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Weight Loss Program in Patients with Atherosclerosis: A randomised Controlled Clinical Trial

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Abstract - Atherosclerosis(AS) is one of the most common disease sleading tosevere clinical complications. Atherosclerosis forms pockets of fat and lipid deposit sunder the mucous tunic of blood vessels, which cause progressive vasoconstriction until total luminal blockage occurs. [1]Many "Civilization diseases" such as coronary artery disease (CAD), arterial hypertension (AH), diabetes mellitus (DM), psoriasis, gout, and such conditions as hyper lipidemia, hyperglycemia, dyslipidemia, hyperinsulinemia, hypercortisolemia, hyperuricemia, and microalbuminuria are connected with AS development. In these regards, the problem of developing a proven clinical solution for this prevalent disease process is a socially significant one.[1,2]

During the last 20 years, ASmorbidity has been globally increasing.[2,3] Mortality from AS complications, which account for 75-85% of general mortalityinwhole,[4] is simultaneously increasing.Thus AS is one of the global problems of conventional medicine.

Keywords : *atherosclerosis, fatty overweight, low-calorie, fat-free diet, weight loss therapy.*

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Weight Loss Program in Patients with Atherosclerosis: A randomised Controlled Clinical Trial

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List of Abbreviations

AH - arterial hypertension

AS - Atherosclerosis

BP - blood pressure

BMD - bone mass density

BMI - body mass index

CAD - coronary artery disease

CI - confidence interval

DBP - diastolic blood pressure

DM - diabetes mellitus

FOW - fatty overweight

IGT - impaired glucose tolerance

LPs - lipoproteins

M±SEM - mean ± standard error of the mean

OR - odds ratio

PICS – postinfarctioncardiosclerosis

SBP -systolic blood pressure

TGs – triglycerides

WCf - waist circumference

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Abstract -

Item	Description
Background	Mortality from atherosclerosis (AS) complications, which account for 75-85% of general mortality in whole, is simultaneously increasing. Recently published studies have revealed the close correlation between AS and overweight.
Objective	The purposes of the current study were to study the result of a weight loss program in AS patients using a randomized clinical design.
Design	A randomized controlled prospective clinical cross-sectional trial.
Methods and Participants	97 people were enrolled, among them 71 patients (34 females) with various manifestation of AS aged 46.4±3.6 years (patient group), and 26 healthy volunteers subjects (12 females) aged 48.5±2.2 years as a healthy control group. Statistical analysis was performed using SPSS for Windows version 17.0 (SPSS: An IBM Company, Armunk, NY).
Results:	With increased fat mass, impedance indicators and metabolic age were increased accordingly and significantly ($p<0.001$).The weight loss program reduced fat from26.75±2.94 kg to 15.76±2.98 kg, ($p=0.005$). Metabolic parameters including blood pressure, and glucose, cholesterol, and triglyceride serum levels improved significantly ($p<0.001$). Hemoglobin levels ($p<0.001$) and bone mass density ($p<0.001$) also increased significantly. Echocardiography demonstrated an increased ejection fraction from 56.3±1.1% to 72.1±1.3% ($p<0.0001$) and systolic output from 65.4±1.8 ml to 89.6±1.7 ml ($p<0.0001$) in weight loss AS

Item	Description
	patients.
Conclusions	Weight loss approach on a caloric restriction, low-fat vegetables and salt diet and includes a physical activity is a effective method of treating clinical AS manifestations. Endogenic atheromatouslipids are used during weight loss therapy, and it will reduce underlying AS processes.

Keywords : atherosclerosis, fatty overweight, low-calorie, fat-free diet, weight loss therapy.

I. Introduction

Atherosclerosis(AS) is one of the most common disease sleading to severe clinical complications. Atherosclerosis forms pockets of fat and lipid deposit sunder the mucous tunic of blood vessels, which cause progressive vasoconstriction until total luminal blockage occurs.[1]Many "Civilization diseases" such as coronary artery disease (CAD), arterial hypertension (AH), diabetes mellitus (DM), psoriasis, gout, and such conditions as hyper lipidemia, hyperglycemia, dyslipidemia, hyperinsulinemia, hypercortisolemia, hyperuricemia, and microalbuminuria are connected with AS development. In these regards, the problem of developing a proven clinical solution for this prevalent disease process is a socially significant one.[1,2]

During the last 20 years, ASmorbidity has been globally increasing.[2,3] Mortality from AS complications, which account for 75-85% of general mortality in whole,[4] is simultaneously increasing. Thus AS is one of the global problems of conventional medicine.

