OBES SURG (2009) 19:1664–1671
DOI 10.1007/s11695-009-9891-x

RESEARCH

Noninvasive Evaluation of Abdominal Fat and Liver Changes Following Progressive Weight Loss in Severely Obese Patients Treated with Laparoscopic Gastric Bypass

Federica del Genio · Gianmattia del Genio · Ilario De Sio · Maurizio Marra · Lucia Alfonsi · Carmine Finelli · Franco Contaldo · Fabrizio Pasanisi

Received: 3 November 2008 / Accepted: 28 May 2009 / Published online: 13 June 2009

Abstract
Background Obesity is a chronic complex disease, consequence of an unbalance between energy intake and expenditure and of the interaction between predisposing genotype and facilitating environmental factors. The aim of the study was to evaluate body composition, abdominal fat, and metabolic changes in a group of severely obese patients before and after laparoscopic gastric bypass (LGBP) at standardized (10% and 25%) total weight loss.
Methods Twenty-eight patients (14 M, 14 F; age 41.71± 6.9 years; body mass index (BMI) 49.76±5.8 kg/m^2) were treated with laparoscopic gastric bypass. All evaluations were performed before surgery and after achieving ~10% and ~25% weight loss (WL). Body composition was assessed by bioimpedance analysis; resting metabolic rate (RMR) was measured by indirect calorimetry.
Results Body weight, BMI, and waist circumference significantly decreased at 10% and 25% WL. We observed a significant reduction of both RMR (2,492±388 at entry vs. 2,098 ± 346.6 at 10% WL vs. 2,035±312 kcal per 24 h at 25% WL, p=0.001 vs. baseline) as well as of RMR corrected for fat-free mass (FFM; 35.7±6.7 vs. 34.9±9.0 at 10% WL vs. 33.5±5.4 at 25% WL kilocalorie per kilogram FFM×24 h, p=0.041 vs. baseline). Body composition analysis showed a relative increase in FFM and a reduction of fat mass at 25% WL. A significant reduction in blood glucose, insulin, homeostasis model assessment index was observed. Ultrasonography showed a marked decrease in the signs of hepatic steatosis.
Conclusion In conclusion, our study confirms that LGBP is a safe procedure in well-selected severely obese patients and has early favorable effects on both metabolic parameters and body composition. Longer-term observations are required for in-depth evaluation of body composition changes.

Keywords Severe obesity · Laparoscopic gastric bypass · Ultrasound · Abdominal fat · Weight loss

F. del Genio (✉) · M. Marra · L. Alfonsi · C. Finelli · F. Contaldo · F. Pasanisi
Department of Clinical and Experimental Medicine,
Interuniversity Center for Obesity and Eating Disorder (CISRO),
Federico II School of Medicine,
Via Pansini 5,
80131 Naples, Italy
e-mail: federicadelgenio@tin.it

G. del Genio
1st Department of General and Gastroenterologic Surgery,
Second University of Naples,
Naples, Italy

I. De Sio
Department of Clinical and Experimental Medicine
"F. Magrassi e A. Lanzara", Second University of Naples,
Naples, Italy

Introduction

Obesity is a chronic complex disease, consequence of an unbalance between energy intake and expenditure and of the interaction between predisposing genotype and facilitating environmental factors, mainly sedentarism and the so-called transition diet of affluent societies. The excess energy is stored as triglycerides into fat cells but also into other cells as hepatocytes, myocytes, perivascular tissue, etc.

Adipose tissue, now considered as an endocrine/autocrine organ, when enlarged, leads to an increase in

endoplasmic reticulum and in the number of macrophages and other stromal cells, which may contribute to the abnormal secretion of many peptides involved in several regulatory functions. Furthermore, increased secretion of free fatty acids from enlarged adipocytes, together with abnormal peptides secretion, contributes to the development of many typical complications of obesity: type 2 diabetes, hypertension, hyperlipidemia, nonalcoholic fatty liver disease (NAFLD), and some cancers [1, 2]. On the other hand, complications of obesity are also mechanical, being a direct consequence of the increased weight of the fat mass (FM) as in the case of osteoarthritis, hypoventilation syndrome, some physical disabilities, etc.

Nowadays, the rising prevalence and severity of obesity may offset the arrays of positive influence on longevity, typical of developed and affluent societies, and mostly attributable to lower mortality rates among individuals above 50 years of age [3]. Indeed, increased abdominal adiposity associated with a sedentary lifestyle may significantly affect mortality [4]. Furthermore, sudden death prevalence, mostly due to cardiovascular events, is extremely high in severely obese patients, even at a young age [5].

NAFLD comprises the liver abnormalities associated with obesity, in the absence of other pathogenetic factors, and is represented by hepatomegaly, increased liver enzymes, steatosis with a more complicated pattern up to nonalcoholic steatohepatitis (NASH), fibrosis, and cirrhosis [6]. NAFLD usually is reversible after weight loss; vice versa, NASH has a poorer prognosis and may worsen to cirrhosis and cancer.

