Follow up case study for ninth months related lipostatic non-alcoholic fatty liver disease (NAFLD) characteristic values under nutrition intervention only

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Abstract :The project study aims to shed light on follow up case study for ninth months related lipostatic non-alcoholic fatty liver disease (NAFLD) characteristic values under nutrition intervention only .Because lipostatic (NAFLD) is considered by many researcher to be the hepatic manifestation of metabolic syndrome, defined as the presence of 3 or more of the following: obesity, hypertriglyceridemia, low high-density lipoprotein levels, abnormality values of thyroxin and high fasting glucose levels, So, characteristic parameter conducting for Lipostatic (NAFLD) was carry on such as ultrasound scan , some Anthropometric measurements', Seroandocrinological assay of the thyroxin T3 and T4 phase, Serological study of lipid profile , atherogenic index of plasma (AIP), Hematology study of cumulative hemoglobin A1c (HbA1c%) . The results showed an improvement in all special indicators. Ultrasound showed a normal picture of all study cases, such as blood lipoprotein, and atherosclerotic plasma AIP (also BMI, T3ng/dL, and T4ug/dL, respectively). In conclusion nutrition intervention only revealed correct characteristic values reach to normal after ninth months follow up every three months. Study recommended decreased body weigh 10% from actual weight, fat 15% /total calories, α- tocopherol, Ginger not more 5gm divided/daily and coffee. Also restricted some food items as fructose, maize syrup, Grape, Date, Molasses, Honey bees, fried's and grilling.

Key words: lipostatic (NAFLD), nutrition intervention, lipid profile, BMI, thyroxin, T3,T4, cumulative hemoglobin, Anthropometric measurements, atherogenic index, ultrasound, restricted, food items, fructose, Ginger, α- tocopherol.

1. Introduction

NAFLD is considered by many to be the hepatic manifestation of metabolic syndrome, defined as the presence of 3 or more of the following: ,1) abdominal obesity (waist circumference _102 cm in men, _88 cm in women), 2) hypertriglyceridemia (150 mg/dL), 3) low high-density lipoprotein (HDL) levels (_40 mg/dL in men, _50 mg/dL in women), 4) hypertension (_130/80 mm/Hg), and 5) high fasting glucose levels (_110 mg/dL) (Ford *et al.*,2002).Other conditions that result in hepatic steatosis include total parenteral nutrition, rapid weight loss, acute starvation, abdominal surgery (extensive small bowel resection, biliopancreatic diversion, jejunoileal bypass), drugs or toxins (amiodarone, tamoxifen, glucocorticoids, estrogen, antiretroviral agents, tetracycline), abetalipoproteinemia, lipodystrophy, and Wilson's disease (Ford *et al.*,2002). Lack of physical activity, an overabundance of calorie rich food, and genetic/epigenetic influences have resulted in an epidemic of NAFLD throughout the world. Recent data

demonstrate that NAFLD patients consume fast food more frequently and exercise less than non-NAFLD patients (Lazo and Clark, 2008).Hepatic steatosis is the net result of impaired insulin signaling and development of IR as a result of excessive accumulation of free fatty acids (FFA). The accumulation of intramyocellular lipid as a result of excess FFA within skeletal muscle leads to the development of toxic lipid metabolites such as diacylgylcerol, ceramides, and fatty acyl CoA, which results in insulin signaling defects and eventual impaired skeletal muscle glucose metabolism. Hyperglycemia leads to activation of carbohydrate response element binding protein, (Cusi, 2009). and elevated circulating plasma insulin levels lead to the activation of sterol regulatory element binding protein-1 (SREBP), which increases de novo lipogenesis (Kanuri *et al.*, 2011). Based on the above pouch us to apply this work as trailed to investigate and shed light for the if role of diet when follow up case study for ninth months related lipostatic (NAFLD) lead to enhancement of characteristic values under nutrition intervention only.

2. Subject and Methods

The study cohort was planned for a number of males and females, 43 of whom were 20 males and 23 females, but the experiment was actually performed on cohort including (16 males and 11 females which applied research, half count of male was smoker while rest of the number of male and all count of females were non-smoker) Others were excluded as a result of inadequate health information, lack of cooperation and constant absence and lack of commitment to the research plan. Cohort subject was volunteers were diagnosed as NAFLD in special abdominal clinic while follow up was in both obesity & thin private center and special abdominal clinic since 2014-2017.

