Potential Additional Effect of Omentectomy on Metabolic Syndrome, Acute-Phase Reactants, and Inflammatory Mediators in Grade III Obese Patients Undergoing Laparoscopic Roux-en-Y Gastric Bypass

A randomized trial

MIGUEL F. HERRERA, MD, PHD[1]
JUAN PABLO PANTOJA, MD[1]
DAVID VELÁZQUEZ-FERNÁNDEZ, MD, PHD[2]
JAVIER CABIEDES, PHD[3]
CARLOS AGUILAR-SALINAS, MD[4]
EDUARDO GARCÍA-GARCÍA, MD[5]
ALFREDO RIVAS, MD[6]
CHRISTIAN VILLEDA, MD[1]
DIEGO F. HERNÁNDEZ-RAMÍREZ, MSC[2]
ANDREA DÁVILA, MD[6]
AARÓN ZARAÍN, MD[1]

OBJECTIVE — To assess the additional effect of sudden visceral fat reduction by omentectomy on metabolic syndrome, acute-phase reactants, and inflammatory mediators in patients with grade III obesity (G-III O) undergoing laparoscopic Roux-en-Y gastric bypass (LRYGB).

RESEARCH DESIGN AND METHODS — Twenty-two patients were randomized into two groups, LRYGB alone or with omentectomy. Levels of interleukin-6, C-reactive protein, tumor necrosis factor-α, leptin, adiponectin, glucose, total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides, as well as clinical characteristics, were evaluated before surgery and at 1, 3, 6, and 12 months after surgery. Results were compared between groups.

RESULTS — Baseline characteristics were comparable in both groups. Mean operative time was significantly higher in the group of patients who underwent omentectomy ($P < 0.001$). Median weight of the omentum was 795 ± 341 g. In one patient, a duodenal perforation occurred at the time of omentectomy. BMI, blood pressure, glucose, total cholesterol, LDL, and triglycerides significantly improved in both groups at 1, 3, 6, and 12 months of follow-up when compared with basal values. However, there were no consistent statistically significant differences among the groups in terms of metabolic syndrome components, acute-phase reactants, and inflammatory mediators.

CONCLUSIONS — Omentectomy does not have an ancillary short-term significant impact on the components of metabolic syndrome and does not induce important changes in the inflammatory mediators in patients undergoing LRYGB. Operative time is more prolonged when omentectomy is performed.

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Metabolic syndrome is an important comorbid condition of grade III obesity (G-III O). There are several definitions for metabolic syndrome, which include a constellation of metabolic abnormalities such as glucose intolerance (type 2 diabetes, impaired glucose tolerance, or impaired fasting glycaemia), insulin resistance, central obesity, dyslipidemia, and hypertension, all well-documented risk factors for cardiovascular disease (1–3).

Surgical weight reduction improves metabolic syndrome in patients with G-III O. In particular, the Roux-en-Y gastric bypass has shown in addition to endurable weight reduction (4) a significant positive impact on the components of the syndrome (5). The benefit from weight reduction in this group of patients has been related to changes in the plasmatic levels of adipocytokines (i.e., tumor necrosis factor [TNF]-α, interleukin [IL]-6, retinol binding protein-4, among others). These are produced in both visceral and subcutaneous fat, which are believed to participate in the pathophysiology of the obesity-related metabolic comorbidities (6–8).

Visceral adiposity has been associated with insulin resistance, glucose intolerance, dyslipidemia, and cardiovascular disease. The mechanism by which increased central adiposity causes hepatic insulin resistance is unclear, but there are at least two major hypotheses to explain it. The "portal hypothesis" suggests that an increased delivery of free fatty acids by visceral adipose tissue to the portal vein

From the [1]Department of Surgery, Instituto Nacional de Ciencias Médicas y Nutrición, Salvador Zubirán, Tlalpan, Mexico; the [2]Department of Immunology and Rheumatology, Instituto Nacional de Ciencias Médicas y Nutrición, Salvador Zubirán, Tlalpan, Mexico; the [3]Hap Map Laboratory, Instituto Nacional de Medicina Genómica, Mexico City, Mexico; the [4]Department of Endocrinology and Metabolism, Instituto Nacional de Ciencias Médicas y Nutrición, Salvador Zubirán, Tlalpan, Mexico; [5]The Obesity Clinic, Instituto Nacional de Ciencias Médicas y Nutrición, Salvador Zubirán, Tlalpan, Mexico; and [6]The School of Medicine, Universidad Nacional Autónoma de México, Mexico City, Mexico.
Corresponding author: Miguel F. Herrera, herreram@quetzal.innsz.mx.
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causes insulin resistance. The alternative hypothesis implies that secretion of adipokines may induce hepatic insulin resistance (9). Removal of subcutaneous fat has failed to improve metabolic syndrome. This was proved by Klein et al. (10) who performed massive liposuction to a group of patients with metabolic syndrome, with very little or no impact on the components of the syndrome. Thörne et al. (11), on the other hand, in a randomized controlled trial, performed omentectomy to a group of patients who underwent adjustable gastric banding. The authors found a positive impact of omentectomy on metabolic syndrome in the long term (11).