Increased steatosis in obesity is also considered a clinical consequence of the frequent association with the metabolic syndrome (MS) and may reflect the increased free fatty acid flux to the liver associated with hyperinsulinemia, to increased—albeit insufficient—very-low-density lipoprotein (VLDL) production, and to increased secretion of proinflammatory cytokines due to excess adipose tissue.

The prevalence of liver abnormalities in obesity is quite high, particularly in severely obese patients as reported in various studies also carried out in our department [7, 8].

Cholelithiasis is another frequent, often asymptomatic, finding in obesity especially in obese premenopausal women [9].

Despite the expected positive effects of weight loss on NAFLD, there are limited data evaluating body composition and liver improvements following laparoscopic gastric bypass (LGBP) in severely obese patients.

The aim of the study was to evaluate body composition, abdominal fat, and metabolic changes in a group of severely obese patients before and after LGBP at standardized (10% and 25%) total weight loss.

Patients and Methods

Twenty-eight adult outpatients attending the Obesity Clinic of the Federico II University Hospital in Naples were enrolled in the study.

All patients (14 males, 14 females; age 41.71±6.9 years; body mass index (BMI) 49.76 ± 5.8 kg/m^2) underwent surgery and were treated with laparoscopic gastric bypass. Patients were recruited, balancing for sex, among those regularly attending the clinic. Patients underwent all evaluations before surgery and after achieving ~10% and ~25% weight loss (WL).

At entry, all patients underwent a complete clinical assessment to evaluate the presence and severity of associated medical conditions.

Dietary history, food habits, and previous weight loss efforts were accurately recorded.

To be eligible for the study, patients had to meet the following criteria: (a) no history of current or past significant alcohol abuse, i.e., more than 20 g alcohol per day; (b) no current use of psychotropic drugs or other medications potentially affecting liver function; (c) serum transaminases not higher than twice normal value.

Anthropometry

Initial assessment included anthropometric measurements with standard procedures: height was measured to the nearest 0.1 cm with a stadiometer and body weight to the nearest 0.1 kg on a balance beam scale with the subject barefoot and wearing only light undergarment; height and weight were recorded and BMI (kg/m^2) was calculated; waist circumference was assessed with a tape measure at the iliac crests.

Hemato-biochemical Examination

All patients underwent a routine hemato-biochemical examination including: total and high-density lipoprotein (HDL) cholesterol, triglyceride (TG), glucose and insulin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), alkaline phosphatase, total bilirubin, hemoglobin, total red blood cell count, ferritin, transferrin, and fibrinogen. All analytes were detected using routine laboratory methods.

Metabolic Syndrome Diagnosis

Blood pressure was measured on the right arm, after the patient had been seated for at least 5 min; a standard sphygmomanometer, with an adequately sized cuff, was used. The diagnosis of high blood pressure was made when values were >130/85 mmHg. Hypercholesterolemia, hyper-

triglyceridemia, and low-HDL cholesterolemia were diagnosed when values were over 200 and 150 mg/dl and below 40 mg/dl in men and <50 mg/dl in women, respectively. These cutoff points were chosen according to the Adult Treatment Panel III (ATP III) and the American Heart Association (AHA) criteria to identify the metabolic syndrome. The cutoff points for waist circumference were 102 cm in men and 88 cm in women according to ATP III and 94 cm in men and 82 cm in women according to AHA, respectively.

The cutoff points for glycemia were ≥110 mg/dl according to ATP III and ≥100 mg/dl according to AHA, respectively. The prevalence of metabolic syndrome was determined considering the combination of three out of five risk factors as described elsewhere [10].

The homeostasis model assessment (HOMA), based on serum fasting glucose (mmol/l)×insulin levels (μU/l)/22.5, was also used as an index of insulin resistance. The MS was evaluated using both ATP III and AHA criteria.

Fatty Liver Index

The risk of hepatic steatosis was evaluated with the fatty liver index (FLI), an algorithm based on BMI, waist circumference, triglycerides, and GGT, as previously described by other authors [11] and already used by our group [12].

Single-Frequency Bioimpedance Analysis

Body composition was assessed by bioimpedance analysis (BIA). Single-frequency BIA was carried out by the same operator using a BIA 101 device (injection of an alternating current, at 800 μA and 50 kHz; RJL/Akern System, Florence, Italy). Measurements were performed on the nondominant side of the body, at an ambient temperature of 22–24°C, after voiding and after being in the supine position for 20 min. A standard tetrapolar technique was used, placing the measuring electrodes on the anterior surface of the wrist and ankle and the injecting electrodes on the dorsal surface of the hand and foot, respectively. The BIA variables considered were resistance (*R*), reactance (Xc), and phase angle (PhA). The bioimpedance index (BI) was calculated as the ratio height-squared/resistance ($cm^2\ \Omega^{-1}$). The instrument was routinely checked with resistors and capacitors of known values.

