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ORIGINAL CONTRIBUTIONS

Impact of Intragastric Balloon Treatment on Adipokines, Cytokines, and Metabolic Profile in Obese Individuals

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Abstract

Background Obesity is accompanied by adipose tissue remodeling characterized by increased production of tumor necrosis factor-alpha (TNF-α), interleukin (IL)-6, leptin and resistin and reduced secretion of adiponectin, which favors inflammation, metabolic disorders, and cardiovascular diseases. Although intragastric balloon (IGB) can be considered safe and effective for weight loss, its effect on serum levels of these biomarkers has been evaluated only in a few studies, while no previous study evaluated its effect on circulating levels of resistin, TNF-α, and IL-6. The aim of this study was to evaluate the changes in serum levels of metabolic and inflammatory biomarkers in obese patients submitted to IGB treatment.

Methods A prospective observational study involving 42 patients with obesity using IGB for 6 months. The patients were evaluated, on the day of insertion and withdrawal or adjustment of IGB, for the following: anthropometric measures and serum levels of adiponectin, leptin, resistin, TNF-α, IL-6, high-sensitivity C-reactive protein (hs-CRP), glucose, insulin, uric acid, triglycerides, and total cholesterol and fractions.

Results The body mass index decreased from 35.15 ± 0.41 to 29.50 ± 0.54 kg/m^2. There was a reduction ($p < 0.05$) in leptin, hs-CRP, glucose, insulin, HOMA-IR, and triglycerides, while the adiponectin/leptin ratio increased ($p < 0.05$). Moreover, weight loss presented (1) a positive association with the decrease in leptin, hs-CRP, glucose, insulin, HOMA-IR, uric acid, and total cholesterol and (2) a negative association with the reduction in adiponectin/leptin ratio.

Conclusions The present study suggests that 6 months of IGB treatment in obese individuals reduce serum leptin and hs-CRP and improves insulin resistance and lipid profile which may decrease cardiovascular risk.

Keywords Intragastric balloon · Obesity · Weight loss · Adipokines · Cytokines

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Introduction

Obesity is a chronic disease that continues to be highly prevalent, representing a global public health problem [1]. As reported by the World Health Organization (WHO), in 2016, 39% of the adult world population were overweight, while 11% of men and 15% of women were obese [2]. These data are of concern since obesity is a risk factor for the development of several chronic diseases, including type 2 diabetes, hypertension, dyslipidemia, chronic kidney disease, obstructive sleep apnea, osteoarthritis, and cancer [3, 4]. Many studies have shown that obesity is associated with an increased risk of all-cause mortality [5, 6]. Therefore, a high body mass index (BMI) can be considered a predictor of overall mortality with a reduction in survival as the index increases [1, 7].

Few of the consequences of obesity, such as sleep apnea and osteoarthritis, can be attributed to the excessive

accumulation of fat mass per se [4]. However, inflammatory and metabolic derangements associated with excessive fat accumulation are involved in the increased cardiometabolic risk [4, 8]. Adipose tissue functions as an endocrine organ, by secreting bioactive substances known as adipokines, and contains several different cellular populations including adipocytes, pre-adipocytes, and macrophages. Obesity is accompanied by adipose tissue remodeling with adipocyte hypertrophy and changes in cellular composition that leads to low-grade chronic inflammation [8, 9]. This pro-inflammatory state is mediated by an unbalanced production of cytokines and adipokines, with an increase in tumor necrosis factor-alpha (TNF-α), interleukin (IL)-6, IL-1β, leptin, and resistin, and a reduction in adiponectin and IL-10, promoting metabolic disorders, such as insulin resistance [8, 10, 11].

Current guidelines recommend that obesity treatment should be based on the severity of the excess weight and the presence of co-morbidities, prioritizing initially a clinical approach [12, 13]. For individuals with excess weight refractory to clinical treatment, there are other options including the intragastric balloon (IGB) [13–16]. IGB is considered a temporary, minimally invasive, effective, and safe method for weight loss [16–19].