Design Experience

The nature of lipostatic and the need to implement the food intervention strategy were explained for all cases of study referred to in Table 2.

Calories, protein, carbohydrate and fat were adjusted for all cases study cohort as indicated in Table 1.

A preliminary estimate was made of the nature of the lipostatic for all cohort cases study as a preliminary study at the beginning of the baseline and then every three months and continued for nine months.

Methods

The following laboratory studies and tests were performed.

A descriptive study of the age and sex of the study cohort according to (Hitoshi *et al.*, 2012)

Sonar before and after nutrition intervention an ultrasound scan was carry on according to (Lee and Park 2014).

Model and Sonar logistic

ACUSON X 700 Ultrasound Systems.

Ultra-sensitive wideband transducers, matched with user-selectable MultiHertz™ multiple frequency imaging, improve resolution and penetration. Up to seven 2D and THI frequencies and up to two color Doppler and spectral Doppler frequencies expand the clinical versatility of a single transducer, thereby maximizing transducer investment. Innovative ultra low-loss lens materials and microelectronic technologies for efficient performance and increased signal bandwidth microCase™ transducer miniaturization technology and SuppleFlex™ transducer cables SuppleFlex cables and integrated cable management provide protection during exams and transport Independent 2D and color frequencies for optimal resolution and penetration Frequency range: 1.3 – 16.0 MHz acoustic technology Universal, stainless steel and disposable biopsy guides for specified linear and curved array transducers.Made in USA.

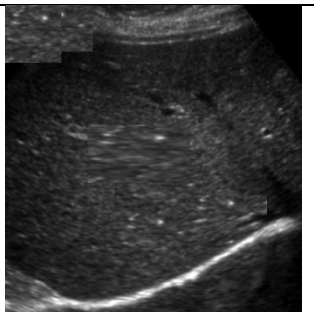
Anthropometric Study of BMI before and after nutrition intervention an ultrasound scan was carry on according to (Adams *et al.*, 2005).

Serological study.

Seroandocrinological assay of the thyroxin T3 and T4 phase, according to (Carole, 2017). Serological study of lipid profile according to Anon (2010).Calculation of arteriosclerosis factor expressed as atherogenic index of plasma (AIP applied to the formula, log (TG/HDL-C) according to (Dobiasova & Frohlich, 2001; Dobiášová & Frohlich, 2001 and Tan MH *et al.*, 2004; Nwagha *et al.*, 2010 finally Mudhaffar,2015).Hematology study of cumulative hemoglobin according to (Perlemuter *et al.*, 2007)

3. Results and discussion

Nonalcoholic fatty liver disease (NAFLD) is a frequent cause of chronic liver diseases, ranging from simple steatosis to nonalcoholic steatohepatitis (NASH)-related liver cirrhosis. Although liver biopsy is still the gold standard for the diagnosis of NAFLD, especially for the diagnosis of NASH, imaging methods have been increasingly accepted as noninvasive alternatives to liver biopsy. Ultrasonography is a well-established and cost-effective imaging technique for the diagnosis of hepatic steatosis, especially for screening a large population at risk of NAFLD. Ultrasonography has a reasonable accuracy in detecting moderate-to-severe hepatic steatosis although it is less accurate for detecting mild hepatic steatosis, operator-dependent, and rather qualitative (Lee and Park 2014).



Diffuse fatty liver replicate 27. Before

Normal replicate 27 .After

Figure (1): Script fallow-up examine sonar before and after dietary intervention on lipostatic (NAFLD)

When comparing the total frequency of sonar images before and after the food intervention, the difference was observed. Before the food intake, the bright spots on the liver showed signs image of a steatotic liver, showing increased echogenicity of the liver parenchyma of food interference a normal liver, showing that the echogenicity of liver parenchyma. The sonar images showed that the liver was normal and the bright spots disappeared (Lee and Park 2014).