Indirect Calorimetry

RMR was measured by indirect calorimetry using a canopy system (V max 29 N, Sensor Medics, Anaheim, USA) in a quiet environment and with patients in the supine position for 30 min before measurement. After allowing patients to adapt to the instrument for 15–20 min, oxygen consumption and carbon dioxide production were determined for 45 min. Energy expenditure was derived from CO_2 production and O_2 consumption, with the appropriate Weir formula, neglecting protein oxidation. The apparatus was calibrated with gas mixtures of known composition before each test and regularly checked by burning ethanol.

Ultrasonography

Liver ultrasonography scanning was performed and read by two independent operators unaware of the patients' laboratory data. An Esaote Mpx Biomedica apparatus equipped with a convex 3.5-MHz probe and a linear high-frequency probe (7–13 MHz) was used. Liver steatosis was assessed semiquantitatively on a scale ranging from 0 to 3 (0: absent; 1: mild; 2: moderate; 3: severe). Steatosis was graded according to Saverymuttu et al. [13] on the basis of abnormally intense and high-level echoes from the hepatic parenchyma, a liver–kidney difference in echo amplitude, echo penetration into the deep portion of the liver, and clarity of the liver blood vessel structure [14, 15].

Visceral fat (VF) *thickness* is the first sonographic index used for evaluation of visceral adiposity. It was assessed measuring the distance between the anterior wall of the aorta and the posterior surface of the rectus abdominis muscle, with a 3.5-MHz probe [16–18], 1–5 cm above the umbilicus at the xiphoumbilical line or midway between the xiphoid process and the umbilicus [19]. The probe was moved lightly to avoid distortion of tissue due to excess compression and weight of the probe. The thickness of subcutaneous fat (SF) was measured at the same level above the umbilicus, using the high-frequency linear probe directly on the screen with electronic calipers.

Abdominal Fat Thickness

This value was calculated as the sum of visceral fat thickness and subcutaneous fat thickness both expressed in centimeters and measured, as mentioned above, with the abdominal ultrasound.

Visceral and Subcutaneous Fat Thickness Ratio

This ratio was calculated as visceral fat thickness in centimeters divided by subcutaneous fat thickness, both measured, as mentioned above, with the abdominal ultrasound. [20]

Patients' Evaluation and Follow-up

After hospital discharge, all patients were followed weekly by the doctor and the dietician for evaluation of symptoms

and careful dietary monitoring and supplementation. One month after surgery, all patients were invited to change their lifestyles and were encouraged to start mild physical activity sessions; in particular, walking up to 30 min a day was recommended.

Before surgery, all patients underwent psychiatric consultation. This assessment included personal and relational history, history of psychiatric problems, and current living state. None of the participants had any evidence of psychiatric diseases.

All patients were studied at entry and after achieving 10% and 25% weight loss after laparoscopic Roux-en-Y gastric bypass.

Operative Technique The laparoscopic procedure began with the hemi-omentectomy to facilitate the antecolic transposition of the Roux limb. The height of the gastroesophageal junction was localized by the transillumination of the endoscope and the gastric pouch was realized 6 cm caudally and completed toward the angle of His alongside a 38-F bougie. The gastrojejunostomy was fashioned manually. The Roux (alimentary) and the biliopancreatic limb lengths were 150 and 50 cm, respectively. The anastomosis was checked intraoperatively by an endoscopic hydropneumatic test and, on postoperative day 5, by water-soluble contrast Rx swallow before starting oral feeding.

Statistical Analysis

The statistical analysis was performed using the SPSS software (14.0 rel.). Results are expressed as mean± standard deviation. Comparisons were performed using unpaired *t* test and one-way analysis of variance.

The null hypothesis was rejected at a two-tailed $p \leq 0.05$. X^2 was used for comparison of categorical data.

Results

Twenty-eight severely obese adult patients (age 41.71± 6.9; range 25–53 years) underwent laparoscopic gastric bypass. Two of them with cholelithiasis underwent a cholecystectomy during bariatric procedure. The 10% WL (true average value being 12.0±2.5%) was achieved on average after 6 weeks and the 25% WL (true average value being 24.7±4.2%) about 30 weeks after LGBP. At entry, five patients had type 2 diabetes treated with diet and/or hypoglycemic agents; 11 patients took antihypertensive drugs; one patient had hypolipidemic tablets; at 25% WL, all diabetic patients stopped hypoglycemic agents; only five patients continued antihypertensive drugs at lower doses; none required lipid-lowering agents.

