Published in final edited form as:
Surg Obes Relat Dis. 2013 ; 9(2): . doi:10.1016/j.soard.2011.09.027.

Omentectomy added to Roux-en-Y gastric bypass surgery: A randomized, controlled trial

TTroy H. Dillard, **Jonathan Q. Purnell**, **Mark D. Smith**, **William Raum**, **Dennis Hong**, **Jamie Laut**, and **Emma J. Patterson**
Department of Medicine, Division of Endocrinology, Diabetes, and Clinical Nutrition (T.H.D., J.Q.P.); Oregon Health & Science University, Portland, OR; Department of Surgery (M.S., E.P., J.L.), Legacy Good Samaritan Hospital, Portland, OR; Department of Surgery, St. Joseph's Hospital, McMaster University, Hamilton, Ontario, Canada (D.H.)

Abstract

BACKGROUND—Visceral adipose tissue (VAT) predicts incipient diabetes mellitus and cardiovascular disease. Human data is mixed regarding the benefits of selective VAT reduction.

OBJECTIVES—We investigated omentectomy added to laparoscopic Roux-en-Y gastric bypass (LRYGB) on glucose homeostasis and lipids, inflammatory markers and adipokines after 90-days in non-diabetic patients.

SETTING—Legacy Good Samaritan Hospital and Oregon Health & Science University in Portland, Oregon.

METHODS—A single-blinded, randomized study of LRYGB plus omentectomy *vs.* LYRGB alone in 28 subjects (7 male, 21 female). Groups were matched at baseline for gender, age, and body mass index (BMI). Eligibility included age ≥ 18 years old, a body mass index (BMI) ≥ 40 and < 50 kg/m^2 without co-morbid conditions or BMI ≥ 35 and < 50 kg/m^2 with co-morbid conditions. The primary outcome measures were changes in fasting plasma glucose, insulin and HOMA-IR. Secondary measures were BMI and levels of hs-CRP, TNF-α, interleukins, total and HMW adiponectin, fibrinogen, and PAI-1.

RESULTS—After surgery, BMI decreased significantly in both groups and was not different at follow-up. While many outcome parameters improved with weight loss in both groups post-operatively, only the omentectomy group experienced statistically significant decreases in fasting glucose (p<0.05), total (p=0.004) and VLDL (p=0.001) cholesterol, and an increase in the HMW:total adiponectin ratio (p=0.013).

CONCLUSIONS—Omentectomy added to a LRYGB results in favorable changes in glucose homeostasis, lipid levels, and adipokine profile at 90-days. These data support the hypothesis that selective ablation of VAT conveys metabolic benefit in non-diabetic humans.

Keywords

omentectomy; omentum; obesity; bariatric surgery; Roux-en-Y; gastric bypass surgery; visceral adipose; intra-abdominal adipose; adiponectin; high-molecular weight adiponectin

Corresponding author's ground mail address, telephone number and fax number: Emma Patterson, MD, Oregon Weight Loss Surgery, LLC, Legacy Good Samaritan Hospital, 1040 NW 22nd Ave, Suite 500, Portland, Oregon 97210, Voice: 503 227-5050, Fax: (503) 227-2462, EPatterson@OregonWeightLossSurgery.com.

ClinicalTrials.gov listing: NCT00552942

INTRODUCTION

Obesity is a global health threat [(1)]. Research has elucidated a complex relationship between adiposity and expression of co-morbid diseases. For example, excess visceral adipose tissue (VAT) predicts type 2 diabetes mellitus (DM) and cardiovascular disease (CVD) independent of total body fat or subcutaneous adipose tissue (SAT)[(2,3)] and contributes to the development insulin resistance, dyslipidemia, and inflammation[(4,5)]. The mechanisms are poorly understood but candidates include: direct signaling between VAT and liver via induction of local and systemic inflammation or changes in flux of lipids and intermediates of lipid metabolism, and alterations in adipokine secretion[(6,7)]. Adiponectin, in animal models, demonstrates a causal role in maintaining insulin sensitivity[(8)]. In humans, decreased adiponectin levels are associated with development of type 2 DM[(9,10)] and levels are negatively correlated with visceral adiposity[(11)]. Adiponectin is lower in obese and insulin resistant subjects and increases with weight loss[(12)]. Decreases in high-molecular weight (HMW) adiponectin are believed more important for expression of insulin resistance, diabetes mellitus and cardiovascular disease than alterations in total adiponectin[(13,14)].