Table (1): Basic characteristics related to the lipostatic (NAFLD) case study

Variables	males (n = 16)			females (n = 11)			
	Age\years			Age\years			
mean ±SD	42.5±12.195			48±12.193			
BMI (kg/m2)	27.066±2.040			29.395±2.234			
Total thyroxin ug/dl	maleT3ng/dL	MaleT4 ug/dL		femaleT3ng/dL	femaleT4 ug/dL		
mean ±SD	64±9.063	3.281±0.555		63.091±8.336	5.818±1.722		
Hemoglobin A1c (HbA1c) %	male			female			
mean ±SD	7±0.23			6.4±0.4			
lipid profile	male\female						
	cholesterol mg/dl	Non-HDL-c	LDL-c	HDL-c	VLDL-c	TGr	AIP
mean	241.50\229	169.05\160.3	144.90\137.4	72.45\68.7	24.15\24.15	120.75\114.5	0.01\114.5
±SD	12.04\3.18	8.43\2.22	7.23\1.91	3.61\0.95	1.20\0.32	6.02\1.59	0.00\0.00
strategically nutrition intervention roles	Criteria every three months with nutrition intervention .only						
weight	Decreased 10% from actual weight						
protein	2gm/Kg actual weight						
CH.	(Total cal) - (protein cal +fat cal) /4						
Fat	15%						
Vegetables	recommended						
Fruits	recommended						
water	Actual w. /8 formula						
recommended	α- tocopherol, Ginger not more 5gm divided/daily, coffee.						
Restricted food items	Fructose, Maize syrup, Grape, Date, Molasses, Honey bees, fried's, grilling.						

In table (1), brief basic characteristics related to the lipostatic (NAFLD) case study. Non-alcoholic fatty liver disease (NAFLD) is a pathological condition characterized by aberrant triglycerides accumulating in the hepatocytes,
(Chalasani *et al.,* 2012). No established therapy of NAFLD has been identified yet (EASD, 2016) and (Liyanagedera *et al.,* 2017). The growing pattern of NAFLD prevalence is generally attributed to a global increase in the prevalence of obesity and other metab.lic risk factors (Day CP. 2011) also, Impaired gluc.se tolerance, and central obesity, are am.ng the risk factors for NAFLD (Amarapurkar *et al.,* 2007) and (Yamada *et al.,* 2010). The thyroid gland is significantly involved in energy homeostasis, lipid and carbohydrate metabolism, regulation of body weight and dipogenesis (Michalaki *et al.,* 2006 & Raftopoulos 2004). In a clinical setting, subclinical hypothyroidism has been associated with metabolic syndrome, cardiovascular mortality and disturbance of lipid metabolism (Pucci *et al.,* 2000&Rodondi *et al.,* 2010). In recent years, growing body of evidence has led to speculation on the association between NAFLD and thyroid dysfunction. Herein, also, Nutritional intervention strategically depending on the vast clinical burden of NAFLD and its .overlapping pathogenesis with CVD and diabetes, there is a huge amount of ongoing search and knowledge is advancing rapidly. Nutrition research in NAFLD is complicated by methodological challenges and to date there is not a clear single dietary approach for patients with NAFLD. Literature from the past time trailed to investigate on shed light the role of diet in NAFLD. Nutrition also, may be the key (Armstrong *et al.,* 2013). Fructose is a lipogenic carbohydrate that contributes to insulin resistance, hypertriglyceridemia and appears to be associated with the severity of NAFLD. Fructose absorption and malabsorption may alter and which c.uld be mediating effects on the liver. The role of fructose in NAFLD appears to be two-fold; one of inducing TG production via de-novo. Lipogenesis and resulting in hyperlipidemia and 2nd, contributing to inflammation resulting in insulin resistance, hepatic inflammation and fibrosis. Early work in mice suggested a role for the influencing response to fructose increased endotoxin in portal venous blood and administration of antibiotics decreased both endotoxin and hepatic steatosis associated with fructose (Bergheim *et al.,* 2008, Spruss *et al.,* 2009& Kanuri *et al.,* 2011). An improvement in hepatic steatosis with combination atorvastatin, vitamin C, and vitamin E compared with placebo. group but was limited by lack of biochemical and histological end points (Foster *et al.,* 2011). The most studied agents in this class are vitamin E, vitamin C, and betaine. Vitamin E (tocopherol) is a lipid-soluble antioxidant that has been investigated in the treatment of NASH. Sanyal *et al.,* (2010) shed light vitamin E therapy (at doses of 800 IU/d) with significant improvement in steatosis and lobular inflammation. An inverse correlation with coffee consumption and decreased risk of liver disease was first described 20 years age. In alcoholic cirrhotic patients (Michalaki *et al.,* 2012). Amounts of weight loss in placebo. and or list at arms and linked improvement in hepatic steat.sis and necroinflammation to a threshold weight loss of 9% body weight, recommendations Agree with both(Zelber-Sagi *et al.,* 2006) and (Harrison *et al.,* 2009). Also, ginger consumed for enhancement NAFLD (5g/d as portion divided into 3time) according Tarek *et al.,* (20015).