Despite the important role of adipokines and cytokines in the cardiometabolic risk of obese individuals, to date, the impact of weight loss during IGB treatment on these biomarkers are still not completely understood. Only a few studies assessed serum levels of leptin and/or adiponectin [20–24], but none evaluated the effect of IGB therapy on circulating levels of resistin and pro-inflammatory cytokines such as TNF-α and IL-6. In our opinion, the evaluation of inflammatory status during IGB treatment is recommended due to few previous studies that observed the following: (1) inflammatory process in the gastric mucosa after IGB insertion [25, 26] and (2) non-significant increase in adiponectin during IGB treatment [21–24], as this adipokine can be downregulated by pro-inflammatory cytokines [27, 28]. Therefore, the aim of this study was to evaluate the changes in adipokines, pro-inflammatory cytokines, and metabolic profile in obese individuals submitted to 6 months of IGB treatment.

Patients and Methods

This prospective observational study was conducted with patients suffering from obesity that were submitted to IGB treatment for 6 months at a private clinic, between March 2016 and October 2017. The design of this study has already been described in a previous publication [29], in which data about changes in body adiposity, dietary intake, physical activity, and quality of life during IGB treatment were evaluated. The data of our study were divided into two papers to allow a comprehensive discussion of the different results. In the present publication, we included data about modifications in serum levels of adipokines and cytokines, in insulin resistance, lipid profile, and serum uric acid during IGB treatment.

Potential participants were recruited among patients who had already scheduled the placement of non-adjustable IGB (Orbera) or adjustable IGB (Spatz). The inclusion criteria were as follows: adult patients (20–59 years) presenting with BMI ≥ 30 and < 40 kg/m^2 that had failed to lose weight in well-conducted clinical treatments. The exclusion criteria were as follows: diagnosis of endocrine disorders (diabetes mellitus, hypothyroidism, polycystic ovary syndrome), acquired immunodeficiency syndrome (AIDS), inflammatory conditions, malignant diseases, autoimmune diseases, chronic kidney disease, heart failure, or hepatic failure.

Patients who met the eligibility criteria and agreed to take part in the study were submitted to nutritional and laboratory evaluation at the day of balloon placement (baseline). The same evaluations were performed after 6 months at the day of balloon retrieval (for patients using non-adjustable IGB) or balloon adjustment (for patients using adjustable IGB). Half of the patients used the non-adjustable IGB and half the adjustable balloon. For patients using the adjustable balloon, only one adjustment in balloon volume was scheduled at 6 months of treatment, while IGB withdrawal was scheduled at 1 year.

Balloon Placement

As described in a previous publication of our group [30], balloon insertion was performed after a diagnostic endoscopy to detect pathologies that might contraindicate balloon placement, such as active peptic ulcer, grades C–D esophagitis, large-volume hiatal hernia, esophageal/fundus varices, esophageal strictures, and prior gastric surgery.

The endoscopy procedure was performed under deep sedation without endotracheal intubation, with continuous oxygen support of 5 L/min, under the supervision of an anesthesiologist. After implant of the balloon, correct positioning was checked endoscopically, with the balloon valve positioned 2 cm below the cardia. The balloon inflation was under direct vision with the endoscope in rear-view position (u-turn maneuver). The balloon was filled with 3% saline solution and 10 mL of 4% methylene blue. The balloon volume ranged from 600 to 700 mL.

After filling, the Orbera balloon valve was closed with a syringe vacuum and the catheter disconnected by traction, while the filling catheter of Spatz balloon was pulled up until the externalization of the valve through the patient's mouth and was disconnected from the valve that was covered with a suitable cap with a blue nylon loop on its top. Holding the loop, the valve was gently returned into the patient, releasing it at the oropharynx. Then, both balloons were visually inspected to detect possible deflation or valve malfunctions

and to confirm correct positioning in the gastric fundus. If a leakage was detected, a prompt replacement of the defective balloon was conducted. After balloon placement, patients remained in the anesthesia recovery room until complete recovery from sedation had been confirmed and were then discharged.