RESEARCH DESIGN AND METHODS

— We conducted a prospective, controlled, randomized pilot trial to evaluate the additional effect of sudden visceral fat reduction by omentectomy on metabolic syndrome in patients with G-III O undergoing laparoscopic Roux-en-Y gastric bypass (LRYGB). A total of 24 patients, with BMI >40 kg/m^2 and metabolic syndrome according to the Adult Treatment Panel III classification were invited to participate in the study (see online appendix at http://care.diabetesjournals.org/cgi/content/full/dc09-1833/DC1). They were patients who were scheduled to LRYGB performed by two of the authors (M.F.H. and J.P.P.) and were enrolled between December 2005 and October 2006. Patients were randomized at their arrival to the operating room by the surgeon using sequentially numbered sealed envelopes into two groups, LRYGB alone or LRYGB with omentectomy. The randomization sequence was computer generated and blocked in groups of eight. All patients signed an informed consent and the protocol was approved by our institutions review board.

Body weight and height were measured using a daily calibrated balance (Tanita TBF310) and BMI was estimated. Blood pressure was measured in a sitting position using a sphygmomanometer and a stethoscope after a 10-min rest period. LRYGB was performed according to the following standards: gastric pouch was constructed using the lesser curvature of the stomach. A 45-mm stapler was initially fired horizontally 2–3 cm below the gastroesophageal junction, and then two to three additional fires toward the angle of His and against a 32 French intragastric tube completed the vertical transection.

Table 1—*General characteristics of the studied groups*

	Omentectomy + LRYGB	LRYGB	*P*
Age (years)	36.8 ± 12.9	39.8 ± 11.1	0.56
Sex (male/female)	3/8	1/10	0.58
BMI (kg/m^2)	44.9 ± 3.1	44.5 ± 4.3	0.79
Systolic blood pressure (mmHg)	131.8 ± 9.8	134.6 ± 6.9	0.46
Diastolic blood pressure (mmHg)	85.5 ± 7.9	89 ± 3.9	0.20
Glucose (mg/dl)	99.8 ± 8.6	104.9 ± 35.6	0.65
2-h glucose oral glucose tolerance test (mg/dl)	172.6 ± 53.9	170 ± 39.8	0.92
Insulin (μU/μl)	21.64 ± 9.8	22.0 ± 12.4	0.94
Cholesterol (mg/dl)	182.8 ± 36.6	192.3 ± 55.4	0.64
Triglycerides (mg/dl)	180.4 ± 83.8	292 ± 248.3	0.18
HDL (mg/dl)	38.6 ± 9.1	35.5 ± 10.1	0.44
LDL (mg/dl)	106.8 ± 32.3	126.6 ± 49.9	0.30

Data are means ± SD.

Lengths of the biliopancreatic and alimentary limbs were ~50 and 150 cm, respectively. An antecolic and antegastric gastrojejunostomy, 1.0–1.5 cm in size, was hand sewn, and the jejunojejunostomy was completed in a latero-lateral fashion using one fire of a 45-mm lineal stapler with hand-sewn closure of the common enterotomy. All defects were closed using nonabsorbable sutures. For the omentectomy, the greater omentum was divided in the middle from the free edge to the colonic margin using ultrasonic energy. Attachments between the omentum and the transverse colon were dissected. The omentum was detached from the stomach, transecting the vessels between the right gastroepiploic vessels and the greater curvature of the stomach. Once the omentum was freed from the stomach, the duodenum, and the lower pole of the spleen, it was extracted from the abdominal cavity in a sterile plastic bag.