Table (2): Total thyroxin (ug/dl) ninth month's fallow-up far dietary intervention lipostatic (NAFLD) case study

Total thyroxin ug/dl baseline (first fallow-up) without dietary intervention				
Category	maleT3ng/dL	maleT4 ug/dL	femaleT3ng/dL	femaleT4 ug/dL
mean	64	3.281	63.091	5.8181
±SD	9.063	0.555	8.336	1.722
Total thyroxin ug/dl second Fallow-up with dietary intervention				
mean	65.063	6.6563	66.091	6.509
±SD	5.079	0.318	7.370	1.825
Total thyroxin ug/dl thirdly Fallow-up with dietary intervention				
mean	65.213	5.275	70.182	7.300
±SD	0.307	0.177	6.129	1.236
Total thyroxin ug/dl fourth Fallow-up with dietary intervention				
mean	62.867	5.567	66.600	7.360
±SD	1.455	0.215	1.430	0.276

Data was clarifying in Table (2) which showed total thyroxin (ug/dl) ninth month's fallow-up far dietary intervention lipostatic (NAFLD) case study. Data investigated and manifested enhancement far T4 and T3 for males and females respectively when compared with normal values 57-159 nmol/L (4.5-12.5 ug/dL) and 1.2-2.8 nmol/L (80-180 ng/dL) according (Carole, 2017). Thyroid hormones help regulate of body weight, lipid metabolism, and insulin resistance. Therefore, thyroid hormones may have a role in the pathogenesis of non alcoholic fatty liver disease (NAFLD) and none alcoholic steatohepatitis (NASH). Nan alcoholic fatty liver disease (NAFLD) represents a broad clinical spectrum ranging from simple fatty liver t. non alcoholic steatohepatitis (NASH), which may Endocrine hormones are generally involved in cell metabolism, regulation of energy expenditure and fat distribution in the human body and thereby play an important role in the development of metabolic abnormalities. The thyroid gland is significantly involved in energy homeostasis, lipid and carbohydrate metabolism, regulation of body weight and dipogenesis (Raftopoulos *et al.*, 2004 and Michalaki, 2006). In a clinical setting, subclinical hypothyroidism has been associated with metab.lic syndrome, cardiovascular mortality and disturbance of lipid metabolism (Pucci ,2000&Rodondi *et al.*, 2001).