Several studies have focused on the metabolic impact of reduction in total fat mass versus regional adipose. Selective reduction in SAT through “large volume” liposuction had no impact on glucose and lipid parameters in humans[(15)]. On the other hand, omentectomy resulted in marked improvements in metabolic parameters in multiple animal studies[(16,17,18,19)]. Human studies of omentectomy have yielded mixed results. One study showed significant improvements in oral glucose tolerance, insulin sensitivity and fasting glucose and insulin in subjects who underwent omentectomy with laparoscopic gastric banding (LAGB) compared to LAGB alone[(20)]. Other studies, adding omentectomy to LRYGB, failed to demonstrate a metabolic advantage to omentectomy[(21,22,23,24)]. Many of these studies included small numbers of subjects, mixtures of patients with and without diabetes, or included long-term follow up endpoints when much larger weight loss had occurred proportional to the amount of VAT removed. Given early positive results by Thörne and colleagues and limitations in existing LRYGB studies, we sought to determine if omentectomy added to LRYGB confers additive metabolic benefit on inflammatory markers, glucose homeostasis, and adipokine levels compared to LRYGB alone at an early time point (90-days) in a larger group of subjects.

MATERIALS AND METHODS

Based on results by Thorne and colleagues[(20)], a sample size of 17 patients per group was calculated using a 2-tailed Student’s T-test with an alpha of 5% and sigma of one to achieve a power of 80%. After completion of enrollment between July 2006 and October 2009, samples for a total of 28 patients, age 27 to 68 years old were available for analysis. Samples from 15 patients (3 male, 12 female) randomized to LRYGB with omentectomy and 13 patients (4 male, 9 female) randomized to LRYGB alone were analyzed. Two subjects from the control arm did not have data for the 90-day follow-up due sample handling and insufficient quantities. Results are reported for 26 subjects for whom follow-up data is available. The target population included all morbidly obese patients undergoing LRYGB at Legacy Health System and patients were enrolled by study coordinators within these institutions. All patients were enrolled at a single center, Legacy Good Samaritan Hospital, Portland, OR. Inclusion criteria included age ≥ 18 years old; a body mass index (BMI) ≥ 40 and < 50 kg/m^2 without co-morbid conditions or BMI ≥ 35 and < 50 kg/m^2 with co-morbid conditions; patients accepted into the obesity surgery program at Legacy Health System; and self-selection for laparoscopic gastric bypass. Exclusion criteria included patients < 18 years old, patients who elected to have bariatric procedures other than laparoscopic gastric bypass, inability to give informed consent, co-morbid conditions which preclude surgery, previous

major pelvic surgery including hysterectomy, bloodless surgeries, self-pay status and known type 1 diabetes mellitus. No patients with known type 2 diabetes mellitus were enrolled due to institutional screening which favored their recruitment into a different and simultaneous clinical trial. For three days prior to surgery and testing, subjects were advised to drink only liquids. After surgery subjects were advised to maintain a diet low in concentrated calories (high fat, processed carbohydrates). Randomization occurred on the morning of surgery after induction of anesthesia using sequentially numbered, opaque, sealed envelopes. Patients were blinded to their treatment allocation. One subject from the control group was unblinded post-operatively when an operating room nurse inadvertently informed the patient's family that omentectomy had not been performed. It was decided to keep the patient in the study. Results were not meaningfully altered when performed with and without this subject's data. The institutional review boards at Legacy Good Samaritan Hospital and Oregon Health & Science University approved the protocol and all patients signed an informed consent prior to study entry. The trial was listed on clinicaltrials.gov (NCT00552942).

LRYGB was performed according to standardized technique. A lesser curvature-based 15-mL gastric pouch was created and calibrated around a trans-oral gastric balloon. An ante-gastric, ante-colic circular stapled gastrojejunostomy was constructed. Roux-limb and biliopancreatic limbs were measured at 100cm and jejunojejunostomy constructed. The common channel length was not recorded. During formation of the gastrojejunostomy, a pathway in the greater omentum was created from the free edge to approximately 1cm from the transverse colon. If the patient was randomized to omentectomy, the omentum was fully dissected free from the transverse colon and greater curvature of the stomach, from medial to lateral, beginning at the omental pathway. Then each side of the omentum was placed in an endocatch bag and removed from the abdomen via a laparoscopic trocar site and weighed.