An oral glucose tolerance test using a dose of 75 g of glucose after an overnight fasting (12 h) period was performed in all patients. Glucose and serum insulin concentrations were measured in blood samples obtained 10 min before and 0, 60, and 120 min after the glucose intake. Diabetes was diagnosed when glucose was ≥200 mg/dl 2 h postload. Impaired fasting glycemia was considered when the basal glucose level was 100–125 mg/dl, and impaired glucose tolerance was diagnosed when the 2-h postload glucose was between 140 and 199 mg/dl.

Blood samples for plasma glucose, insulin, lipid profile (total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides), blood urea nitrogen, creatinine, and liver function tests (total bilirubin, transaminases, and albumin) were taken after a 12-h fasting period before surgery and at 1, 3, 6, and 12 months after surgery. The homestasis model assessment of

Table 2—*Changes in comorbidities between groups at the end of the study period*

	Before surgery			12 months after surgery		
Comorbidities	Total	LRYGB	Omentectomy + LRYGB	Total	LRYGB	Omentectomy + LRYGB
Impaired glucose tolerance	11/22 (50)*	7	4	1 (4.5)	0	0
Diabetes	10/22 (45.5)*	4	6	1 (4.5)	0	1
Hypertension	16/22 (72.7)*	8	8	3 (13.6)	2	1
Dyslipidemia	16/22 (72.7)*	6	10	2 (9.1)	1	1

Data are *n* (%), unless otherwise indicated. *$P < 0.001$ when compared with 12 months after surgery (Fisher exact test). Comparative analysis of LRYGB and Omentectomy + LRYGB before and after surgery = NS (Fisher exact test). Dyslipidemia was considered when measurements of triglycerides and/or cholesterol were above the upper normal limit values or when patients were under medical treatment with hypolipemiant drugs.

Table 3—*Comparative analysis of metabolic variables between groups*

	Before surgery		1 month	
Variables	Omentectomy + LRYGB	LRYGB	Omentectomy + LRYGB	LRYGB
BMI (kg/m^2)	45.0 ± 3.1	44.5 ± 4.3	−10.9 (−14.7 to −7.1)	−10.1 (−13.5 to −6.7)
SBP (mmHg)	131.8 ± 9.8	134.6 ± 6.9	−7.6 (−40.2 to 25.0)	−14.9 (−33.3 to 3.6)
Glucose (mg/dl)	99.8 ± 8.6	104.9 ± 35.6	−3.2 (−18.6 to 12.3)	−15.8 (−40.9 to 9.2)
Insulin (μU/μl)	21.6 ± 9.8	22.0 ± 12.4	NA	NA
Homeostasis model assessment of insulin resistance	5.2 ± 2.5	6.6 ± 5.0	NA	NA
Cholesterol (mg/dl)	182.8 ± 36.6	192.3 ± 55.4	−28.4 (−37.5 to −19.3)	−16.9 (−24.4 to −9.5)*
Triglyceride (mg/dl)	180.9 ± 83.8	292 ± 248.3	−33.9 (−56.8 to −11.1)	−48.7 (−105.8 to 8.4)
HDL (mg/dl)	38.6 ± 9.1	35.5 ± 10.1	−19.7 (−31.9 to −7.3)	−11.5 (−25.9 to 2.8)
LDL (mg/dl)	106.8 ± 32.3	126.6 ± 49.9	−29.3 (−43.8 to −14.8)	−19.7 (−33.9 to −33.9)*

Data after surgery are means ± SD or percent of change from basal (95% CI). Minus signs denote decreases and plus signs increases. *$P \leq 0.05$.

insulin resistance was calculated and used as a measure of insulin resistance.

Adiponectin, TNF-α, leptin, C-reactive protein (CRP), and IL-6 were measured at baseline and at 3, 6, and 12 months after LRYGB. Samples were collected using standardized phlebotomy procedures. Standard handling and processing was used. Serum and plasma were separated by sample spinning. Specimens were immediately aliquoted, frozen, and stored at −80°C. For the multiplex analysis, we used the xMAP technology (Luminex), which combines the principle of a sandwich immunoassay with fluorescent bead–based technology (12). The xMAP serum assay for CRP, adiponectin, leptin, IL-6, and TNF-α was done in 96-well microplate format according to the protocol of LINCOplex kits (Linco, Millipore, Billerica, MA). The analysis was performed using five-parametric-curve fitting. Interassay variabilities for individual cytokines were in the range of 7.0–11.0% and intra-assay variabilities were in the range of 8.2–14.6%. The limit of detection for PCR, TNF-α, and IL-6 was 1 pg, for adiponectin 3.2 pg, and for leptin 16 pg.