Body weight (137.6±23.7 at entry vs. 120.4±20.6 at 10% WL, and vs. 103.9±19.6 kg at 25% WL; $p=0.001$), BMI (49.8±5.8 vs. 43.8±5.0 vs. 37.4±4.5 kg/m^2; $p=0.001$), and waist circumference (141.3±16.1 vs. 132.5± 14.6 vs. 117.9±13.1 cm; $p=0.001$) significantly decreased at 10% and 25% WL.

We observed a significant reduction of both RMR, in absolute values (2,492±388 at entry vs. 2,098±346.6 at 10% WL vs. 2,035±312 kcal per 24 h at 25% WL, $p=0.001$ vs. baseline) as well as of RMR corrected for FFM (35.7±6.7 vs. 34.9±9.0 at 10% WL vs. 33.5±5.4 at 25% WL kilocalorie per kilogram FFM×24 h, $p=0.041$ vs. baseline). Body composition analysis with BIA showed a relative increase in FFM (52.2±6.2 vs. 51.5±7.4 at 10% WL vs. 64.1±8.9% at 25% WL; $p=0.001$ vs. baseline) and a concomitant reduction of FM (47.8±6.2 vs. 48.5±7.4% at 10% WL vs. 34.9±9.0 at 25% WL; $p=0.001$ vs. baseline) at 25% WL (Table 1). Concerning other BIA parameters: BI was significantly reduced at 10% WL compared to baseline (73.1±13.9 vs 65.7±13.1 at 10% $p<0.05$, vs. 69.3± 16.6 cm^2/Ω at 25% WL) whereas PhA changes did not

Table 1 Anthropometric data, body composition, and resting metabolic rate at entry and following 10% and 25% WL in 28 (14 males, 14 females) severely obese patients

	Baseline Mean±SD	~10% WL Mean±SD	~25% WL Mean±SD
Body weight (kg)	137.6±23.7	120.4±20.6*	103.9±19.6**, ***
BMI (kg/m^2)	49.8±5.8	43.8±5.0**	37.4±4.5**, ****
Waist circumference (cm)	141.3±16.1	132.5±14.6	117.9±13.1**, ****
FFM (%)	52.2±6.2	51.5±7.4	64.1±8.9**, ****
FM (%)	47.8±6.2	48.5±7.4	35.9±8.9**, ****
RMR (kcal/24 h)	2,492±388	2,098±346.6	2,035±312.9**
RMRc (kcal/kg FFM×24 h)	35.7±6.7	34.9±9.0	33.5±5.4*

RMR resting metabolic rate, *RMRc* corrected for fat-free mass

*$p=0.05$ vs. baseline; **$p=0.01$ vs. baseline; ***$p=0.05$ vs. 10% WL; ****$p=0.01$ vs. 10% WL

reach statistical significance (7.2±0.8 vs. 7.0±1.1 at 10% WL and 6.6±0.9° at 25% WL, ns).

A significant reduction in blood glucose (104.8±28.2 vs. 87.6±10.9 vs. 78.4±8.0 mg/dl; p=0.001), insulin (24.2± 12.3 vs. 11.1±6.4 vs. 8.5±4.3 μg/U; p=0.001), and HOMA index (from 6.4±3.8 at baseline to 2.4±1.4 at 10% WL, p=0.001, and to 1.6±0.9 at 25% WL, p=0.001 vs. baseline) was observed. Serum cholesterol levels decreased in the first 6 weeks (198.0±30.1 vs. 170.9±38.7 mg/dl, p=0.012) and then remained unchanged at 25% WL (170.9±38.7 vs. 176.4±33.6 mg/dl, p=ns). Triglycerides (166.5±99.3 vs. 131.2±51.4 vs. 101.9±38.6 mg/dl; p=0.002) showed a rapid and significant reduction, while HDL cholesterol slightly decreased at 10% WL (46.5±12.7 vs. 41.9±11.1 mg/dl, p=ns) and significantly increased at 25% WL versus 10% WL evaluation (41.9±11.1 vs. 51.0±11.1 mg/dl, p=0.017).

Blood pressure values (131.1/85.3±10.5/8.3 vs. 126.9/ 82.9±16.9/8.0 at 10% WL vs. 120.7/79.3±13.8/10.0 mmHg at 25% WL, p=0.017/0.035) and heart rate (79.6±8.3 vs. 74.7±10.7 vs. 71.7±9.6 beats per minute, respectively, p=0.007) were significantly decreased after surgery compared to baseline.

The prevalence of MS at entry was 60.7% or 67.9% (n= 17/28 or 19/28) according to ATP III or AHA, respectively. Following treatment, MS prevalence significantly decreased to 44.4% (n=12/28) at 10% WL and to 17.9% (n=5/28) at 25% WL (p=0.001) considering both criteria.

FLI decreased from baseline to 10% WL (99.3±1.1 vs. 96.1±5.1; p=ns) and to 25% WL (96.1±5.1 vs. 80.4±21.0; p=0.001).