One of the scientific ways of working out of effective treatment strategies for AS requires the development of novel conceptions based on the accumulated scientific facts and knowledge in the field sof AS etiology and pathogenesis. For the last 20-30 years, several dozenhypotheses have been offered toexplainthe origin and progression of AS processes. However, to date, none of these has been universally recognized and proven in practice.[5,6]

Recently published studies have revealed the close correlation between AS and overweight.[7,8] The purposes of the current study were to study the result of a weight loss program in AS patients using a randomized clinical design.

II. Methods and Participants

a) Study Design

A randomized controlled prospective clinical trial.

b) Participants

We enrolled a total of 97 people, among them 71 patients (34 females) with various manifestation of A Saged 46.4±3.6 years (patient group), and 26 healthy volunteers subjects (12 females) aged 48.5±2.2 years as a healthy control group.

Thirty-one of the patients (16 females, aged 42-82 years, mean 54.6±2.8 years) with various AS manifestations were randomly recruited for the weight loss program. The clinical picture of the AS patients included: Leriche's disease (athero sclerosis of the coxo femoralartery) in 4 patients, CAD with anamnesis of the disease until 21 years of age was present in12 patients, CAD with postin farction cardiosclerosis (PICS) was present in 6 patients, cerebral stroke had occurred in 7patients,and Alzheimer's disease affected 2 patients. All 31 patients had AH, and of these 19 had DM and 12 had impaired glucose tolerance (IGT;Table 1) All enrolled AS patient shad abdominal lobesity.

The comparison group (control group) included 30 AS patients (aged 26-70 years, mean 47.5±1.9 years, including 14 females) who were receiving a conventional (traditional) drug therapy that included hypoglycemic (metformin 500-1500 mg per day, exenatide5-10 μg per day), lipid lowering (atorvastatin 40mg per day), antihypertensive (lisinopril 20mg per day, calcium channel blockers referring to benzodi-azepines 90mg per day), anti-inflammatory (acetylsalicylate acid up to 2 g per day and/or thienopyridine class anti platelet agent 75 mg per day)treatment, and symptomatic therapy. The study was carried out between October 2009 and April 2012 at the scientific research of cardiology and internal diseases (in Almaty, the Republic of Kazakhstan) and at the republican scientific center for emergency medicine (in Astana, the Republic of Kazakhstan).

Inclusion criteria included: written informed consentfor participation in the study; dyslipidemia (bloodserumhigh-density lipoprotein <1.0 mmol/L, or triglycerides(TGs) ≥1.7 mmol/L or cholesterol ≥5.6 mmol/L); waist circumference (WCf) in men >94.0 cm or in women >80.0 cm; overweight (body fat%>21);blood pressure (BP)>140mmHg of systolic blood pressure (SBP) and >95 mmHg of diastolic blood pressure (DBP), or ongoing treatment with antihypertensive drugs, fasting glucose >6.1 mmol/L or treatment with glucose-reducing drugs; absence of contraindications to weight loss; the possibility of treatment for 6 months and dynamic observation for 1 year.

c) Outcome Measures

The primary efficacy end point of the trial was full recovery from atherosclerotic diseases for 12 months. The secondary efficacy end point of the trial was data imaging methods (Doppler-ultrasound, computed tomography scans) and measurement of clinical presence status.

d) Randomisation

An independent statistician unconnected with clinical practice used computer generated random numbers (SPSS for Windows version 17.0: An IBM Company, Armunk, NY) to prepare randomisation lists. The block randomization was two (one on conventional drug therapy, one on weight loss therapy) with stratification by sex, age (47.81±2.3 and 45.7±2.26; p=0.199) and baseline body mass index (BMI) (30.15±1.38 and 29.35±1.36; p=0.34).