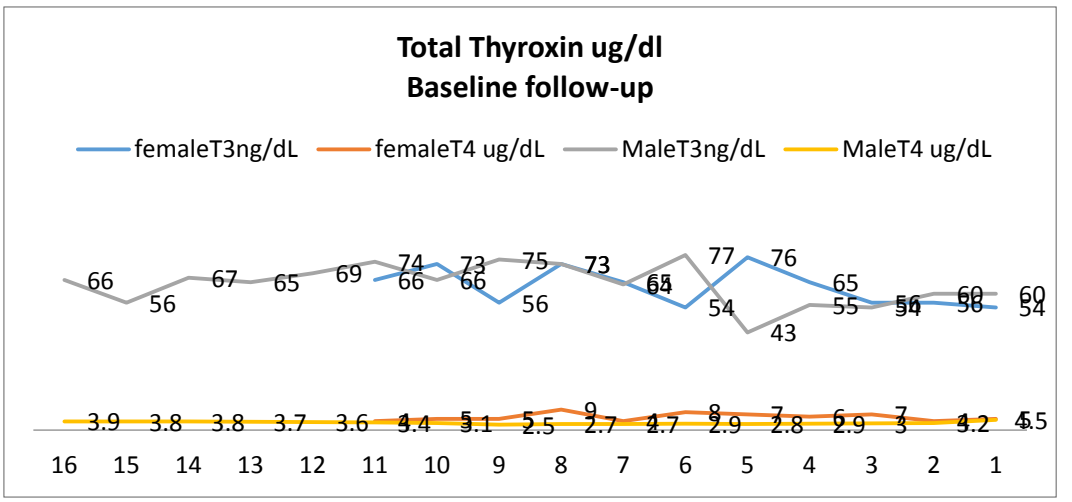


Chart (1): total thyroxin Ug/dl baseline follow-up

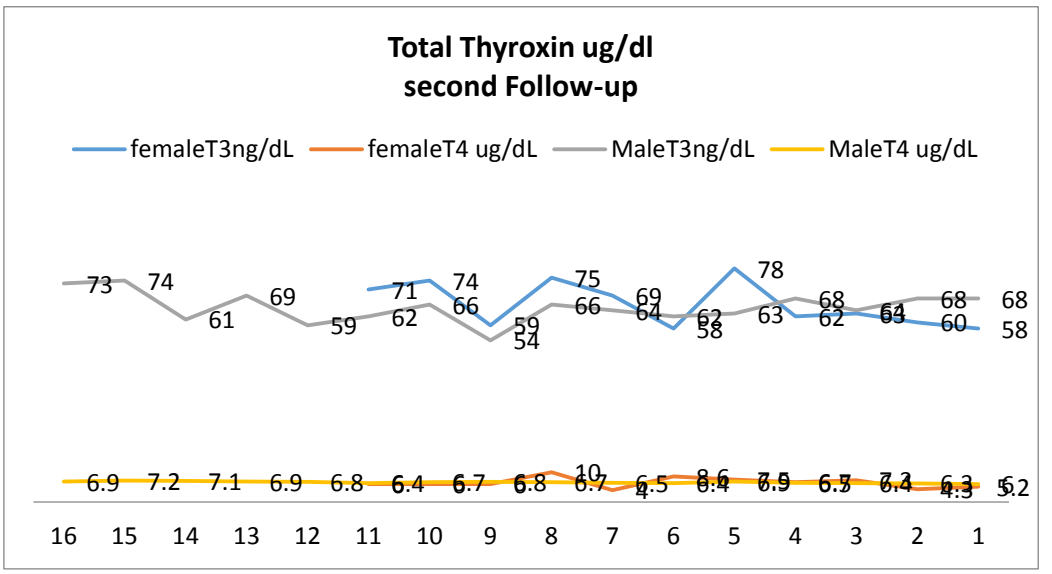


Chart (2): Total thyroxin Ug/dl second follow-up

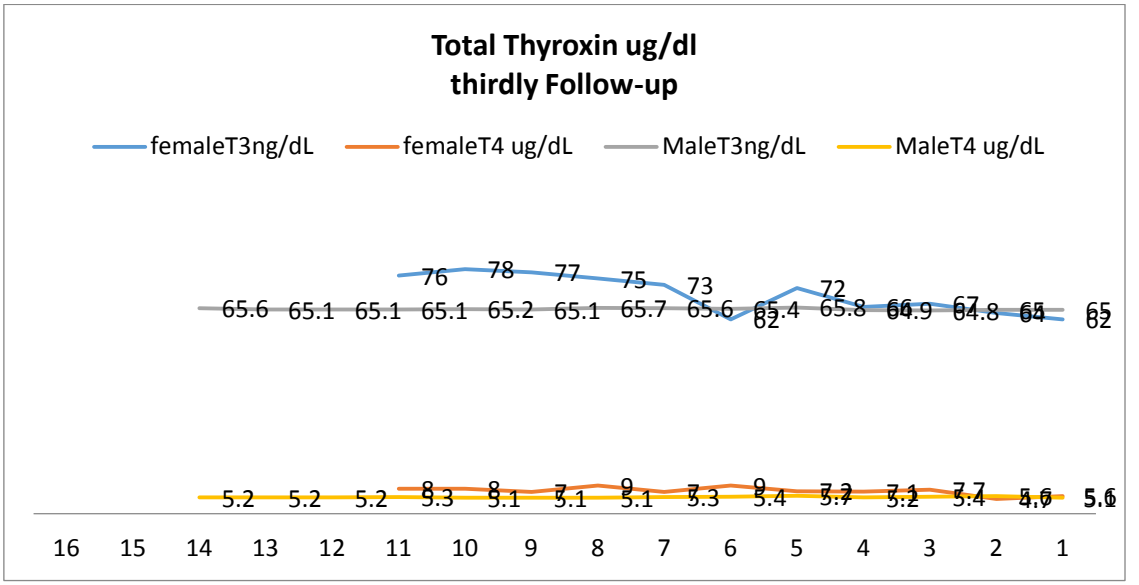


Chart (3): Total thyroxin Ug/dl thirdly follow-up

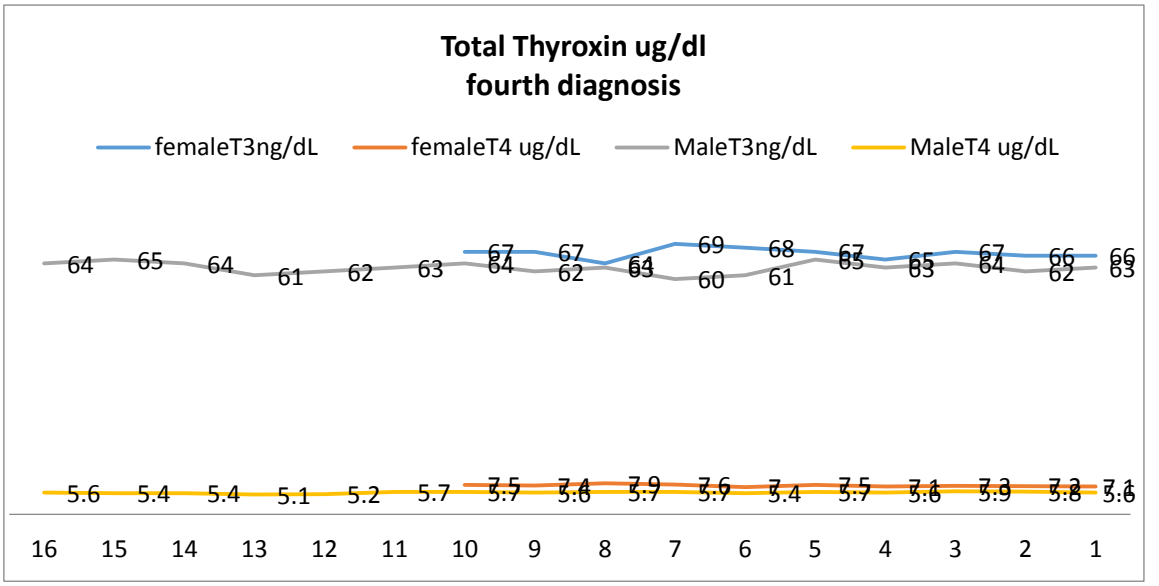


Chart (4): Total thyroxin Ug/dl fourth follow-up

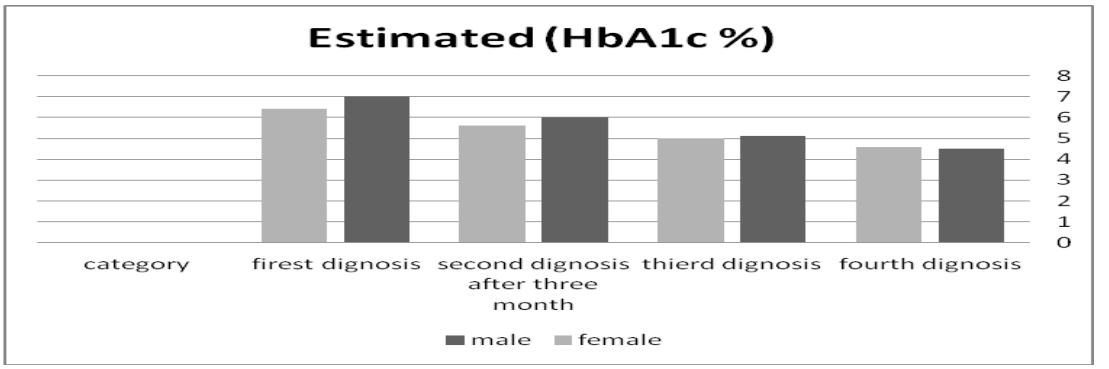


Figure (5): Estimated HbA1c refers to glaciated hemoglobin (A1c), as average plasma glucose concentration

Table (3): Estimated (HbA1c %) and BMI (kg/m2) for lipostatic (NAFLD) cases after nutritional intervention (Mean±SD)

Gender	category	second fallow-up	third fallow-up	fourth fallow-up
male	Hemoglobin A1c (HbA1c) %	6±0.30	5.1±0.5	4.5±0.60
female		5.6±0.4	5±0.9	4.6±0.2
male	BMI (kg/m2)	26.776±2.970	25.795±2.956	24.816±2.857
female		29.643±1.659	27.643±1.659	25.043±1.603