Follow-up

In the first 3 days after balloon insertion, patients were instructed to use three antiemetic drugs (metoclopramide, ondansetron, and dimenhydrinate), an anti-foaming drug (dimethicone), and analgesics/antispasmodics (scopolamine plus dipyrone, acetaminophen). All patients were put on a proton pump inhibitor (PPI; pantoprazole magnesium) throughout the treatment: a double dose in the first month (80 mg) and a full dose from the second month to the end (40 mg).

Patients were also instructed to follow a 5-day liquid diet (with progressive increase in ingested volume). On the sixth day, a semi-solid diet was prescribed, and after the 13th day, solids were introduced. Thereafter, the patients were referred for personalized nutritional counseling, when an individualized low-calorie diet was prescribed. The total energy intake was determined as 12 Kcal/kg actual body weight/day, which varied from 900 to 1400 Kcal/day, and was similar to that recommended in previous studies with IGB [31, 32]. Additionally, vitamin and mineral supplementation were given to all patients during the entire treatment. Follow-up with a multidisciplinary team including a physician, a nutritionist, and a psychologist once a month was available for all participants.

Nutritional Assessment

Height was measured with a stadiometer accurate to ± 0.5 cm, and weight was obtained using a calibrated scale accurate to ± 0.1 kg (Welmy®) after participants, without shoes and wearing light clothing, attempted to empty their bladder. BMI was calculated using the standard equation (kg/m^2) [33]. Waist circumference (WC) was measured in the standing position, midway between the lower margin of the last rib and the iliac crest, at mid exhalation. Hip circumference (HC) was measured at the widest point of the hip/buttocks area with the measuring tape parallel to the floor [34]. Waist-to-hip ratio was determined by dividing WC (cm) by HC (cm). Waist-to-height ratio was obtained by dividing WC (cm) by height (cm). The % excess weight loss (%EWL) was determined considering ideal body weight as the equivalent to a BMI of 25 kg/m^2.

Laboratory Evaluation

Blood samples were analyzed to measure glucose, insulin, total cholesterol, high-density lipoprotein cholesterol (HDL-c), triglycerides, uric acid, adiponectin, resistin, leptin, TNF-α, IL-6, and high-sensitivity C-reactive protein (hs-CRP). Serum levels of adiponectin, resistin, TNF-α, and IL-6 were determined by Luminex method, using commercial kit (MILLIPLEX® MAP). Commercial ELISA kit (Merck Millipore®) was used to measure serum concentration of leptin. The turbidimetry method was used to determine the serum levels of hs-CRP (BioSystems).

Glucose was determined by glucose hexokinase method. Total cholesterol, HDL-c, triglycerides, and uric acid concentrations were assessed by using enzymatic colorimetric method. Low-density lipoprotein cholesterol (LDL-c) was estimated by using the Friedewald formula [35]. Serum levels of insulin were determined by electro-chemiluminescence immunoassay (ECLIA) using a commercial kit (Roche Diagnostics, Basel, Switzerland). Insulin resistance status was assessed using the homeostasis model assessment of insulin resistance (HOMA-IR) index, which was calculated as fasting insulin (μU/mL) × fasting plasma glucose (mmol/L)/22.5 [36].

The presence of modifiable risk factors for cardiovascular disease was evaluated considering the following parameters and cutoff points: obesity—BMI ≥ 30 kg/m^2 [33]; increased hs-CRP—serum hs-CRP > 0.30 mg/dL [37]; insulin resistance—HOMA-IR ≥ 2.71 [38]; increased LDL-cholesterol—LDL-cholesterol ≥130 mg/dL; and hypertriglyceridemia—TG ≥ 150 mg/dL [37].

Statistical Methods

Categorical variables were expressed as absolute numbers and percentage. The Shapiro-Wilk test was used to test the normality of the continuous variables and skewed data were log transformed to improve normality. Continuous variables with normal distribution were presented as mean ± standard errors and those without normal distribution were presented as median and interquartile range.

Repeated measures analysis of variance (ANOVA) was used to evaluate changes in continuous variables obtained pre- and post-IGB treatment. The Pearson or Spearman correlation was performed to evaluate the association of % total weight loss (%TWL) with inflammatory and metabolic biomarkers. Partial correlation controlled for different confounders were also used.