Methods we diagnosed prior CAD and PICS by case history and by electrocardio graphic changes of ischemia in patient anamnesis. We diagnosed AH by blood pressure readings and from medical records. Abdominal obesity was assessed WCF using the standards for the Asian nationality by the International Diabetes Federation.[9] the weight loss intervention study period was between 2-6 months in duration, depending on the individual patient clinical course, disease severity, and disease stage. Physical activity was assessed as the number of steps taken by patients, as determined by an individual pedometer from Hoffmann-La Roche, Ltd (Basel, Switzerland). Mental status was defined by the method the test of numbers binding by Reitano.[10]

The structure of lean and fatty mass before and after the weight loss program was determined with aTanita-SC330S Body Composition Analyzer (Tanita Corp., Tokyo, Japan). We defined anthropometrical indicators including age (years), weight (kg), BMI (kg/m^2). We also evaluated body composition parameters, including as fat mass (in % of total body weight and total kg), visceral fat rating (units), fat free mass (kg), total body water (in % and kg), muscle mass (in % and kg), bone mass (in % and kg), metabolic age (years), basal metabolic rate (kcal per day), and bioimpedance (Ohms).General clinical study of blood and urine chemistry, liver and kidneys function tests, and imaging methods (GE Vivid 7 Ultrasound ;GE Healthcare Worldwide USA, Michigan),bone densitometry (Lunar Achilles Express Ultrasound; GE Healthcare USA, Madison), and computed tomography scans(AG Siemens Soma tom Emotion 6, Germany, Muenchen) were performed.

We employed weight loss therapy using the weight loss program was conducted by administering a caloric restriction, fat-free vegetables and salt diet and includes an increased physical activity.[11-14] Participants were asked to reduce their meals to no more than 1200-1500 kcal per day. Behavioral weight loss intervention was achieved by a request to walk no less than 10,000 steps per day. Doses of previous symptomatic conventional drugs have been decreasing from the second/third days of the treatment beginning, and up to full withdrawn from the fifth/seventh days of the treatment beginning, as clinical symptoms were improved. A combination of in-person conversations and telephone calls were conducted during the 6-month study period. Weight loss results were assessed by WCf and body mass (kg).

Ethics .Ethical Committee of Scientific research institute of cardiology and internal diseases approved the study. Approval Number is the Protocol #9 from 06.02.2009.Board Affiliation: Health Ministry of the Republic of Kazakhstan Statistics. The two-sample Student's t-test and odds ratios (ORs) with 95% confidence intervals (CIs) were used. The study data are presented in Tables as mean ± standard error of the mean (M±SEM). The correlation analysis (r) and multinomial logistic regression model ORs with CIs were used. P-values of <0.05 was set as significant. Statistical analysis was performed using SPSS for Windows version 17.0 (SPSS: An IBM Company, Armunk, NY) and Microsoft Excel-2010 in updating Lapach, et al (2000).[15]

III. Results

We compared the patient and control groups with respect to metabolic age, basal metabolic rate, anthropometrical data, and body composition (Table 2).

As seen in Table 2, there were no significant differences between the patient group and the control group regarding passport age, weight, and height. The patient group had a significantly higher BMI on the order of an extra≈3.5 kg/m^2 compared with the healthy group. Fat mass was significantly higher in the patient group compared with the healthy group, on the order of≈12.1% or≈10.0 kg. Table 2 makes clear the inverse relationship between fat mass percentage and muscle masspercentage: The greater the fat mass percentage, the lower the muscle mass percentage.

The patient group also had significantly raised visceral fat rating (3.6U), metabolic age (≈6.6 years), basal metabolic rate (≈240 kcal/day), and bioimpedance (≈35 ohms) than did the control group. Increased body fat mass is associated with an accordingly increased impedance.

The healthy group displayed a significantly greater percentage of total body water on (≈8%), percentage of muscle mass (≈11.5%, and percentage of bone mass (≈0.3%) than the patient group. The regression linear analysis strongly correlated the relationships between fat mass and muscle/water/bone masses in the patients group (n=71) (Figure 1).

As seen in Figure 1, there are significant inverse regression correlations between fat mass in percentage and muscle mass, total body water, and bone mass ($p<0.0001$). This data analysis provides evidencethat fat mass could be a main risk factor for AS patients.

We studied the regression correlation between the levels of obesity (fat mass in % and visceral fat level

in units) and the metabolic age in the patient group (n=71) (Figure 2). As seen here, increased parameters of obesity (fat mass in % and visceral fat level in units) are significantly correlated with increased metabolic age ($p<0.0001$).

We began the weight loss program with the patient group (n=31) that had been randomly assigned into this category. As a result of the treatment, average weight lost varied from6 to 18kg. Weight loss led to positive changes of the cardiovascular diseases symptoms: SBP and DBP decreased in 94.4% of patients ($p<0.001$), more than 19% from the initial state (Table 3).