After a fast of at least 8 hours, subjects had blood taken at pre-operatively and 90 days post-operatively. Specimens were immediately spun, separated, aliquoted and frozen at −70° C until analysis. Glucose was assayed by glucose oxidase method (Stanbio Laboratory, Boerne, TX). Insulin and hsCRP were analyzed via the Immulite system (Siemens Diagnostics, Deerfield, IL). ELISA testing was used to quantify TNF-α, IL1-β, IL-6, IL-8, and IL-10 (R&D Systems, Minneapolis, MN), PAI-1 (Invitrogen, Camarillo, CA), fibrinogen (ALPCO Diagnostics, Salem, NH) and HMW adiponectin (Millipore, Billerica, MA). Total adiponectin was measured by RIA (Millipore, Billerica, MA). Lipids were assessed by photometric assay (Olympus AU640e, Beckman Coulter, Brea, CA).

Weight loss is expressed as %Excess Weight Loss (%EWL), using the middle of the 1983 Metropolitan Life Insurance tables for median frame; %Weight Lost (%WL); and %Excess BMI Lost (%EBMIL) with excess >25 kg/m^2. Statistics were performed using SigmaStat (v. 3.5, Aspire Software International, Ashburn, VA). Assessment of changes within groups and between groups was performed using Student's *t*-test or Mann-Whitney *U* test, as appropriate. Insulin resistance was estimated using the homeostasis insulin resistance index (HOMA-IR)[25]. All reported *P* values are two sided, and $P < 0.05$ was considered statistically significant.

RESULTS

Treatment and control groups were closely matched at baseline for gender, age, BMI, and laboratory tests (Tables 1 and 2) except for slightly higher HMW:total adiponectin ratio ($p<0.05$) and slightly lower IL-1β ($p=0.007$) in the treatment vs. control group (Table 2). The mean operative time was 153 min for the treatment group and 135 min for the control group ($p=0.37$). The mean weight of the resected omentum was 505 (range: 200–865) gm.

There were no intraoperative complications related to omentectomy. Post-operative complications included two patients in the omentectomy group (gastroenterostomy stenosis) and one patient in the control group (urinary retention).

Both groups experienced significant weight loss compared to baseline with no statistically significant difference between the groups at 90 days (Table 1). Average percent excess weight loss (%EWL) was 38% in both groups (p=0.93). Average percent weight loss was 23% and 17% in the treatment and control groups, respectively (p=0.38). Percent excess BMI lost (%EBMIL) was 45% and 43% in the treatment and control groups, respectively (p=0.77). Although both groups experienced decreases in fasting glucose, this was statistically significant only in the omentectomy group ($p<0.05$). There was no significant change in insulin levels in either group (p=0.21 for the omentectomy group; p=0.06 for the control group). The pre-operative insulin level in the control group was influenced by a single outlier value (208 μIU/mL). Excluding this outlier decreased the mean ± SD pre-operative insulin level to 23.1 ± 20.4 μIU/mL and a post-operative level to mean of 11.7 ± 7.3 μIU/mL, a difference that remained not statistically significant (p=0.16).

Both groups experienced significant decreases in triglyceride levels, but only the omentectomy group had significant reductions in total (p=0.004) and VLDL (p=0.001) cholesterol. Although adiponectin and HMW adiponectin increased significantly in both groups, only the omentectomy group showed a statistically significant increase in the HMW:total adiponectin ratio (p=0.013) (Table 2). Finally, there were no statistically significant differences in inflammatory markers between group, except for IL-10 levels (a difference which persisted from baseline) and a significant decrease in PAI-1 levels in the control group (p=0.035) (Table 2).

DISCUSSION

Visceral adiposity is strongly linked to dysregulation of glucose and lipid metabolism and risk for CVD[2,3]. Several studies link increased VAT with decrements in serum levels of adiponectin and unfavorable alterations in the ratios of adiponectin multimers. Evidence supports a role for adiponectin in maintenance of normal glucose and lipid homeostasis and suggests that changes in the HMW:total adiponectin ratio is an important contributor to expression of DM and CVD[12,13,14]. In the present study, we show that omentectomy, when added to LRYGB, results in statistically significant improvements in short-term glucose levels, lipid levels, and the ratio of HMW:total adiponectin, effects not seen in LRYGB surgery alone. Although the long-term clinical significance of these changes found at 90-days after omentectomy is likely negligible, this finding contributes to our understanding of the physiologic changes associated with VAT reduction on glucose metabolism and adipocyte function.

