Laparoscopic Adjustable Gastric Banding for Morbidly Obese Adolescents Affects Android Fat Loss, Resolution of Comorbidities, and Improved Metabolic Status

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BACKGROUND: The distribution of weight loss and its impact on metabolic health has not been documented for laparoscopic adjustable gastric banding (LAGB) in the adolescent population. We hypothesized that LAGB in obese adolescents would result in loss of android fat mass, resolution of comorbidities, and improvement in metabolic status.

STUDY DESIGN: Adolescents ages 14 to 17 who met criteria for bariatric surgery were enrolled in our FDA-approved LAGB trial. Demographic data, body mass index, body composition and bone density, laboratory evaluations, and comorbid conditions were assessed pre- and postoperatively.

RESULTS: Forty-five patients had complete 1-year followup and 41 patients had complete 2-year followup. Mean preoperative weight was 299 ± 57 lb and body mass index was 48 ± 6.4 kg/m^2. The percent excess weight losses at 6 months, 1 year, and 2 years were 31 ± 16, 46 ± 21, and 47 ± 22, respectively. At 1-year followup, patients after LAGB had a significant decrease in their total and android fat mass. In addition, 47 of 85 identified comorbidities (55%) were completely resolved and 25 (29%) were improved in comparison with baseline. Improvements in alkaline phosphatase, aspartate aminotransferase, alanine aminotransferase, hemoglobin A1c, fasting insulin, triglycerides, and high density lipoprotein, were also seen.

CONCLUSIONS: The percent excess weight loss after LAGB in morbidly obese adolescents is approximately 45% at 1- and 2-year followup, with the majority of weight loss consisting of android fat mass. Resolution or improvement of comorbidities is seen, and improved metabolic status, as demonstrated by liver function tests, lipid levels, and measures of glucose homeostasis, may be expected. These data support LAGB as an appropriate surgical option for morbidly obese adolescents. (J Am Coll Surg 2009;209:638–644. © 2009 by the American College of Surgeons)

A new era of bariatric surgery has been ushered in by the suggestion of Roux-en-Y gastric bypass (RYGB) as a treatment for diabetes mellitus, even for patients who do not meet criteria for morbid obesity.[1,2] This enthusiasm coincides with an adolescent obesity epidemic that presents a pressing challenge for the medical community[3] and mounting evidence that surgery is the only durable method available to achieve significant weight loss.[4] So, bariatric surgery has gained increasing popularity to treat morbid obesity in adolescents. We began treating morbidly obese adolescents off-label using laparoscopic adjustable gastric banding (LAGB) since the LAP-BAND (Inamed Corp) device gained FDA approval in 2001, and starting in June 2005 we began a prospective study with an investigation device exemption from the FDA. Our initial results, including a cohort of patients in whom LAGB was performed off-label, demonstrated approximately 50% excess weight loss (EWL) at 1- and 2-year followup, a significant decrease in body mass index (BMI), and resolution or improvement in the majority of comorbidities.[5,6] But the distribution of this weight loss and its impact on metabolic health have yet to be documented. We hypothesized that

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Abbreviations and Acronyms

ALT = alanine aminotransferase
AST = aspartate aminotransferase
BMI = body mass index
DEXA = dual-energy x-ray absorptiometry
EWL = excess weight loss
LAGB = laparoscopic adjustable gastric banding
RYGB = Roux-en-Y gastric bypass
TSH = thyroid stimulating hormone

LAGB in morbidly obese adolescents would result in loss of android fat mass, resolution of comorbidities, and improvement in metabolic status, without impairing nutritional status. We used only patients in our FDA-approved trial to investigate these issues.