Some biochemical parameters related to liver function showed a significant reduction, such as ALT (35.0±15.7 vs. 33.9±18.7 at 10% WL vs. 22.1±16.1 U/ml at 25% WL, p=0.015), GGT (39.3±32.2 vs. 21.9±12.1 vs. 17.1±12.5 U/ ml, p=0.001), pseudocholinesterase (10,039.8±1,791.6 vs. 7,654.3±1,359.3 vs. 7,845.2±1,870.0 U/l, p=0.001), or a slight reduction, such as ferritin (176.9±162.0 vs. 116.6± 112.0 vs. 104.7±118.1 ng/ml, p=ns) whereas AST (25.0± 8.2 vs. 28.0±11.9 vs. 22.8±11.8 U/ml, p=ns) remained unchanged (Table 2). At entry, none of the patients had a clinically significant increase in transaminases, i.e., not more than twice of normal values.

Ultrasonography showed a marked decrease in the signs of hepatic steatosis (one patient missed the echo examination at 10% WL) as reported in Table 3 and a significant reduction of total abdominal thickness (12.2±3.0 vs. 6.6± 1.9 cm; p=0.001), as well as of VF and SF in the whole group (Table 3). This was due to the early and significant reduction of VF thickness in males (n=14; baseline vs. 10% WL 10.0±2.0 vs. 7.3±2.4 cm; p=0.001 and vs. 25% WL 10.2±2.1 vs. 4.7±1.2 cm; p=0.001) and to the subsequent significant reduction of SF thickness in females (n=14; baseline vs. 10% WL 3.5±0.9 vs. 2.9±1.0 cm; p=ns and vs. 25% WL 3.5±0.9 vs. 2.2±0.7 cm; p=0.001).

VF/SF significantly decreased from baseline to 10% WL (3.2±1.5 vs. 2.4±1.0; p=0.026) and to 25% WL (3.2±1.5 vs. 1.6±0.7; p=0.001). When the data were evaluated by gender, VF/SF significantly decreased in males from baseline compared to 10% WL (3.9±1.4 vs. 2.8±1.0; p=0.020) and to 25% WL (3.9±1.4 vs. 1.9±0.7; p=0.001); in females, the VS/SF was lower at baseline and decreased significantly only when compared to 25% WL (2.4±1.2 vs. 1.4±0.6; p=0.011).

Discussion

Based on available evidence, bariatric surgery seems to be the only consistent, effective, long-term treatment for morbidly obese patients [21], and the Roux-en-Y gastric bypass technique has gained the greatest acceptance among bariatric surgeons in the US [22] and Europe [23].

In the present study, all patients underwent a laparoscopic gastric bypass. Our patients were studied before undergoing surgery and once they achieved ~10% and ~25% weight loss. This is the first time, to our knowledge, that a follow-up has been performed when a standardized weight loss (10% and 25% WL) has been achieved.

Table 2 Hemato-biochemical data and liver function tests at entry and following 10% and 25% WL in 28 (14 males, 14 females) severely obese patients

Biochemistry	Baseline Means±SEM	~10% WL Means±SEM	~25% WL Means±SEM
Glucose (mg/dl)	104.8±28.2	87.6±10.9**	78.4±8.0**
Insulin (μU/ml)	24.2±12.3	11.1±6.4**	8.5±4.3**
HOMA	6.4±3.8	2.4±1.4**	1.6±0.9**
Total cholesterol (mg/dl)	198.0±30.1	170.9±38.7*	176.4±33.6
HDL cholesterol (mg/dl)	46.5±12.7	41.9±11.1	51.0±11.1***
Triglycerides (mg/dl)	166.5±99.3	131.2±51.4	101.9±38.6**
AST (U/ml)	25.0±8.2	28.0±11.9	22.8±11.8
ALT (U/ml)	35.0±15.7	33.9±18.7	22.1±16.1*, ***
GGT (U/ml)	39.3±32.2	21.9±12.1**	17.1±12.5**

*p=0.05 vs. baseline; **p=0.01 vs. baseline; ***p=0.05 vs. 10% WL

Table 3 Liver ultrasound at entry and at 10% and 25% WL in 28 (14 males, 14 females) severely obese patients

Liver ultrasonography	Baseline *N* (%)	~10% WL *N* (%)	~25% WL *N* (%)
No steatosis	1 (3.6)	2 (7.4)	13 (46.4)
Mild steatosis	7 (25.0)	11 (40.7)	9 (32.1)
Moderate and severe steatosis	20 (71.4)	14 (51.9)	6 (21.4)
Total abdominal thickness (cm)	12.2±3.0	9.3±2.8**	6.6±1.9**
Visceral fat (cm)	9.0±3.0	6.4±2.4**	4.4±1.5**, ***
Subcutaneous fat (cm)	3.2±1.1	2.9±0.8*	2.2.±0.8**, ***
VF/SF	3.2±1.5	2.4±1.0*	1.6±0.7**