Our data are in agreement with animal studies in which omentectomy favorably impacts glucose and lipid metabolism[16,17,18,19] and a single human study[20]. In that study, 50 subjects were randomized to either LAGB alone or LAGB plus omentectomy[20]. After two years of follow-up, the omentectomy group showed significant metabolic benefits in insulin sensitivity compared to the control group. These metabolic improvements were independent of changes in BMI.

Four recent human studies have been published examining the effect of adding omentectomy to LRYGB surgery. All failed to show additive benefit on parameters of glucose metabolism. In a study by Herrera et al, no consistent statistically significant differences were noted between groups at 1, 3, 6 and 12 months follow-up in terms fasting glucose, HOMA-IR, lipid levels, total adiponectin, TNF-α, leptin, C-reactive protein and

IL-6[22]. That study enrolled fewer subjects than our study and did not include a measure of HMW adiponectin. The addition of omentectomy added an average of 85 minutes to the operative time ($P < 0.001$) and was associated with a severe complication (duodenal perforation). In a study by Csendes et al, 70 patients were randomized to a modified LRYGB surgery with and without omentectomy[21]. These investigators failed to show any additive benefit of omentectomy in terms of fasting glucose, fasting insulin levels, lipid levels or blood pressure after substantial total weight loss of 35% at 2-year follow-up. Adipokines were not assessed and no metabolic evaluations were undertaken at intermediate time points. In a study by Lima et al, 19 women, including 6 with type 2 DM, were randomized to either LRYGB with or without omentectomy and studied one month later[23]. Both groups improved with regard to insulin, glucose, and HOMA-IR and were not different by treatment group. Interestingly, non-esterified free fatty acids levels increased in both groups, suggesting ongoing metabolic adaptation to acute caloric restriction. This may have independently contributed to improvements in glucose metabolism, superseding the effect of omentectomy. Lastly, in a study by Fabrini and colleagues, 22 obese subjects were randomized to either LRYGB with or without omentectomy and underwent metabolic testing utilizing the hyperinsulinemic-euglycemic clamp with follow-up at 6 and 12 months[24]. BMI, body composition, glucose homeostasis, lipids, CRP, leptin, and total adiponectin were assessed. No significant differences were noted between groups at follow-up. Follow-up values were not obtained at 90 days in this study, however, and HMW adiponectin was not measured.

Possible explanations for the differences between our findings and those of the other LRYGB surgery studies may relate to differences in sample size and timing of follow-up. The post-operative period is characterized by a resolving hormonal milieu of stress hormones, inflammation, and dramatic caloric reduction. Each of these may have confounding effects on measures of glucose metabolism. On the other hand, proportionally large reductions in total fat mass are seen at long-term follow-up time points, especially after LRYGB surgery. Metabolic improvements resulting from these greater reductions in total fat mass (30 to 40 kg, on average) may dwarf any contribution from removal of 0.5 to 1.0 kg of omental tissue at the time of surgery. By studying our subjects at 3-months and including a larger number of patients than some previous studies, we were able to show benefits of selective visceral fat reduction by omentectomy demonstrable when enough time had elapsed for recovery from the surgery but before effects of larger total fat reductions could play a role on improved lipids and glucose metabolism.

An interesting substudy of Fabrini et al [24] examined omentectomy alone (without bariatric surgery) in a small cohort (n=7) of obese subjects with type 2 DM. This cohort underwent metabolic testing at 3-months after omentectomy, the same timepoint as our study. No assessment of adipokines was undertaken in this group but body weight, metabolic variables, and indices of insulin sensitivity, glucose effectiveness and β-cell function did not change significantly. Patients with type 2 diabetes have greater insulin resistance and impaired islet cell secretory capacity than those without diabetes[25,26,27,28] and may, therefore, have been less likely to manifest improvement in glucose metabolism in response to omentectomy than our subjects.