Table 3 shows that in the control group such parameters as SBP, glucose, cholesterol and triglyceride serum levels were improved significantly ($p<0.001$). However, the traditional treatment in AS patients did not lead to a significant improvement in DBP ($p=0.289$), blood hemoglobin levels ($p=0.281$), and bone mass density (BMD; $p=0.405$). It is noteworthy that in the main weight loss group we observed more significant ($p<0.001$) declines in SBP, DBP, glucose, cholesterol, and triglyceride levels. There was a significant increase in hemoglobin levels ($p<0.001$) and BMD ($p<0.001$).

Therapy by maintaining the weight loss program led to weight loss ranging between9-16 % from the initial state ($p=0.035$).Importantly, the weight loss achieved in these 31 patients was due to reduction of fatty mass only (before 26.75±2.94 kg, and after15.76±2.98 kg, $p=0.005$; Table 4).

The data of Table 4 show that the weight loss in AS patients was due to significant fat loss (before 29.87±2.01%, and after 20.23±2.55%). The percentages of water and muscle masses had tendency to increase at the study endpoint. We noted with fat mass loss does not change lean body mass. And the weight loss in the patients was due to loss from the overweight abdominal component.

During the first 2-3 days of the treatment, the patients noticed an intense sense of hunger, slight dizziness, weakness, lower extremity and abdominal muscle tremors, a sensation of heating or fever in the umbilical are a and/or in the solar plexus area, and psychogenic fear due to alt ered habitual food intake. All of these uncomfortable sensations disappeare don subsequent days.

Urine was becoming cloudier and intensely colored (dark) starting from Days 3-5 after initiating the weight loss treatment that was not present before, and persisted for several days. Microscopy of urocheras revealed that urine cloudiness was mainly been due to the organic salts such as oxalates, urates, phosphates, and carbonates of calcium and magnesium. An increase ederythrocyte sedimentation rate and elevated leukocyte count was observed between Days 4-6 after weight loss program initiation.

Regression of clinical AS symptoms was also gradually observed in patients; physical and mental working ability of the patients increased, and AS patients noticed a physical relief. Doppler-ultrasound imaging data revealed that blood flow in the lower extremities was restored and gastrocnemius musclemass increases were noted in four patients with Leriche's disease. The clinical picture of angina pectoris was reduced with an OR of 1.52 (95% CI 1.24–1.81; $p=0.034$), exercise tolerance was improved, and objective electrocar-diographyindicators of cardiovascular function were improved. Ejection fraction increased from 56.3 ±1.1% to 72.1 ±1.3% ($p<0.0001$) and the systolic output increased from 65.4 ±1.8 ml to 89.6 ±1.7 ml ($p<0.0001$) in CAD patients. All patients including those of with transient ischemic attack and with Alzheimer's disease noticed improvement of memory, and decreased mental fatigue with and OR of 1.47 (95% CI 1.18–1.77; $p=0.039$).

As clinical symptoms have been improving, the doses of previous symptomatic drugs were decreased from the second/third days of the treatment beginning and were withdrawn from the fifth/seventh days of the treatment beginning. The observation period was up to1 year and there was no recurrence of clinica lAS manifestations. Shed weight was usually not regained during this time period. Appropriate clinical AS symptoms occasionally and gradually relapsed in patients that regained at fatty overweight (FOW), but the weight lossre-therapy reversed these clinical symptoms.

IV. Discussion

Cardiovascular risk due to AS depends on visceral obesity developing over time.[16]Much attention has been paid to understanding the interactions of plasma lipoproteins (LPs), blood monocytes and the with arterial endothelium.[17] The genetic theory of AS development cannot be the complete underlying cause of AS disease, because corresponding parameters may be changed within the same persons, including inflammatory, immunologic, endothelial, metabolic and otherdisorders.[18-20]

The results of our study suggest that in the patient with AS compared with the healthy people a fat mass was significantly higher on the order of ≈12.1%. The patient group had significantly raised metabolic age (on≈6.6 years), basal metabolic rate (on≈240 kcal/day), and bioimpedance (on≈35 ohms) than did the healthy people. The regression linear analysis strongly significant inverse correlated the relationships between fat mass in percentage and muscle/water/bone masses in the body of the patients ($p<0.0001$). The more a percentage of fat mass the less percentage of

muscle/water/bone masses. Also increased parameters of obesity (fat mass in % and visceral fat level in units) are significantly correlated with increased metabolic age ($p<0.0001$). This data analysis provides evidencethat fat mass could be a main risk factor for AS patients.