Statistical analyses were conducted using the Statistical Package for the Social Sciences software (SPSS 16.0). Clinical characteristics were compared among patients with and without omentectomy using Student *t* test. Changes in body composition, metabolic syndrome markers, and adipocytokines were assessed by Mann-Whitney *U* test. Correlations between changes in BMI and adipocytokines were determinate by the Spearman coefficient. All reported *P* values are two sided, and $P < 0.05$ was considered statistically significant.

RESULTS — Of 24 patients, 1 patient of each group was lost for follow-up (drop-out) after surgery. There were no significant differences between groups in terms of age, sex, BMI, and laboratory tests (Table 1).

Mean operative time was 4 h 31 min and 3 h 6 min, respectively, in the group of patients who underwent and did not undergo omentectomy ($P < 0.001$). Mean weight of the resected omentum was 795 ± 341 g. There was one complication related to the omentectomy, a duodenal perforation that required laparoscopic revision for suturing.

As shown in Table 2, there was a significant impact of bariatric surgery on the components of metabolic syndrome at 1-year follow-up. However, there were no differences between groups. BMI, systolic blood pressure, diastolic blood pressure, glucose, insulin, total cholesterol, LDL, and triglycerides also had a significant change from basal values at 1, 3, 6, and 12 months follow-up. However, there were no statistical differences among the groups in the majority of the analyzed variables (Table 3). Isolated differences at some points of the study were found. LDL values were significantly lower 1 month after surgery in the group who underwent omentectomy, which may be related to the fact that preoperative values of LDL in this group were also lower. As can be seen in Table 4, behavior of adipocytokines throughout the studied period was also very similar in both groups.

The relationship between weight loss and changes in the studied variables was assessed in the whole group, and a negative correlation was seen between BMI and IL-6 at 3 months ($P = 0.013$) and BMI and TNF-α at 12 months ($P = 0.017$). When we analyzed the data by group, there was no correlation between the behavior of the BMI and the analyzed variables, except for IL-6 and BMI at 3 months ($P = 0.027$) (RYGBP + omentectomy), TNF-α and BMI at 12 months ($P =$

Table 4—*Comparative analysis of adipocytokines between groups*

	Before surgery		3 months	
	Omentectomy + LRYGB	LRYGB	Omentectomy + LRYGB	LRYGB
CRP (μg/ml)	64.5 ± 75.4	69.2 ± 57.2	−62.2 (−118.2 to −5.6)	−76.2 (−124.9 to −27.5)
TNF-α (pg/ml)	5.2 ± 3.3	5.7 ± 2.7	−1.9 (−40.4 to 36.5)	−12.3 (−36.8 to 10.5)
IL-6 (pg/ml)	4.8 ± 9.5	8.9 ± 16.9	+247.9 (−264.6 to 762.5)	+24.7 (−20.2 to 70.8)
Leptin (ng/ml)	55.4 ± 30	103.6 ± 153.1	−18.2 (−57 to 20.6)	−56.9 (−129.8 to 36.1)
Adiponectin (ng/ml)	10.7 ± 5.1	9.5 ± 4.8	+42.1 (8.4–76.6)	+66.3 (32.6–101.1)

Data after surgery are means ± SD or percent of change from basal (95% CI). Minus signs denote decreases and plus signs increases. All comparisons P = NS.

Table 3—*Continued*

3 months		6 months	
Omentectomy + LRYGB	LRYGB	Omentectomy + LRYGB	LRYGB
−18.9 (−22.4 to −14.9)	−18.7 (−23.1 to −14.4)	−26.7 (−32.7 to −23.2)	−25.6 (−29.7 to −21.3)
−10.9 (−19.8 to −2.0)	−14.9 (−109.2 to 79.6)	−17.7 (−21.2 to −13.6)	−8.4 (−17.4 to 0.7)*
−12.3 (−19.3 to −5.3)	−13.8 (−37.7 to 10.1)	−12.1 (−20.5 to −3.7)	−18.5 (−38.6 to 1.7)
−64.8 (−111.6 to −18.5)	−51.8 (−96.8 to −6.8)	−57.4 (−88.0 to −26.9)	−14.5 (−115.0 to 125.9)
−69.2 (−121.2 to −19.2)	−54.5 (−106.1 to 1.5)	−59.6 (−155.8 to −23.1)	−36.4 (−157.6 to 83.3)
−21.6 (−26.4 to −16.8)	−12.7 (−21.7 to −3.8)	−18.0 (−26.5 to −16.8)	−10.6 (−21.6 to 0.5)
−39.4 (−61.1 to −18.2)	−52.0 (−109.0 to 5.0)	−38.7 (−68.3 to −9.0)	−45.6 (−109.9 to 19.0)
−9.8 (−24.1 to 4.1)	+0.3 (−8.2 to 8.7)	−2.3 (−18.4 to 13.7)	+15.5 (5.6–25.1)
−28.8 (−50.3 to −7.3)	−14.2 (−22.4 to −6.0)	−17.2 (−35.5 to 1.1)	−33.8 (−65 to −2.6)