It can be argued that there should be systematic screening for NAFLD, at least among higher-risk individuals attending diabetes and obesity clinics. However, at present there are significant gaps in our knowledge regarding the diagnosis, natural history, and treatment of NAFLD. As liver biochemistries can be within normal ranges in patients with NAFLD and NASH, they may not be sufficiently sensitive to serve as screening tests, whereas liver ultrasound is potentially more sensitive but it is expensive and cumbersome as a screening test (Naga *et al.*,2012) A report published in 2009 by an International Expert Committee on the role of HbA1c in the diagnosis of diabetes recommended that HbA1c can be used to diagnose diabetes and that the diagnosis can be made if the HbA1c level is_6.5%(David, 2009). Diagnosis should be confirmed with a repeat HbA1c test, unless clinical symptoms and plasma glucose levels >11.1mmol/l (200 mg/dl) are present in which case further testing is not required. Levels of HbA1c just below 6.5% may indicate the presence of intermediate hyperglycemia. The precise lower cut-off p.int for this has yet to be defined, although the ADA has suggested 5.7 – 6.4% as the high risk range (Anon, 2010). While recognizing the continuum of risk that may be captured by the HbA1c assay, the International Expert Committee recommended that persons with a HbA1c level between 6.0 and 6.5% were at

particularly high risk and might be considered for diabetes prevention interventions. Several factors, including BMI, and diabetes, are known to affect an individual's susceptibility to NAFLD .BMI was inline border, values agree with (Perlemuter *et al.*, 2007 & Adams *et al.*, 200

Table (4): Case study related lipostatic (NAFLD) characteristic serum lipid profile values every three months under nutrition intervention only

Category	Baseline fallow-up clinical characteristic serum lipid						
	cholesterol mg/dl	Non-HDL-c	LDL-c	HDL-c	VLDL-c	TGr	AIP
Male				*n=16*			
Mean	241.50	169.05	144.90	72.45	24.15	120.75	0.01
±SD	12.04	8.43	7.23	3.61	1.20	6.02	0.00
females				*n=11*			
Mean	229	160.3	137.4	68.7	22.9	114.5	0.012
±SD	3.18	2.22	1.91	0.95	0.32	1.59	0.00
Category	Second fallow-up clinical characteristic serum lipid						
male				*n=16*			
Mean	226	158.2	135.6	67.8	22.6	113	0.012
±SD	5.983	4.188	3.590	1.795	0.598	2.992	0.000
females				*n=11*			
Mean	215	150.5	129	64.5	21.5	107.5	0.013
±SD	14.659	10.261	8.795	4.398	1.466	7.330	0.001
Category	Thirdly fallow-up clinical characteristic serum lipid						
male				*n=16*			
Mean	214	149.8	128.4	64.2	21.4	107	0.013
±SD	1.25	0.875	0.75	0.375	0.125	0.625	6.195
females				*n=11*			
Mean	210	147	126	63	21	105	0.013
±SD	9.199	6.439	5.519	2.760	0.920	4.599	0.001
Category	fourth fallow-up clinical characteristic serum lipid						
male				*n=16*			
Mean	201.5	141.05	120.9	60.45	20.15	100.75	0.014
±SD	1.628	1.140	0.977	0.488	0.163	0.814	0.000
females				*n=11*			
Mean	202	141.4	121.2	60.6	20.2	101	0.014
±SD	1.250	0.875	0.750	0.375	0.125	0.625	0.000