*p=0.05 vs. baseline; **p=0.01 vs. baseline; ***p=0.01 vs. 10% WL

The degree and duration of overweight, a more central distribution of body fat, and weight gain over the years represent well-known risk factors for type 2 diabetes [24]. Weight gain often precedes the onset of diabetes [25]; vice versa, weight loss, after bariatric surgery, induces a stable reversion of diabetes [26]. The main metabolic link between excess body fat and diabetes is hyperinsulinemia with insulin-resistance known to be the pathogenetic determinant of the metabolic syndrome. Among other things, hyperinsulinemia increases hepatic VLDL synthesis and secretion, PAI-1 synthesis, sympathetic nervous system activity and renal sodium reabsorption, thus facilitating the onset of hypertension, hypertriglyceridemia, and liver steatosis, all typical features of the MS [27].

According to the results of our study, all the MS risk factors improved with weight loss: our patients showed a rapid and significant reduction of blood glucose, insulin, and HOMA, an improvement of dyslipidemia, particularly hypertriglyceridemia, and a reduction in the prevalence of the metabolic syndrome. This improvement is probably due to the drastic restriction of food intake and possible gastrointestinal hormone adaptation. In fact, the reduction in HOMA index—due to a drop in insulin serum concentration and resistance—indicates that the gastric bypass can modify gastrointestinal endocrine pattern [28].

As recently stated, excess body fat simultaneously induces a reduced secretion of adiponectin, with tumor necrosis factor alpha and interleukin 6 hypersecretion contributing to the insulin resistance; this represents an endocrine and a metabolic link between adipose tissue and the metabolic complications associated with excess visceral body fat and is now encompassed in the definition of the metabolic syndrome [29].

The frequent association of hypertension with obesity leads us to consider also in this case a direct active role of adipose tissue, in particular of splanchnic adipose tissue, in the development of hypertension.

RMR is known to account for a large percentage of daily calorie expenditure. Obese individuals generally have elevated RMR and experience a significant drop in RMR with weight loss [30, 31]. Carey and colleagues described a significant RMR reduction only in the first month after surgery, and these results are in line with lean body mass (LBM) changes. However, WL may produce a disproportionate reduction in RMR [32], even when adjusted for changes in LBM. At 25% WL, we observed a significant reduction of basal metabolic rate both in absolute values and when correcting for FFM. In our study, percentage of FFM appeared unchanged in the initial phase of WL, suggesting a similar loss of FM and FFM also related to the higher fluid loss in the short term; vice versa, FFM appeared increased about 30 weeks after surgery, suggesting a prevalent fat loss after the acute postoperative period of weight loss. BI has been demonstrated to be well correlated with total body water by other authors [33] and in this study it was significantly reduced in the more precocious phase of weight loss. PhA is related to intracellular/extracellular body water ratio and was not significantly changed at 10% and 25% weight loss, demonstrating a balanced loss of water from the two compartments as also shown in other groups of patients [34].

The use of ultrasound for the evaluation of fat distribution was introduced in the early 1990s [28]. In a study carried out in obese women, a strong correlation between visceral fat thickness estimated with sonography and visceral adipose tissue area measured with computed tomography (CT) has been observed [35]. Recently, a review [36, 37] and a research study have confirmed the accuracy and repeatability of sonographic measurement of visceral thickness in various patient groups [38–40] and the correlation of sonographic measurements with CT- and magnetic-resonance-imaging-based estimates [39, 40]. Also, sonographic measurements have been associated with metabolic values and central adiposity, more strongly than anthropometric data [39, 41–43].

Intra-abdominal fat thickness correlates with total cholesterol and fasting glucose levels in men and women at high risk of cardiovascular disease (CVD) [41], apolipoprotein B, fasting insulin, and TG levels in obese women [44] and inversely with HDL cholesterol levels in diabetic men and women and those at high risk of CVD [41, 44, 45]. Furthermore, intra-abdominal fat thickness has been

correlated with the HOMA index in diabetic patients and obese women [44, 45]. In a multivariate analysis by Leite et al. [46], visceral fat thickness was the most significant CVD marker in both sexes and was a significant independent predictor of the presence of MS [41].

Our sonographic data showed a marked decrease in hepatic steatosis and a significant reduction of total abdominal thickness due to the early and significant reduction of visceral fat thickness in males and to the significant reduction of subcutaneous fat thickness in female. These data were confirmed by the observation of the reduction in VF/SF; this reduction was significant in males both from baseline to 10% WL and from baseline to 25% WL, while in females the VF/SF decreased significantly only from baseline to 25% WL.