Patients who (1) withdraw the IGB treatment before completing the 6 months of follow-up, (2) remained with Orbera balloon for more than 8 months, or (3) performed Spatz balloon adjustment before 6 months or after 8 months of follow-up were not included in the final analyses. All statistical

analyses were performed using STATA (v.12.0, Stata Corp, College Station, TX, USA) and a p value of < 0.05 was considered to be significant.

Results

A total of 173 subjects were interviewed, of whom 56 met the eligibility criteria and agreed to participate in the study. Forty-two patients completed all evaluations and were included in statistical analyses. The baseline characteristics of the participants are detailed in Table 1. After 6 months of IGB treatment, body weight and BMI decreased significantly (Table 2). The mean % TWL and % EWL were 15.88 ± 1.42 and 56.04 ± 4.90, respectively. There was a significant reduction in serum levels of leptin and hs-CRP, while the adiponectin/leptin ratio presented a significant increase (Table 3). After adjustment for confounders, %TWL presented a positive association with the reduction in leptin and hs-CRP, as well as presented a negative association with the reduction in adiponectin/leptin ratio (Table 4).

There was a significant reduction in serum levels of glucose, insulin, HOMA-IR and triglycerides (Table 5) and after adjustment for confounders, %TWL presented a positive association with the reduction in glucose, insulin, HOMA-IR, total cholesterol, and uric acid (Table 6).

The frequency of modifiable risk factors for cardiovascular disease presented a marked decrease from pre- to post-IGB treatment: obesity (100% vs. 49%), increased hs-CRP (78% vs. 46%), insulin resistance (41% vs. 10%), increased LDL-cholesterol (51% vs. 43%), and hypertriglyceridemia (34% vs. 5%).

Table 1 Baseline characteristics of study participants

Characteristics	Total group ($n = 42$)
Age (years)	37.60 ± 1.28
Gender (male/female) (n; %)	10 (24%)/32 (76%)
Smoking habit (n; %)	2 (5%)
Alcohol intake (n; %)	27 (64%)
Body mass index (kg/m^2)	35.15 ± 0.41
Hypertension (n; %)	6 (14%)
Dyslipidemia (n; %)	32 (76%)
Laboratory Variables	
Glucose (mg/dL)	88.12 ± 1.51
Urea (mg/dL)	31.00 (26.00–36.00)
Creatinine (mg/dL)	0.80 (0.70–0.90)
Uric acid (mg/dL)	4.93 ± 0.20
Total cholesterol (mg/dL)	200.74 ± 6.24
HDL-c (mg/dL)	47.53 ± 1.99
LDL-c (mg/dL)	132.70 (100.20–158.90)
Triglycerides (mg/dL)	113.00 (83.00–157.00)

Values as mean ± error deviation for normal distribution or as median (interquartile range) for not normal distribution or absolute values (%)

HDL-c, high-density lipoprotein cholesterol; *LDL-c*, low-density lipoprotein cholesterol

Discussion

In the present study, there was a significant decrease in circulating levels of leptin after 6 months of IGB treatment. This result is in accordance with previous studies that evaluated the effects of IGB treatment during 6 months on serum leptin [20–24]. There is no divergence among the published studies considering the significant reduction in serum leptin after IGB-induced weight loss. Leptin is produced predominantly by adipose tissue and its secretion is directly proportional to total body fat mass; thus, as weight loss occurs, leptin decreases [39, 40]. This significant association between % TWL and the reduction in leptin was observed in the present study ($r = 0.75$; $p < 0.0001$).

Under normal conditions, in lean individuals, leptin participates in the hypothalamic regulation of food intake suppressing appetite. Obese individuals develop leptin resistance which abolishes the "anorexigenic" effect of leptin and hyperleptinemia [39, 41], which is associated with adverse cardiovascular outcomes through vascular inflammation, oxidative stress, atherothrombosis, left ventricular hypertrophy, increased blood pressure, and systemic insulin resistance [40, 42]. Thus, the decrease in serum levels of leptin in the present study not only reflects a reduction in adipose tissue but also indicates beneficial effects on cardiometabolic alterations associated with obesity.