The concept of insulin atherogeneity supports an independent atherogenic role for hyperinsulinemia.[21] In patients with simultaneously increased levels of very low density of LP sand TGs, high concentration of insulin were found after anoraltest for glucose tolerance.[22]

The nutriceptive-metabolic theory of AS has been developed recently. There exist substantial amounts of scientific data indicating an interrelation-between AS and visceral obesity.[23] Researchers[35] indicate that accumulation of visceral adiposity along with hyperinsulinemia and insulin resistance may be considered as fundamental signs of AS. One characteristic metabolic abnormality in insulin resistance is a high blood concentration of free fatty acids, both fasting and also postprandial. Permanently elevated free fatty acid concentrations in insulin resistance is a way to damage of vascular structures, endothelial function, and hinders insulin-mediated glucose metabolism.[24] Young persons with FOW have hyperinsulinemia, insulin resistance, and impaired fat toleranceeven on the background of normal glucose tolerance.[25] Intake of excess fats and carbohydrates leads to overload of the blood transport system and increased fatre serves. The fat deposit in organisms leads to aging of lipids.[26,27]

Exogenously-induced alimentary (postprandial) hyperlipidemia that develops after food intake is a suspected origin of AS.[28] Alimentary hyperlipidemia usually develops after each food intake and lasts for 6 hours or more.[29] A direct connection between the levels of postprandial lipidemia/hypertriglyceridemia and atherogenic changes after fatty overload manifested in angiographic coronary AS changes was revealed.[30] Alimentary hyperlipidemia is a physiological process whose primary function is transport of nutritional lipids into storage depots. However, if the depot is in the stage of "overstock," the duration of postprandial hyperlipidemia is increased and develops into an intolerance to nutrients.[31,32]

A treatment program focused on weight loss therapy using the weight loss program including a very low-calorie, fat-free vegetables and salt diet with an adjustment to eating behavior and increased physical activity method by metabolizing "old lipids".[12,14]The weight loss program leads losses of 9-16 % of the initial body mass ($p=0.035$). Weight loss led to positive changes of cardiovascular diseases symptoms ($p<0.001$), glucose/cholesterol/triglyceride levels. Also there was a significant increase in hemoglobin levels ($p<0.001$) and BMD ($p<0.001$).Our results are similar to database.[33,34]

Need to note, the weight loss was due to loss from the overweight abdominal component and reduction of fatty mass only whereas lean mass was not significantly changed. There also was a significant improvement of physical capability of individuals. During the first some days of the weight loss treatment in the patients noticed adverse effects connected with symptoms of endogenous metabolic intoxication. But all of these symptoms disappeared then.

The weight loss method lead to significant regression of clinical AS symptoms in observed patients. There was reduced an angina pectoris clinical picture ($p=0.034$), were improved objective electrocardiography indicators, an ejection fraction ($p<0.0001$), a systolic output ($p<0.0001$), an exercise tolerance and memory ($p<0.05$), decreased mental fatigue ($p=0.039$).

There was noted that with weight loss the disease clinical symptoms were improved and doses of previous symptomatic drugs were decreased from the second/third days of the treatment beginning, and up to full withdraw of its from the fifth/seventh days of the treatment beginning. If weight was not regained then clinical AS symptoms were not relapsed.

Growth of non-communicable diseases in the modern human is directly proportional with growth of the FOW prevalence.[35]Due to current super-nutrient status of humans, we now accumulate more fat than we can use.[36] The balanceis disturbed towards permanent lipid accumulation, wherein FOW become sun used by the body. But the process of fat accumulation does not occur infinitely, and fat in a storage depot may be qualitatively changed over time.[37,38]

The more FOW participates in an organism's metabolism, the more FOW creates the metabolic burden for proper functioning of thatorganism.[39] Trophic feeding, excretion of metabolic products, correction of thermoregulation, provision of biologically active substances (e.g., hormones, enzymes, and mediators) are all necessary for providing the vital activity of the fatty resources. All of these mechanisms create an additional burden to the synthetic, immunological, antitoxic, and other functions of an organism's internal orgas.[40]