A net retention of lipids within hepatocytes, mostly in the form of triglycerides, is a prerequisite for the development of NASH. The primary metabolic abnormality leading to lipid accumulation (steatosis), however, is not well understood, but it could potentially result from insulin resistance and alterations in the uptake, synthesis, degradation or secretary pathways of hepatic lipid metabolism.(Marchesini *et al.*,1999 ;Angulo &Lindor 2001., and Sanyal *et al.*,2010).Also., Terry *et al.*,(2014) clear that classification of cholesterol and triglyceride Levels in mg/dL as flowing ;Non-HDL-C <130 desirable130–159 ab.ve desirable, 160–189 borderline high ,190–219 High, ≥220 Very high, LDL-C <100 Desirable 100–129 Ab.ve desirable, 130–159 Borderline high, 160–189 High, ≥190 Very high, HDL-C <40 (men) Low ,<50 (women) Low ,Triglycerides <150 ,Normal 150–199 ,Borderline high, 200–499 ,High ≥500 ,Very high. Result investigated and manifested enhancement for case study related lipostatic (NAFLD) characteristic serum lipid profile values every three months under nutrition intervention only. Cardiovascular disease (CVD) is the cause of one third of deaths worldwide and this will progress because of increasing CVD's risk factors. The most basic task of dealing with the epidemic of CVD is primary prevention of risk factors. As -Atherogenic Index of Plasma -(AIP) is a strong marker to predict the risk of atherosclerosis and coronary heart disease, assessed the correlation between AIP and other important factors (Niroumand *et al.*, 2015). Normal AIP was 0.41±0.23and 0.35±0.24 for males and females respectively, results revealed improvement agree with (Dobiasova & Frohlich, 2001; Dobiášová & Frohlich, 2001 and Tan MH *et al.*, 2004; Nwagha *et al.*, 2010 finally Mudhaffar,2015).

Conclusion

Results investigate enhancement special for BMI,AIP, T3, T4 finally lipid profile values after follow up case study for ninth months related lipostatic (NAFLD) characteristic values under nutrition intervention only. Study recommended decreased body weigh 10% from actual weight, fat 15% /total calories, α- tocopherol, Ginger not more 5gm divided/daily and coffee. Also restricted some food items as fructose, maize syrup, Grape, Date, Molasses, Honey bees, fried's and grilling.

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متابعة دراسة الحالة لتسعة اشهر للقيم المميزة لصفات التدهن الكبدى (مرض التدهن الكبدى غير الكحولى) تحت التدخل بالتغذية فقط

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الملخص : نظرًا لأن العديد من الباحثين يعتبرون الليبستاتيك (NAFLD) أحد المظاهر الكبديّة لمتلازمة التمثيل الغذائي ، والتي تُعرّف على أنها وجود 3 أو أكثر من الحالات التالية: السمنة ، ارتفاع دهون الدم ، انخفاض مستويات البروتين الدهني عالي الكثافة ، قيم غير طبيعية لهرمون الثيروكسين وارتفاع نسبة الجلوكوز صائم، لذلك ، تم إجراء العلامات المميزة لإجراء عملية Lipostatic (NAFLD) على مثل المسح بالموجات فوق الصوتية ، وبعض القياسات الأنثروبومترية ، والمقايس السيرولوجية لقيم T3 وT4 ، والدراسة المصلية لصورة الدهون ، ومؤشر تصلب الشرايين للبلازما (AIP) ، دراسة هيماتولوجية لسكر الدم الهيموجلوبينى التراكمي A1c٪ (HbA1c) .(أظهرت النتائج تحسنا في جميع المؤشرات الخاصة. أظهرت الموجات فوق الصوتية صورة طبيعية لجميع حالات الدراسة ، مثل البروتينات الدهنية للدم ، والبلازما) أيضا مؤشر كتلة الجسم ، قيم هرمونى (T3ng / dL، و T4ug / dL ، على التوالي). والخلاصة ، كشف تدخل التغذية فقط أن القيم المميزة الصحيحة تصل إلى وضعها الطبيعي فى الشهر التاسع بعد متابعة لكل ثلاثة أشهر. أوصت الدراسة بإنقاص وزن الجسم بنسبة 10٪ من الوزن الفعلي ، والدهون تحدد بنسبة 15٪ من إجمالي السعرات الحرارية ، α- توكوفيرول ، والزنجبيل لا يزيد عن 5 جرام يومياً و القهوة. كما قيدت بعض المواد الغذائية مثل الفركتوز ، شراب الذرة ، العنب ، البلح ، دبس العسل ، عسل النحل ، المشويات ، واخيرا المقليات.

كلمات البحث : *التدهن الكبدى(مرض التدهن الكبدى اللاكحولى) ، والتدخل الغذائى ، صورة دهون الدم ، مؤشر كتلة الجسم ، الثيروكسين،T3.T4 ، الهيموجلوبين التراكمي ، القياسات البشرية ، مؤشر تصلب الشرايين ، الموجات فوق الصوتية، ممنوعات مواد غذائيه، فركتوز، جنزبيل و الفاتوكوفيرول.*