Serum levels of adiponectin did not present a significant increase after 6 months of IGB treatment in the present study. Although the majority of the previous studies also found no significant changes in adiponectin during 6 months of IGB treatment [21–24], Mion et al. (2005) [20] observed an increase in this adipokine. Studies evaluating the effects of weight loss induced by methods other than IGB on circulating adiponectin also present inconsistent results [43–45]. Adiponectin has anti-inflammatory, antioxidant, anti-atherogenic, and insulin-sensitizing action [11]. There is evidence that it inhibits the transformation of macrophages into foam cells [42]. Obese individuals present a marked decrease in serum concentration of adiponectin [8]. In the present study, although without reaching statistical significance, there was an increase in adiponectin levels and a significant increase in adiponectin/leptin ratio, which may confer health benefits during weight loss induced by IGB treatment.

In the present study, there were no significant changes in serum levels of resistin, TNF-α, and IL-6 after 6 months of IGB treatment. Previous studies using other methods for weight loss presented divergent results regarding these

Table 2 Anthropometric parameters in individuals with obesity submitted to intragastric balloon (IGB) treatment during 6 months

Variables	IGB treatment		Reduction	% reduction	*p**
	Pre ($n = 42$)	Post ($n = 42$)			
Body weight (kg)	95.99 ± 1.94	80.57 ± 2.0	15.42 ± 1.48	15.88 ± 1.42	< 0.0001
Excess body weight (kg)	28.10 ± 1.25	11.26 ± 10.54	15.42 ± 1.48	56.04 ± 4.90	< 0.0001
Body mass index (kg/m^2)	35.15 ± 0.41	29.50 ± 0.54	5.65 ± 0.52	15.93 ± 1.42	< 0.0001
Waist circumference (cm)	108.42 ± 1.61	95.09 ± 1.59	13.33 ± 1.39	12.07 ± 1.18	< 0.0001
Hip circumference (cm)	120.54 ± 1.08	109.99 ± 1.36	10.55 ± 1.05	8.72 ± 0.85	< 0.0001
Waist-to-hip ratio	0.90 ± 0.01	0.87 ± 0.01	0.04 ± 0.01	3.74 ± 0.83	0.0001
Waist-to-height ratio	0.66 ± 0.01	0.58 ± 0.01	0.08 ± 0.01	12.03 ± 1.19	< 0.0001

Values as mean ± standard error for normal distribution or as median (interquartile range) for not normal distribution

Reduction = pre-IGB treatment − post-IGB treatment

*p value refers to differences between pre- and post-IGB treatment (repeated measures ANOVA)

adipokines/cytokines [43, 45–49]. TNF-α and IL-6 are secreted mainly by macrophages present in adipose tissue and are positively associated with body adiposity and hyperinsulinemia [50, 51]. Both have been associated with inflammatory pathways, insulin resistance, and atherosclerosis [52, 53].

The divergence in the results of the studies that evaluated the effects of weight loss on adiponectin, resistin, TNF-α, and IL-6 may be explained by the high variability in study designs such as the length of follow-up, type of intervention for weight loss, the degree of adiposity at baseline, and the amount of weight lost. However, two factors seem to be important determinants of changes in these inflammatory and metabolic biomarkers: the amount of weight lost and the length of the study. Studies with severely obese subjects undergoing a marked weight loss induced by bariatric surgery during a follow-up of 6 months observed a significant increase in adiponectin [46, 47, 54, 55] and decrease in IL-6 [56], TNF-α [47, 55] and resistin [47]. While some studies that used lifestyle modifications to obtain weight loss during at least 9 months also observed a significant increase in adiponectin [44, 49, 57] and a decrease in TNF-α [49] and IL-6 [43].