In another study [47] investigating the relationship between visceral obesity and hepatic steatosis in a small group of obese women treated with adjustable silicone gastric banding, the authors found that in the early phase of rapid weight loss there was also a preferential mobilization of visceral fat.

In conclusion, in our view, longer-term observations are required for in-depth evaluation of body fat compartments and body composition changes following bariatric surgery.

References

1. Renehan AG, Tyson M, Egger M, et al. Body mass index and incidence of cancer: a systematic review and metabolic prospective observational studies. Lancet. 2008;371:569–78.
2. Hsu IR, Kim SP, Kabir M, et al. Metabolic syndrome, hyperinsulinemia, and cancer. Am J Clin Nutr. 2007;86:867–71.
3. Olshansky SY, Passaro DJ, Hershow RC, et al. A potential decline in life expectancy in the United States in the 21st century. N Engl J Med. 2005;352(11):1138–45.
4. Hamilton MT, Hamilton DG, Zderic TW. Role of low energy expenditure and sitting in obesity, metabolic syndrome, type 2 diabetes, and cardiovascular disease. Diabetes. 2007;56:2655–67.
5. Contaldo F, Pasanisi F, Finelli C, et al. Obesity, heart failure and sudden death. Nutr Metab Cardiovasc Dis. 2002;12(4):190–7.
6. Marchesini G, Bugianesi E, Forlani G, et al. Nonalcoholic fatty liver, steatohepatitis, and the metabolic syndrome. Hepatology. 2003;37:917–23.
7. Bugianesi E, Leone N, Vanni E, et al. Expanding the natural history of nonalcoholic steatohepatitis: from cryptogenic cirrhosis to hepatocellular carcinoma. Gastroenterology. 2002;123:134–40.
8. Colicchio P, Tarantino G, del Genio F, et al. Nonalcoholic fatty liver disease in young adult severely obese non-diabetic patients in South Italy. Ann Nutr Metab. 2005;49:289–95.
9. Stampfer MJ, Maclure KM, Colditz GA, et al. Risk of symptomatic gallstones in women with severe obesity. Am J Clin Nutr. 1992;55:652–8.
10. NIH. Third report of the National Cholesterol Education Program Expert Panel (Adult Treatment Panel III). Bethesda: NIH Publication; 2001. p. 01-3670.
11. Bedogni G, Bellentani S, Miglioli L, et al. The Fatty Liver Index: a simple and accurate predictor of hepatic steatosis in the general population. BMC Gastroenterol. 2006;6:33.
12. del Genio F, Alfonsi L, Marra M, et al. Metabolic and nutritional status changes after 10% weight loss in severely obese patients treated with laparoscopic surgery vs integrated medical treatment. Obes Surg. 2007;17(12):1592–8.
13. Saverymuttu SH, Joseph AEA, Maxwell JA. Ultrasound scanning in the detection of hepatic fibrosis and steatosis. Br Med J. 1986;292:13–5.
14. Ricci C, Longo R, Gioulis E, et al. Noninvasive in vivo quantitative assessment of fat content in human liver. J Hepatol. 1997;27:108–13.
15. Osawa H, Mori Y. Sonographic diagnosis of fatty liver using a histogram technique that compares liver and renal cortical amplitude. J Clin Ultrasound. 1996;24:25–9.
16. Ribeiro-Filho FF, Faria AN, Azjen S, et al. Methods of estimation of visceral fat: advantages of ultrasonography. Obes Res. 2003;11:1488–94.
17. Cignarelli M, DePergola G, Picca G, et al. Relationship of obesity and body fat distribution with ceruloplasmin serum levels. Int J Obes Relat Metab Disord. 1996;20:809–13.
18. Targher G, Tonoli M, Agostino G, et al. Ultrasonographic intra-abdominal depth and its relation to haemostatic factors in healthy males. Int J Obes Relat Metab Disord. 1996;20:882–5.
19. Rissanen P, Makimattila S, Vehmas T, et al. Effect of weight loss and regional fat distribution on plasma leptin concentration in obese women. Int J Obes Relat Metab Disord. 1999;23:645–9.
20. Kinoshita T, Ittoh M. Longitudinal variance of fat mass deposition during pregnancy evaluated by ultrasonography: the ratio of visceral fat to subcutaneous fat in the abdomen. Gynecol Obstet Invest. 2006;61(2):115–8.
21. Anonymous. Gastrointestinal surgery for severe obesity: National institutes of health consensus development conference draft statement. Obes Surg. 1991;1:257–65.
22. Buchwald H, Williams SE. Bariatric surgery worldwide 2003. Obes Surg. 2004;14(9):1157–64.
23. Sjöström L, Narbro K, Sjöström CD, et al. Effects of bariatric surgery on mortality in Swedish obese subjects. N Engl J Med. 2007;357(8):741–52.
24. Harris ML, Flegal KM, Cowie LL, et al. Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in US adults: the Third National Health and Nutrition Examination Survey 1988–94. Diabetes Care. 1998;21:518–24.
25. Colditz GA, Willet WC, Rotnitky A, et al. Weight gain as a risk factor for clinical diabetes in women. Ann Intern Med. 1995;122:481–6.
26. Pories WJ, Swanson MS, MacDonald KG, et al. Who would have thought it? An operation proves to be the most effective therapy for adult-onset diabetes mellitus. Ann Surg. 1995;222(3):339–50.
27. Despres JP. The insulin-resistance-dyslipidemic syndrome of visceral obesity: effect on patient's risk. Obes Rev. 1998;6(1):8–17.
28. Rubino F, Gagner M, Gentileschi P, et al. The early effect of the Roux-en-Y gastric bypass on hormones involved in body weight regulation and glucose metabolism. Ann Surg. 2004;240:236–42.
29. Hoene M, Weigert C. The role of interleukin-6 in insulin-resistance, body fat distribution and energy balance. Obes Rev. 2008;9:20–9.
30. Benedetti G, Mingrone G, Marcoccia S, et al. Body composition and energy expenditure after weight loss following bariatric surgery. J Am Coll Nutr. 2000;19:270–4.
31. Carey DG, Pliego GJ, Raymond RL. Body composition and metabolic changes following bariatric surgery: effects on fat mass, lean mass and basal metabolic rate: six months to one-year follow-up. Obes Surg. 2006;16:1602–8.
32. de Boer JO, van Es AJH, Roovers LCA, et al. Adaptation of energy metabolism of overweight women to low-energy intake, studied with whole body calorimeters. Am J Clin Nutr. 1986;44:585–95.
33. Kushner RF, Schoeller DA, Field CR, et al. Is the impedance index (ht2/R) significant in predicting total body water? Am J Clin Nutr. 1992;56(5):835–9.