Obviously, the depositing function of an organism is not without limits. With increased fatty stores, the metabolic burden for transport function of and organism increases accordingly.[41]

Accumulation of FOW in an organism requires appropriate spaces for deposition. The process of fat deposition occurs by the physicochemical "in duration" (hardening) of lipids.[42] This lipid in duration is a process that occursin AS development.[43] The AS plaque

development process is not a random phenomenon, but it is a logical result of aging of fatty stores. Lipid aging occurs because they have remained unused by the body over a long period of time. The protective homeostatic mechanisms could be body temperature, blood pressure, or insulin resistance, which increase the rate of biochemical metabolism.[44,45]Consequently, the AS processes could be considered physiological, in which an organism exists in conditions of excess nutrient intake on the background of unused FOW.

Isolated struggles with clinical expressions of AS lesions such as CAD, AH, DM, hyperglycemia, microalbuminuria, and hyperlipidemia did not influence the risk of cardiovascular disaster and decrease mortality, though it did improve patient quality oflife.[46]Improvement of therapeutic and diagnostic methods has not lead to definitive cures of human chronic non-communicable diseases.[47,48]

V. Conclusion

We observed that the more fat mass is stored in organism the less a muscle, bone and water masses are present. Increased fat mass in an organism is an impedance indicator, and impedance and metabolic age are significantly increased, accordingly.

Weight loss therapy using the weight loss program including a caloric restriction, fat-free vegetables and salt diet with an increased physical activity is a performance method of curing the clinical manifestations of AS. The weight loss treatment led to positive changes of diseases symptoms, improve in laboratory and instrumental parameters in patients with AS. The weight loss programs lead to loss of adipose tissue only. If the "old" lipids are used during weight loss-mediated FOW reduction it will be possible to influence AS processes, as desired.

Study limitation. Published studies into the possible role of fatty overweight in AS etiology are limited in scope and number. We acknowledge that the current randomized clinical trial had a small sample size, and that future studies into the exact relationship between fatty overweight and AS development should be carried out on a larger scale.

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Authors' Contributions

Kuat P. Oshakbayev: design and performance, scientific executor, collect of the clinical material, treatment and diagnosis of the patients, invention patent fulfillment, bibliography review, scientific analysis, statistical advancing, writing the paper.

Kenneth Alibek: design and performance, scientific executor, invention patent fulfillment, paper review, writing the paper.

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Table 1 : Demographic details of the study patients (n=31) who were recruited for the weight loss program

Groups	Gender	Number
Patients with Leriche's disease	M	3
	W	1
Patients with CAD	M	5
	W	7
Patients with CAD+PICS	M	3
	W	3
Patients with cerebral stroke	M	3
	W	4
Patients with Alzheimer's disease	M	1
	W	1
Patients with AH	M	15
	W	16
Patients with DM	M	10
	W	9
Patients with IGT	M	5
	W	7
Total	M	15
	W	16

Abbreviations: M, men; W, women; CAD, coronary artery disease; AH, arterial hyper tension; PICS, postinfarction cardiosclerosis; DM, diabetes mellitus; IGT, impaired glucose tolerance.

Table 2 : Anthropometrical data, metabolic data, body composition in study groups

Parameters	Patientgroup(n=71)		Healthy group(n=26)		t-test	P-value
	M	SEM	M	SEM		
Passport age (years)	46.45	2.29	48.55	3.69	0.484	0.315
Weight (kg)	83.90	2.70	75.61	4.45	1.687	0.057
Height (cm)	167.93	1.12	169.27	1.99	0.587	0.279
BMI (kg/m^2)	29.75	0.78	26.22	1.37	2.239	0.014
Fat mass (%)	32.40	1.28	20.27	2.67	4.097	0.00004
Fat mass (kg)	28.34	1.61	17.64	3.18	3.002	0.0017
Visceral fat rating (Unit)	10.04	0.73	6.40	0.70	3.599	0.0003
Fat free mass (kg)	54.80	1.53	58.37	2.04	1.400	0.08
Total body water (kg)	40.91	1.16	41.22	1.63	0.155	0.44
Total body water (%)	48.70	0.80	56.18	2.01	3.458	0.0004
Muscle mass (kg)	52.05	1.46	55.39	1.96	1.367	0.087
Muscle mass (%)	64.19	1.21	75.74	2.56	4.079	0.00005
Bone mass (kg)	3.16	0.07	3.01	0.10	1.229	0.111
Bone mass (%)	3.77	0.09	4.01	0.10	1.784	0.039
Metabolic age (years)	49.00	1.73	42.35	2.60	2.129	0.018
Basal metabolic rate (kcal/day)	1661.6	46.45	1419.8	52.77	3.439	0.0004
Bioimpedance (ohms)	502.10	10.30	467.42	9.11	2.522	0.0067