Serum levels of hs-CRP decreased significantly in the present study, which is in agreement with previous studies with IGB treatment [58, 59]. The decrease in circulating hs-CRP is consistently observed during weight loss induced by different methods [43, 46, 54, 60–62]. Considering that TNF-α promotes stimulation of IL-6 secretion, which in turn stimulates the production of CRP by the liver [51, 52], the decrease in hs-CRP without a concomitant reduction in TNF-α and IL-6 in the present study can be considered unexpected. However, Rosc et al. (2015) [63] also observed a decrease in plasma hs-CRP without changes in TNF-α and IL-6 in subjects with morbid obesity after weight loss by lifestyle changes. Pardina et al. (2012) [64] suggest that the role of TNF-α and IL-6 in the induction of CRP in different tissues may be reviewed. Additionally, Li et al. (2017) [65] emphasize

Table 3 Serum levels of adipokines, cytokines, and high-sensitivity C-reactive protein in individuals with obesity submitted to intragastric balloon (IGB) treatment during 6 months

Variables	IGB treatment		*p**
	Pre ($n = 42$)	Post ($n = 42$)	
Adiponectin (pg/mL)	7503.0 (5722.5–9947.5)	7850.0 (6363.0–9660.0)	0.94
Resistin (pg/mL)	76.75 (52.00–96.00)	59.75 (49.00–88.00)	0.38
Leptin (ng/mL)	60.21 (29.76–100.00)	16.42 (8.38–27.05)	< 0.0001
Adiponectin/leptin	144.01 (93.14–269.91)	466.77 (244.53–926.38)	0.007
TNF-α (pg/mL)	12.00 (11.00–14.00)	14.50 (10.00–18.00)	0.22
IL-6 (pg/mL)	15.21 ± 0.51	17.15 ± 0.91	0.11
hs-CRP (mg/dL)	0.58 (0.31–1.21)	0.26 (0.12–0.46)	< 0.0001

Values as mean ± standard error for normal distribution or as median (interquartile range) for not normal distribution

*p value refers to differences between pre- and post-IGB treatment (repeated measures ANOVA)

TNF-α, tumor necrosis factor-alpha; *IL-6*, interleukin-6; *hs-CRP*, high-sensitivity C-reactive protein

Table 4 Correlation between % total weight loss with the reduction in adipokines, cytokines, and high-sensitivity C-reactive protein in individuals with obesity submitted to intragastric balloon treatment during 6 months

Variables	Correlation		Partial correlation*	
	r	*p*	*r*	*p*
Adiponectin (pg/mL)	− 0.17	0.34	− 0.21	0.28
Resistin (pg/mL)	− 0.05	0.78	− 0.18	0.33
Leptin (ng/mL)	0.24	0.18	0.75	< 0.0001
Adiponectin/Leptin	− 0.58	0.0005	− 0.73	< 0.0001
TNF-α (pg/mL)	0.32	0.08	0.09	0.67
IL-6 (pg/mL)	0.15	0.43	0.05	0.80
hs-CRP (mg/dL)	0.39	0.02	0.49	0.004

TNF-α, tumor necrosis factor-alpha; *IL-6*, interleukin-6; *hs-CRP*, high-sensitivity C-reactive protein

*Adjusted for the value of the inflammatory variable in the pre-treatment period, pre-treatment body mass index, sex, and age

Table 6 Correlation between % total weight loss and reduction in glucose metabolism biomarkers, lipid profile, and uric acid in individuals with obesity submitted to intragastric balloon treatment during 6 months

Variables	Correlation		Partial correlation*	
	r	*p*	*r*	*p*
Glucose (mg/dL)	0.33	0.03	0.39	0.02
Insulin (μU/mL)	0.17	0.29	0.38	0.02
HOMA-IR	0.23	0.14	0.41	0.01
Total cholesterol (mg/dL)	0.35	0.02	0.41	0.01
HDL-c (mg/dL)	0.09	0.60	0.18	0.28
LDL-c (mg/dL)	0.35	0.02	0.29	0.08
Triglycerides (mg/dL)	0.08	0.64	0.10	0.56
Uric acid (mg/dL)	0.54	0.0004	0.52	0.001

HOMA-IR, homeostatic model assessment of insulin resistance; *HDL-c*, high-density lipoprotein cholesterol; *LDL-c*, low-density lipoprotein cholesterol

*Adjusted for the values of the metabolic variable in the pre-treatment period, pre-treatment body mass index, sex, and age

that although TNF-α and IL-6 have a role in inflammation and metabolic disorders, only CRP has established cutoff points, being considered a pro-inflammatory biomarker associated with cardiovascular risk.