CONCLUSION

In conclusion, omentectomy when added to LRYGB in non-diabetics results in statistically significant reductions in fasting glucose, total and VLDL cholesterol and favorable changes in the HMW:total adiponectin ratio at 90-days, while no statistically significant reductions were noted in these parameters in a closely matched control group. These findings are not explained by differences in total weight loss or by differences in inflammatory markers. Our

data support the hypothesis that selective ablation of VAT conveys metabolic benefit in non-diabetic humans. In the context of previous reports, however, significant questions remain regarding the role of therapeutic omentectomy in humans undergoing LRYGB. Any positive effects that we demonstrate at 90-days are likely dwarfed by the metabolic improvements conferred by weight loss and favorable changes in levels of hormones such as glucagon-like peptide-1 after LRYGB during longer follow-up[29]. The greater insulin resistance and impaired insulin secretory capacity of patients with type 2 DM, as demonstrated in the Fabrini substudy[24], likely attenuate the response to omentectomy at this time point. On the other hand, subjects undergoing laparoscopic gastric banding, which accounts for over 40% of bariatric surgeries[30], may benefit from this approach and further trials are warranted in this subset of patients.

Acknowledgments

Support provided by grants from the NIH R56 DK088207 (JQP) and Research Advisory Committee Grant from Legacy Health Systems, Portland, OR. This publication was made possible with support from the Oregon Clinical and Translational Research Institute (OCTRI); grant number UL1 RR024140 from the National Center for Research Resources (NCRR), a component of the National Institutes of Health (NIH), and NIH Roadmap for Medical Research.

References

1. Misra A, Khurana L. Obesity and the metabolic syndrome in developing countries. J Clin Endocrinol Metab. 2008; 93:S9–30. [PubMed: 18987276]
2. Balkau B, Deanfield JE, Després JP, Bassand JP, Fox KA, Smith SC Jr, et al. International Day for the Evaluation of Abdominal Obesity (IDEA): a study of waist circumference, cardiovascular disease, and diabetes mellitus in 168,000 primary care patients in 63 countries. Circulation. 2007; 116:1942–51. [PubMed: 17965405]
3. Yusuf S, Hawken S, Ôunpuu S, Bautista L, Franzosi MG, Commerford P, et al. Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: a case-control study. Lancet. 2005; 366:1640–9. [PubMed: 16271645]
4. Colberg SR, Simoneau JA, Thaete FL, Kelley DE. Skeletal muscle utilization of free fatty acids in women with visceral obesity. J Clin Invest. 1995; 95:1846–53. [PubMed: 7706491]
5. Carey DG, Jenkins AB, Campbell LV, Freund J, Chisholm DJ. Abdominal fat and insulin resistance in normal and overweight women: direct measurements reveal a strong relationship in subjects at both low and high risk of NIDDM. Diabetes. 1996; 45:633–8. [PubMed: 8621015]
6. Cartier A, Lemieux I, Alméras N, Tremblay A, Bergeron J, Després JP. Visceral obesity and plasma glucose-insulin homeostasis: contributions of interleukin-6 and tumor necrosis factor-alpha in men. J Clin Endocrinol Metab. 2008; 93:1931–8. [PubMed: 18319319]
7. Xu H, Barnes GT, Yang Q, Tan G, Yang D, Chou CJ, et al. Chronic inflammation in fat plays a crucial role in the development of obesity-related insulin resistance. J Clin Invest. 2003; 112:1821–30. [PubMed: 14679177]
8. Maeda N, Shimomura I, Kishida K, Nishizawa H, Matsuda M, Nagaretani H, et al. Diet-induced insulin resistance in mice lacking adiponectin/ACRP30. Nat Med. 2002; 8:731–7. [PubMed: 12068289]
9. Snijder MB, Heine RJ, Seidell JC, Bouter LM, Stehouwer CD, Nijpels G, et al. Associations of adiponectin levels with incident impaired glucose metabolism and type 2 diabetes in older men and women: the Hoorn study. Diabetes Care. 2006; 29:2498–503. [PubMed: 17065691]
10. Spranger J, Kroake A, Möhlig M, Bergmann MM, Ristow M, Boeing H, et al. Adiponectin and protection against type 2 diabetes mellitus. Lancet. 2003; 361:226–8. [PubMed: 12547549]
11. Côté M, Mauriège P, Bergeron J, Alméras N, Tremblay A, Lemieux I, et al. Adiponectinemia in visceral obesity: impact on glucose tolerance and plasma lipoprotein and lipid levels in men. J Clin Endocrinol Metab. 2005; 90:1434–9. [PubMed: 15598678]