0.022) (RYGBP alone), triglycerides and BMI at 1 month ($P = 0.033$) (RYGBP + omentectomy), and triglycerides and BMI at 3 months ($P = 0.039$) (RYGBP + omentectomy).

CONCLUSIONS — Abdominal obesity has been closely associated to metabolic syndrome (1,3). Several studies have demonstrated that surgically induced weight reduction improves risk factors from metabolic syndrome in morbidly obese patients and that metabolic syndrome can be controlled by bariatric surgery in up to 95% of patients within the first year (13).

As it could be expected, when we analyzed all patients together, there was a significant change in most components of metabolic syndrome after surgery. A meta-analysis published by Buchwald et al. (4), as well as other clinical studies (14), had already shown the positive benefit of bariatric surgery not only in terms of weight loss but also for control of comorbid conditions.

Both subcutaneous and visceral fat are responsible for the production of adipocytokines, such as TNF-α, IL-6, and adiponectin (15,16). It has been shown that they are markers of the inflammatory state in metabolic syndrome (17). There is also evidence that surgically induced weight loss has an important effect on adipocytokines, reducing proinflammatory CRP, TNF-α, and IL-6 and increasing anti-inflammatory adiponectin (18).

In terms of the effect of parietal fat reduction on the components of metabolic syndrome, Klein et al. (9) studied the effect of massive liposuction on metabolic syndrome at 10–12 weeks on 15 obese women, 8 with normal glucose tolerance and 7 with diabetes. The amount of fat removed was 9.1 ± 3.7 kg and 10.5 ± 3.3 kg in the nondiabetic and diabetic groups, respectively. Despite a statistically significant reduction in BMI in both groups, there were no significant changes in fasting glucose, total cholesterol, HDL, LDL, and triglycerides 10–12 weeks after liposuction. In a similar way, there were no significant changes in adipocytokines 10–12 weeks after the procedure.

If the sudden reduction of parietal fat does not have a prevalent role in the components of metabolic syndrome, sudden reduction of visceral fat may be the evident target. Experimental studies in animals have demonstrated an impact of removal the visceral fat on insulin resistance. Barzilail et al. (19) found that surgical removal of selective intra-abdominal fat depots (perinephric and epididymal) leads to a marked increase in the hepatic effect of insulin and suggested that it could regulate gene expression in subcutaneous adipose tissue in elderly obese rats. Pitombo et al. (20) found the same effect in rats with diet-induced diabetes and their study also suggested favorable modulation of adipokines. Kim et al. (21) evaluated the role of visceral and subcutaneous fat tissue on both insulin sensitivity and lipid metabolism. These authors proved that visceral fat has a stronger effect on insulin sensitivity and concentration of free fatty acids in the liver than subcutaneous fat in rats. Lottati et al. (22) studied the metabolic impact of omentectomy in nonobese dogs and found that resection of visceral fat had a positive effect on insulin sensitivity.

In this study, we assessed if an additional effect could be obtained from sudden visceral fat reduction (by omentectomy) on the severity of the metabolic syndrome components, acute-phase reactants, and inflammatory mediators concentrations in patients with G-III O undergoing LRYGB. Omentectomy does not seem to have an ancillary short-term significant impact on the components of metabolic syndrome and does not induce

Table 4—*Continued*

6 months		12 months	
Omentectomy + LRYGB	LRYGB	Omentectomy + LRYGB	LRYGB
−65.6 (−152.1 to −20.8)	−75.9 (−127.7 to −23.8)	−80.0 (−196.7 to 36.7)	−86.27 (−143.4 to −23.8)
−51.9 (−98.1 to −5.8)	−17.5 (−40.4 to 3.5)	+11.05 (−80.8 to 107.7)	−14.0 (−75.4 to 43.9)
+104.2 (−285.4 to 493.8)	−23.6 (−93.3 to 46.1)	+35.4 (−43.8 to 116.7)	−34.8 (−132.6 to 62.9)
−9.7 (−40.1 to 20.6)	−36.5 (−137.0 to 64.0)	+16.8 (−30.9 to 64.4)	−21.8 (−145.8 to 102.0)
+74.8 (40.2–109.3)	+87.4 (46.3–127.4)	+80.4 (30.8–129.0)	+69.5 (−3.2 to 144.2)