The divergent effects of weight loss on the circulating levels of adipokines, cytokines, and hs-CRP observed in our study were also found in studies with weight loss induced by other methods. For example, Ho et al. (2015) [43] observed a decrease in leptin and CRP levels without changes in adiponectin, resistin, TNF-α, and IL-6 after 6 months of intervention program with caloric restriction and weight loss, while Mallipedhi et al. (2014) [66] found a reduction in leptin, CRP, and IL-6 levels without changes in adiponectin after 6 months of bariatric surgery–induced weight loss.

Mechanisms explaining this delay in changes in adiponectin levels remain unknown; however, it seems to be secondary to effects of weight loss on lipolysis. It has been proposed that in the early stages of weight loss, adipocyte size does not change. Therefore, adipose tissues continue presenting high levels of free fatty acid that are associated with an increased number of macrophages, high cytokine production, and, consequentially, low levels of adiponectin [49, 67].

The evaluation of the lipid profile in the present study revealed that 76% of the participants presented with dyslipidemia at baseline. After 6 months of IGB treatment was observed a significant decrease in triglycerides and a positive association between weight loss and a decrease

Table 5 Metabolic biomarkers in individuals with obesity submitted to intragastric balloon (IGB) treatment during 6 months

Variables	IGB treatment		*p**
	Pre (*n* = 42)	Post (*n* = 42)	
Glucose (mg/dL)	88.12 ± 1.51	81.10 ± 1.40	0.0001
Insulin (μU/mL)	11.06 (7.22–15.74)	4.95 (3.00–6.40)	< 0.0001
HOMA-IR	2.36 (1.41–3.51)	0.97 (0.56–1.27)	< 0.0001
Total cholesterol (mg/dL)	200.74 ± 6.24	189.05 ± 6.11	0.05
HDL-c (mg/dL)	47.53 ± 1.99	44.19 ± 2.10	0.09
LDL-c (mg/dL)	132.70 (100.20–158.90)	121.95 (92.80–150.30)	0.93
Triglycerides (mg/dL)	113.00 (83.00–157.00)	90.00 (72.00–115.00)	< 0.0001
Uric acid (mg/dL)	4.93 ± 0.20	5.22 ± 0.22	0.15

Values as mean ± standard error for normal distribution or as median (interquartile range) for not normal distribution

**p* value refers to differences between pre- and post-IGB treatment (repeated measures ANOVA)

HOMA-IR, homeostatic model assessment of insulin resistance; *HDL-c*, high-density lipoprotein cholesterol; *LDL-c*, low-density lipoprotein cholesterol

in total cholesterol levels. In addition, there was an improvement in glucose, insulin, and HOMA-IR that was significantly associated with % TWL. In line with these findings, other studies showed an improvement in glucose, insulin resistance, and/or lipid profile after IGB-induced weight loss [17, 31, 68].

The present study has as limitations the small sample size, the heterogeneous characteristics at baseline, the absence of a control group, and the lack of evaluation of the subjects at least 6 months after IGB removal.

Conclusion

The present study suggests that 6 months of IGB treatment in obese individuals reduce serum levels of leptin and hs-CRP and improve insulin resistance, lipid profile, and adiponectin/leptin ratio. Therefore, weight loss induced by IGB, similarly to weight loss induced by other methods, has beneficial effects on inflammatory status and metabolic profile which can decrease cardiovascular risk.

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Compliance with Ethical Standards

Conflict of Interest The authors declare they have no conflict of interest.

Informed Consent Informed consent was obtained from all individual participants included in the study.

Human Rights The study was reviewed and approved by the Committee on Ethics and Research of Pedro Ernesto University Hospital / Rio de Janeiro State University. All procedures performed in the study were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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