12. Jain SH, Massaro JM, Hoffmann U, Rosito GA, Vasan RS, Raji A, et al. Cross-sectional associations between abdominal and thoracic adipose tissue compartments and adiponectin and resistin in the Framingham Heart Study. Diabetes Care. 2009; 32:903–8. [PubMed: 19223612]
13. Basu R, Pajvani UB, Rizza RA, Scherer PE. Selective downregulation of the high molecular weight form of adiponectin in hyperinsulinemia and in type 2 diabetes: differential regulation from nondiabetic subjects. Diabetes. 2007; 56:2174–7. [PubMed: 17513700]
14. Kobayashi H, Ouchi N, Kihara S, Walsh K, Kumada M, Abe Y, et al. Selective suppression of endothelial cell apoptosis by the high molecular weight form of adiponectin. Circ Res. 2004; 94:e27–31. [PubMed: 14752031]
15. Klein S, Fontana L, Young VL, Coggan AR, Kilo C, Patterson BW, et al. Absence of an effect of liposuction on insulin action and risk factors for coronary heart disease. N Engl J Med. 2004; 350:2549–57. [PubMed: 15201411]
16. Barzilai N, She L, Liu BQ, Vuguin P, Cohen P, Wang J, et al. Surgical removal of visceral fat reverses hepatic insulin resistance. Diabetes. 1999; 48:94–8. [PubMed: 9892227]
17. Pitombo C, Araújo EP, De Souza CT, Pareja JC, Geloneze B, Velloso LA. Amelioration of diet-induced diabetes mellitus by removal of visceral fat. J Endocrinol. 2006; 191:699–706. [PubMed: 17170226]
18. Kim YW, Kim JY, Lee SK. Surgical removal of visceral fat decreases plasma free fatty acid and increases insulin sensitivity on liver and peripheral tissue in monosodium glutamate (MSG)-obese rats. J Korean Med Sci. 1999; 14:539–45. [PubMed: 10576150]
19. Lottati M, Kolka CM, Stefanovski D, Kirkman EL, Bergman RN. Greater omentectomy improves insulin sensitivity in nonobese dogs. Obesity (Silver Spring). 2009; 17:674–80. [PubMed: 19214178]
20. Thörne A, Lönngvist F, Apelman J, Hellers G, Arner P. A pilot study of long-term effects of a novel obesity treatment: omentectomy in connection with adjustable gastric banding. Int J Obes Relat Metab Disord. 2002; 26:193–9. [PubMed: 11850750]
21. Csendes A, Maluenda F, Burgos AM. A prospective randomized study comparing patients with morbid obesity submitted to laparotomic gastric bypass with or without omentectomy. Obes Surg. 2009; 19:490–4. [PubMed: 18712575]
22. Herrera MF, Pantoja JP, Velázquez-Fernández D, Cabiedies J, Aguilar-Salinas C, García-García E. 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. Diabetes Care. 2010; 33:1413–8. [PubMed: 20587720]
23. Lima MM, Pareja JC, Alegre SM, Geloneze SR, Kahn SE, Astiarraga BD, et al. Acute effect of Roux-en-Y gastric bypass on whole-body insulin sensitivity: a study with the euglycemic-hyperinsulinemic clamp. J Clin Endocrinol Metab. 2010; 95:3871–5. [PubMed: 20484482]
24. Fabbrini E, Tamboli RA, Magkos F, Marks-Shulman PA, Eckhauser AW, Richards WO, et al. Surgical removal of omental fat does not improve insulin sensitivity and cardiovascular risk factors in obese adults. Gastroenterology. 2010; 139:448–55. [PubMed: 20457158]
25. Hosker JP, Matthews DR, Rudenski AS, Burnett MA, Darling P, Bowen EG, et al. Continuous infusion of glucose with model assessment: measurement of insulin resistance and beta-cell function in man. Diabetologia. 1985; 28:401–11. [PubMed: 3899824]
26. Purnell JQ, Kahn SE, Schwartz RS, Brunzell JD. Relationship of insulin sensitivity and ApoB levels to intra-abdominal fat in subjects with familial combined hyperlipidemia. Arterioscler Thromb Vasc Biol. 2001; 21:567–72. [PubMed: 11304474]
27. Weyer C, Bogardus C, Mott DM, Pratley RE. The natural history of insulin secretory dysfunction and insulin resistance in the pathogenesis of type 2 diabetes mellitus. J Clin Invest. 1999; 104:787–94. [PubMed: 10491414]
28. Festa A, Williams K, D'Agostino R Jr, Wagenknecht LE, Haffner SM. The natural course of beta-cell function in nondiabetic and diabetic individuals: the Insulin Resistance Atherosclerosis Study. Diabetes. 2006; 55:1114–20. [PubMed: 16567536]
29. Lee HC, Kim MK, Kwon HS, Kim E, Song KH. Early changes in incretin secretion after laparoscopic duodenal-jejunal bypass surgery in type 2 diabetic patients. Obes Surg. 2010; 20:1530–5. [PubMed: 20803098]