Abbreviations: BMI, body mass index; M, mean; SEM, standard error of the mean

Table 3 : Blood pressure, blood hemoglobin, serum glucose and lipid levels, and bone mineral density before and after completion of the weight loss program (n=31) and the control group who received traditional therapy (n=30)

Study Groups		SBP, mmHg	DBP, mmHg	Hemoglobin gram/L	Glucose, mmol/L	Cholesterol, mmol/L	Triglycerol, gram/L	BMD, Units
Control group, n=30	before treatment	149.4 ±3.4	94.8 ±2.2	132.1 ±2.2	6.40 ±0.49	5.60 ±0.1	2.15 ±0.05	74.0 ±2.9
	after treatment	137.6 ±5.1	93.1 ±2.1	134.0 ±2.4	5.27 ±0.37	5.24 ±0.16	1.92 ±0.09	73.2 ±1.6
Weight loss patient group, n=31	before treatment	150.1 ±3.9	99.3 ±2.9	129.5 ±2.67	6.42 ±0.46	5.73 ±0.13	2.31 ±0.1	71.6 ±2.79
	after treatment	121.8 ±2.1	81.6 ±1.8	140.3 ±1.6	4.37 ±0.38	4.26 ±0.15	1.62 ±0.09	97.9 ±2.8
P before and after treatment in controls =		0.03	0.289	0.281	0.036	0.031	0.015	0.405
P before and after treatment in main group		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

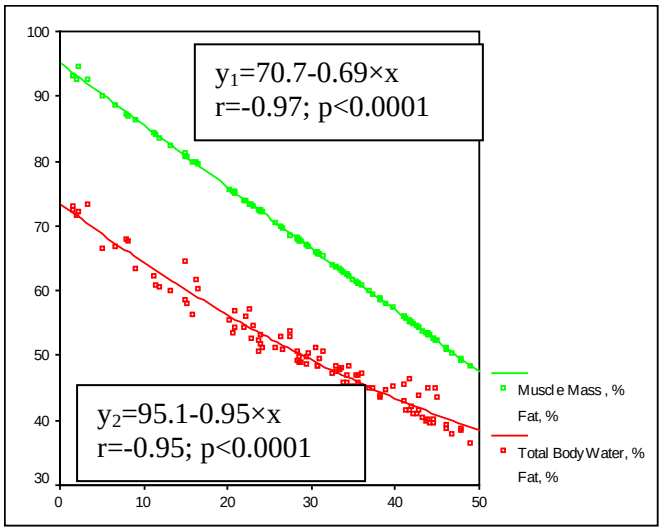
Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; BMD, bone mass density. Data are presented as means ± SEM

Table 4 : Before and after weight loss therapy in the AS patient group (n=31)

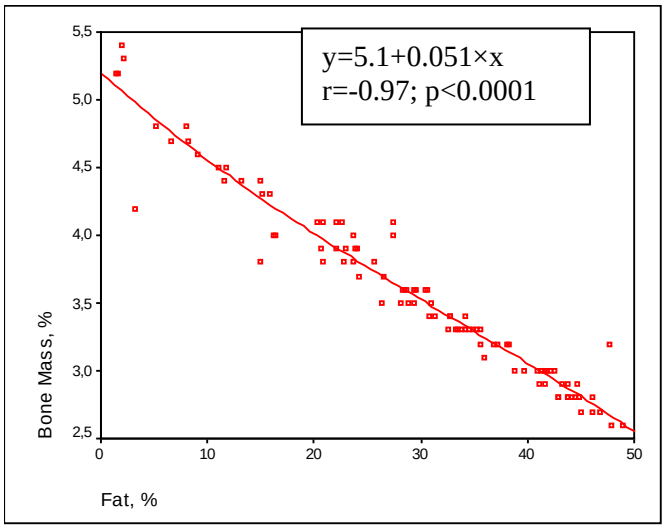
Parameters	Before the weight loss therapy (n=31)		After the weight loss therapy (n=31)		t-test	P-value
	M	SEM	M	SEM		
Passport age (years)	48.11	2.10	-	-	-	-
Weight (kg)	89.56	4.59	77.91	4.39	1.834	0.035
BMI (kg/m^2)	30.15	1.38	26.23	1.30	2.068	0.021
Fat mass (%)	29.87	2.01	20.23	2.55	2.969	0.0019
Fat mass (kg)	26.75	2.94	15.76	2.98	2.625	0.005
Visceral fat rating (units)	11.73	1.26	8.02	1.64	1.794	0.038
Fat free mass (kg)	62.81	2.18	60.82	2.01	0.671	0.25
Total body water (kg)	43.98	1.69	43.11	1.58	0.376	0.35
Total body water (%)	49.11	1.41	55.33	1.99	2.553	0.0061
Muscle mass (kg)	58.56	2.11	57.55	1.91	0.355	0.36
Muscle mass (%)	65.39	1.87	73.87	2.49	2.723	0.0038