Table 3—*Continued*

12 months	
Omentectomy + LRYGB	LRYGB
−32.4 (−37.8 to −27.3)	−31.0 (−37.5 to −24.5)
−12.7 (−19.1 to −6.1)	−7.4 (−15.4 to 0.4)
−11.7 (−21.1 to −2.2)	−15.2 (−36.6 to 6.3)
−19.9 (−169.9 to130.1)	−27.7 (−101.4 to 45.5)
−36.5 (−155.8 to 84.6)	−50.0 (−128.8 to 28.8)
−13.9 (−22.0 to −5.9)	−14.8 (−29.3 to −0.3)
−44.9 (−69.6 to −20.3)	−64.1 (−124.3 to −3.9)
+11.1 (0.3–21.8)	+27.0 (13.5–40.6)
−11.2 (−25.9 to 3.4)	−27.4 (−51.9 to −2.8)

important changes in the inflammatory mediators in patients undergoing LRYGB.

In our study, values of LDL 3 and 6 months after surgery were significantly lower in the group of patients who underwent omentectomy in addition to the LRYGB, and there was also a tendency toward a higher reduction of cholesterol values in the same group of patients. On the other hand, there were no significant differences on adipocytokine levels throughout the studied period. Observed differences in the behavior of LDL and total cholesterol may not be of clinical relevance, since values were within the normal ranges in both groups from the first postoperative month.

What are the potential causes of the lack of significant differences on the postoperative behavior of metabolic syndrome components between the studied groups? An important limitation of our study is the small number of studied patients. For example, homeostasis model assessment of insulin resistance and insulin levels showed a trend toward more improvement in patients undergoing LRYGB and omentectomy. An overt difference may have been obscured by the small number of patients (type II error). However, if the differences are so small that a very large number of patients would be required to make them evident, they may not be sufficient to justify the use of omentectomy. This is particularly true if we consider that there is an important impact of weight reduction alone on metabolic syndrome from the first month after LRYGB and that omentectomy is a time-consuming procedure that may lead to potentially lethal complications. An additional cause could be that in all experimental studies omentectomy was compared against a sham operation, whereas in human studies it has been compared with patients undergoing a bariatric operation.

In terms of the impact of omentectomy on adipocytokines, another aspect that needs to be considered is that any surgical procedure involves an inflammatory reaction that may last for few weeks. If the expected impact of omentectomy on inflammatory markers was more likely to occur soon after surgery, surgical stress may mask the real effect. As an additional aspect related to the minimum or lack of impact of omentectomy on adipocytokines, it is worth mentioning that the omentum accounts for a large proportion of the intraabdominal visceral fat. However, there is also fat in other places such as the liver and around the kidneys that persists in both groups.

In a previous study, Thörne et al. (10) compared the results of adjustable gastric banding (AGB) versus AGB accompanied by omentectomy in 37 morbidly obese patients (18 with AGB and 19 with AGB + omentectomy) who completed a 2-year follow-up. Weight of the resected omentum was similar to that of our patients. The authors found a significantly higher weight reduction in the group of patients with omentectomy (9 ± 6 vs. 13 ± 5 kg/m^2, $P = 0.049$) and significantly lower values of fasting glucose (0.7 ± 0.7 vs. 1.8 ± 0.8 mmol/l, $P = 0.04$). A tendency to a higher reduction of total cholesterol values was also reported. A potential limitation to interpret their results is that the group of patients who underwent omentectomy also had a higher weight loss during the study period. Csendes et al. (23) also performed a comparative evaluation of patients with morbid obesity who underwent LRYGB alone or combined with omentectomy. Two years after surgery, they found no differences between groups in terms of BMI, serum glucose, insulin, total cholesterol, triglycerides, and the behavior of blood pressure.

In this study, where patients undergoing LRYGB and the combination of LRYGB with omentectomy had similar weight loss, we were unable to demonstrate a significant additional impact of omentectomy on the components of metabolic syndrome, acute-phase reactants, and inflammatory mediators. Our current results and those from other published series in humans suggest that the addition of omentectomy to the LRYGB in patients with G-III O and metabolic syndrome is not justified.

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