30. Buchwald H, Oien DM. Metabolic/bariatric surgery Worldwide 2008. Obes Surg. 2009; 19:1605–11. [PubMed: 19885707]

Table 1

Comparisons of parameters of body weight, glucose and lipid metabolism

	Omentectomy Group n=15		Control Group n=11	
	Pre-op	**Post**	**Pre-op**	**Post**
BMI (kg/m^2)	41.6 (4.5)	35.8 (4.7)[a]	42.3 (4.8)	36.6 (3.6)[a]
Glucose (mg/dL)	101.0 (27.3)	86.7 (10.6)[a]	108.8 (52.7)	91.5 (12.86)
Insulin (μIU/mL)	16.4 (7.19)	13.7 (12.3)	38.9 (55.9)	11.8 (6.9)
HOMA-IR	2.1 (0.97)	1.69 (1.4)	2.3 (1.5)	1.5 (1.0)
Cholesterol (mg/dL)	191.4 (42.3)	163.2 (33.6)[a]	189.6 (38.8)	155.7 (56.9)
VLDL-C (mg/dL)	35.9 (14.4)	21.1 (4.9)[a]	36.5 (11.4)	26.8 (9.5)
LDL-C (mg/dL)	109.7 (36.1)	97.5 (27.0)	109.6 (33.2)	95.6 (35.8)
Triglycerides (mg/dL)	179.4 (71.7)	105.8 (25.1)[a]	182.2 (57.4)	125.0 (60.6)[a]
HDL-C (mg/dL)	45.8 (10.4)	44.5 (12.6)	44.3 (15.2)	41.6 (10.7)

Results are Mean (SD).

[a] $p < 0.05$ for within-group changes at post-operative follow-up. All between-group comparisons were not significant at post-operative follow-up.

Table 2

Comparisons of parameters of inflammatory markers and adipokines.

	Omentectomy Group n=15		Control Group n=11	
	Pre-op	**Post**	**Pre-op**	**Post**
Adiponectin (μg/mL)	7.2 (3.1)	8.6 (3.1)[a]	5.8 (3.0)	7.2 (3.2)[a]
HMW Adiponectin (μg/mL)	3.5 (2.1)	4.7 (2.7)[a]	2.4 (1.5)	3.3 (2.1)[a]
HMW/total (%)	0.46 (0.1)[b]	0.52 (0.15)[a]	0.39 (0.06)	0.44 (0.12)
CRP (mg/L)	7.9 (4.7)	6.3 (6.9)	12.2 (13.5)	9.0 (8.7)
TNF-α (pg/mL)	1.6 (1.04)	1.9 (1.6)	1.3 (0.54)	1.6 (0.68)
IL-10 (pg/mL)	1.4 (0.42)[b]	1.5 (0.4)	2.1 (0.66)	2.3 (0.97)[c]
IL-6 (pg/mL)	5.6 (3.0)	3.9 (2.7)	3.7 (1.9)	3.4 (1.4)
IL-1b (pg/mL)	0.03 (0.07)	0.03 (0.10)	0.07 (0.14)	0.02 (0.05)
Fibrinogen (mg/mL)	3.2 (0.49)	3.0 (0.7)	2.8 (0.87)	2.9 (0.68)
IL-8 (pg/mL)	12.6 (10.3)	12.7 (4.2)	13.9 (4.8)	13.2 (6.9)
PAI-1 (pg/mL)	4933 (2901)	4258 (3167)	5552 (3785)	1998 (2104)[a]

Results are Mean (SD).

[a] $p < 0.05$ for within-group changes at post-operative follow-up.

[b] $p < 0.05$ for between-group comparisons at baseline.

[c] $p = 0.001$ for between group comparisons at post-operative follow-up.