Bone mass (kg)	3.26	0.12	3.05	0.10	1.344	0.091
Bone mass (%)	3.64	0.09	3.91	0.08	2.282	0.012
Metabolic age (years)	56.82	3.89	47.78	3.67	1.669	0.047
Basal metabolic rate (kcal/day)	1837.53	67.42	1495.44	64.01	3.680	0.0002
Bioimpedance (ohms)	505.00	10.22	477.54	8.95	2.021	0.023

Abbreviations: BMI, body mass index; M, mean; SEM, standard error of the mean

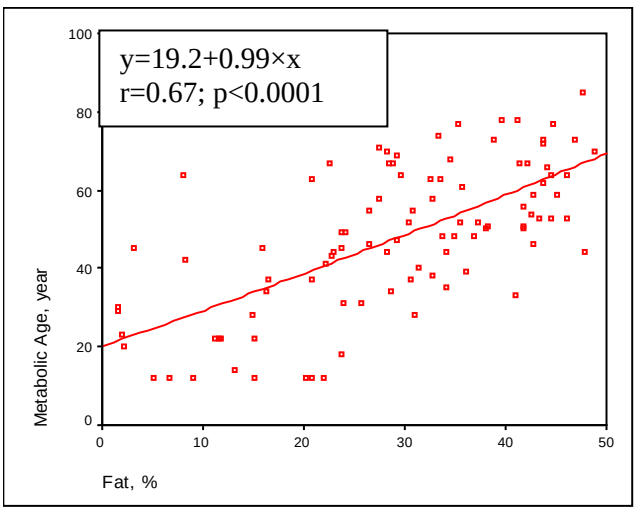


A)Note : x=fat mass (in %); y_1=muscle mass (in %); y_2=total body water (in %);(n=71)

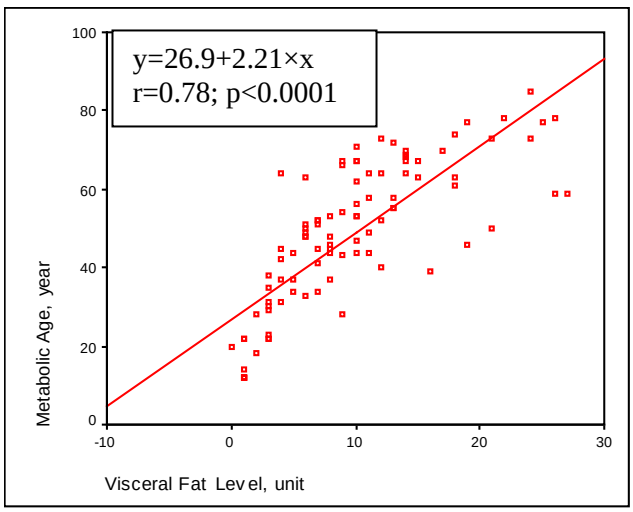


B)Note : x=fat mass (in %); y=bone mass (in %); (n=71)

Figure 1 : The regression correlation between fat mass (in %)and: A)muscle mass (in %), total body water (in %); and B)bone mass (in %) in the patients group (n=71)



A) Note: x=fat mass (in %); y=metabolic age (years); (n=71)



B) Note: x=visceral fat level (units); y=metabolic age (years); (n=71)

Figure 2 : Correlation between metabolic age (years) and: **A)**fat mass (in %); and **B)** visceral fat level (units)in the patients group (n=71)