriction (Fan and Cao, 2013) as low fat diets are safe, cardio-protective and effective in weight loss, as was proven in the present research (Gill and Wu, 2006).

Impact on Frequency of Eating Out

In the experimental group the proportion of those eating out frequently declined. Though non-significant, monthly and rarely eat was more prevalent in subjects who reverted to normal liver. Eating out rarely was significantly prevalent in subjects who lost ≥7% weight. The subjects seem to have abided by the recommendation of having more of home cooked food than eating outside food (Yasutake et al., 2014).

Impact on Glycemic Status

Reaching near significance, the decline in HbA1c was more prominent in the intervention subjects, who improved on their hepatic status, than the controls. The prevalence of excellent and good glycemia increased, that of average glycemia decreased and of poor glycemia remained unaltered in these subjects. Lowering of the HbA1c was associated with improvement in HDL-C, reduction in the number of features of MS and VLDL-C. Thus, glucose control improved with physical activity (Fan and Cao, 2013; Thoma et al., 2012).

Advantages of the study

The presence of the control arm provided scope for comparison between standard care alone vs. standard care plus nutrition counselling. The data was well matched for most of the variables in both the arms. A relatively long duration of the intervention and assessment of the impact by an array of anthropometric, biochemical parameters and imaging data gave a holistic picture of the efficiency of nutrition counselling in the management of NAFLD. Given that there were no significant differences in standard care (the prescription of lipid lowering drugs, anti-hypertensive drugs and hypoglycaemic agents) for both the arms, it is suggestive that any improvement in the biochemical and imaging profile of the experimental arm subjects can be attributed to lifestyle modification *per se*.

A significant disadvantage of the study was no use of liver biopsy to track the hepatic changes. However, since the technique is marred by its own set of disadvantages, ultrasonography was made use of as a guide to weight loss as the technique has been found to be effective in tracking changes in hepatic steatosis owing to weight loss (Tahaei et al., 2010).

Lifestyle intervention was based on the principles of CBT. It was a combination of diet and exercise advice and positive behaviours as proposed by Wadden et al., 2004 targeting the education component that can be modified and relearned, which are likely to harbour greater benefits than dietary prescription alone (Moscatiello et al., 2011; McCarthy and Rinella, 2012). Importantly literature states that interventions with a CBT approach for the treatment of NAFLD are likely to be effective in the long run (Greaves et al., 2011) as behavioural approach provides the patients with practical instruments to achieve the desired lifestyle changes (McCarthy and Rinella, 2012). Various strategies were adopted (Bellentani et al., 2008) to engage and involve the NAFLD subjects to modify their lifestyle for the better. Because targeting behavioural methods to impart nutrition education is an effective and useful way of dealing with a health problem (Yasutake et al., 2014), behavioural techniques were made use of for inducing weight loss through lifestyle modification (Bellentani et al., 2008; Moscatiello et al., 2011; Wadden and Foster, 2000; Fabricatore, 2000); such as goal setting for meeting weight loss targets, apt calorie intake, physical fitness requirements; self monitoring of weight, blood pressure, blood glucose, physical activity, food intake so as to not to under or over eat; stimulus control by removing the negative factors and favouring the positive factors for weight loss, alternative behaviours by engaging in non-eating activities that induce relaxation; problem solving sessions to address barriers to weight loss and cognitive restructuring by promoting rational thinking. Advantages of these strategies, as discussed in the methods chapter, could be one of the possible reasons why inter-personal nutrition counselling led to positive changes in these subjects.

Another significant advantage of the study was zero attrition rates in both the arms, despite no compensation or remuneration for participation. The possible reason behind this could be the regular and intense monitoring of the subjects involved in the intervention that were followed up by monthly visits and weekly telephonic calls and

the controls followed up by monthly visits too. Moreover, the participation in the intervention was enhanced owing to individualistic approach rather than a group approach. However, a significant disadvantage of the study was the possibility of recall bias in the dietary data.

CONCLUSIONS

The four months nutrition counselling intervention non-significantly reduced the anthropometric parameters and significantly reduced the SBP, ferritin, triglycerides, lipid ratios, hs-CRP, alkaline phosphatase, GGT, SGPT, number of features of MS, liver span and significantly increased the HDL-C that led to a significant decline in the prevalence of NAFLD from 100% to 63.3% and in the severity of hepatic steatosis as well.

The ≥7% weight loss in the intervention arm was associated with significant reduction in BMI, WC, WSR, SBP, ferritin, lipid ratios, GGT, number of features of MS, liver span and increased HDL-C and total METminutes/week significantly.

Inter-personal counselling reversed NAFLD in a little more than one third of the subjects. Though the KAP scores showed an overall improvement for the controls as well, of much less intensity than the experimental arm, they failed to make an impact on the biochemical and imaging data. This reflects the impact that inter-personal nutrition counselling has over and above the standard care. In the absence of evidence based guidelines, nutrition counselling to adopt lifestyle modification can be a viable strategy to manage NAFLD in type 2 diabetics and may also be used as a prophylactic measure.