34. Marra M, Caldara A, Montagnese C, et al. Bioelectrical impedance phase angle in constitutionally lean females, ballet dancers and patients with anorexia nervosa. Eur J Clin Nutr. 2008; in press
35. Armellini F, Zamboni M, Rigo L, et al. The contribution of sonography to the measurement of intra abdominal fat. J Clin Ultrasound. 1990;18:563–7.
36. Vlachos IS, Hatziioannou A, Perelas A, et al. Sonographic assessment of regional adiposity. Am J Roentgenol. 2007;189:1545–53.
37. Koda M, Senda M, Kamba M, et al. Sonographic subcutaneous and visceral fat indices represent the distribution of body fat volume. Abdom Imaging. 2007;32:387–92.
38. Armellini F, Zamboni M, Robbi R, et al. Total and intra-abdominal fat measurements by ultrasound and computerized tomography. Int J Obes Relat Metab Disord. 1993;17:209–14.
39. Tornaghi G, Raiteri R, Pozzato C, et al. Anthropometric or ultrasonic measurements in assessment of visceral fat? A comparative study. Int J Obes Relat Metab Disord. 1994;18:771–5.
40. Abe T, Kawakami Y, Sugita M, et al. Use of B-mode ultrasound for visceral fat mass evaluation: comparisons with magnetic resonance imaging. Appl Human Sci. 1995;14:133–9.
41. Pontiroli AE, Pizzocri P, Giacomelli M, et al. Ultrasound measurement of visceral and subcutaneous fat in morbidly obese patients before and after laparoscopic adjustable gastric banding: comparison with computerized tomography and with anthropometric measurements. Obes Surg. 2002;12:648–51.
42. Stolk RP, Meijer R, Mali WP, et al. Ultrasound measurements of intra abdominal fat estimate the metabolic syndrome better than do measurements of waist circumference. Am J Clin Nutr. 2003;77:857–60.
43. Minocci A, Savia G, Lucantoni R, et al. Leptin plasma concentrations are dependent on body fat distribution in obese patients. Int J Obes Relat Metab Disord. 2000;24:1139–44.
44. Ribeiro-Filho FF, Faria AN, Kohlmann O Jr, et al. Ultrasonography for the evaluation of visceral fat and cardiovascular risk. Hypertension. 2001;38:713–7.
45. Kim SK, Kim HJ, Hur KY, et al. Visceral fat thickness measured by ultrasonography can estimate not only visceral obesity but also risks of cardiovascular and metabolic diseases. Am J Clin Nutr. 2004;79:593–9.
46. Leite CC, Wajchenberg BL, Radominski R, et al. Intra-abdominal thickness by ultrasonography to predict risk factors for cardiovascular disease and its correlation with anthropometric measurements. Metab. 2002;51:1034–40.
47. Busetto L, Tregnaghi A, De Marchi F, et al. Liver volume and visceral obesity in women with hepatic steatosis undergoing gastric banding. Obes Res. 2002;10(5):408–11.