METHODS

Study protocol

All patients enrolled in our study met the National Institutes of Health consensus development conference criteria for bariatric surgery.[7] Institutional Review Board approval (H11876-01) was obtained and our local board placed the requirement that all patients have a BMI > 40 kg/m^2. Entry criteria for the FDA-approved trial included: 1) age between 14 and 18 years; 2) BMI > 40 kg/m^2; 3) history of obesity for 5 years, including a 6-month failed attempt at medically supervised weight loss; 4) psychologist or psychiatrist confirmation of maturity to comply with the protocol; 5) willingness to follow protocol requirements; and, 6) use of an appropriate form of contraception. Exclusion criteria included: 1) intention to have another weight-loss procedure; 2) history of anomalies of the gastrointestinal tract; 3) dysphagia or documented esophageal dysmotility; 4) presence of autoimmune connective tissue disorders; 5) presence of acute abdominal infection; 6) pregnancy or intention of becoming pregnant in the next 12 months; 7) presence of psychiatric problems that would compromise cooperation with the study protocol; 8) history of previous bariatric surgery, intestinal obstruction, or adhesive peritonitis; 9) history of gastric or esophageal surgery; and 10) unwillingness to discontinue weight-loss medications.

Consecutive patients who met criteria were enrolled and subject to the baseline evaluations listed in Table 1. All data were entered into a database (Minnesota Database for Bariatrics, Exemplo Medical) and compared at serial time points. One- and 2-year data were compared with baseline using the two-tailed paired *t*-test for normally distributed data and the Wilcoxon signed ranks test for skewed data. Data reported are mean ± standard deviation for normally distributed data. Statistical significance was assigned to p values < 0.05.

Table 1. Baseline Evaluations for All Adolescents Enrolled in Study

Laboratory evaluations
Metabolic profile (sodium [meq/L], potassium [meq/L], chloride [meq/L], CO_2[meq/L], blood urea nitrogen [mg/dL], creatinine [mg/dL], glucose [mg/dL], calcium [mg/dL])
Hemogram with differential
Thyroid panel (T_3[ng/mL], T_4[mcg/dL], TSH [μIU/L], free T_4[ng/dL])
Lipid panel (total cholesterol [mg/dL], LDL [mg/dL], HDL [mg/dL], triglycerides [mg/dL])
Hepatic function panel (albumin [g/dL], alkaline phosphatase [U/L], total bilirubin [mg/dL], direct bilirubin [mg/dL], alanine aminotransferase [U/L], aspartate aminotransferase [U/L], total protein [g/dL])
Hemoglobin A1c (%) and fasting insulin (μIU/mL)
Nutritional panel (iron [mcg/dL], folate [ng/mL], B12 [pg/mL], vitamin D 25-hydroxy [ng/mL], parathyroid hormone [pg/mL], magnesium [mg/dL], phosphorus [mg/dL])
Oral glucose tolerance test, excluding subjects who have diabetes
Other evaluations
History and physical examination
Height and weight
Hip and waist circumference (cm)
Blood pressure
Routine urinalysis
Dual-energy x-ray absorptiometry (DEXA)
Chest x-ray
Electrocardiogram
Gallbladder ultrasound
Esophagram

Surgical and postoperative management

The LAP-BAND (LAP-BAND System, Inamed Corporation) was placed laparoscopically by the pars flaccida technique, which has been previously described.[8] Contrast esophagram was routinely performed in the first postoperative week to confirm band position and evaluate pouch size. All patients were directed to take one multivitamin with iron daily starting immediately after discharge to continue indefinitely. Patients returned monthly for the first postoperative year to monitor weight loss, appetite, dysphagia or food intolerance, and eating behavior. The first adjustment was performed 6 weeks after operation. Adjustments after the initial visit were determined on the basis of appetite, weight loss, food restriction, and satiety. Saline was administered if the patient reported increased appetite, less-than-anticipated weight loss (1 to 2 pounds per week), or minimal food intake restriction. Reasons for saline removal were dysphagia, frequent emesis, complete food intolerance, or restriction intolerance.

RESULTS

Between June 2005 and January 2007, 54 adolescents were evaluated for LAGB. Three patients withdrew and one pa-

Table 2. Percent Excess Weight Loss in Morbidly Obese Pediatric Patients after Laparoscopic Adjustable Gastric Banding

Time	n	Mean weight, lb	Mean BMI, kg/m^2	%EWL	Waist circumference, cm	Hip circumference, cm
Preoperative	45	299 ± 57	48.1 ± 6.4	NA	134 ± 17	142 ± 13
6 mo	45	249 ± 55*	40.2 ± 6.8*	30.8 ± 15.8	NA	NA
1-y	45	227 ± 58*	36.3 ± 7.5*	46.0 ± 21.1	112 ± 20*	121 ± 16*
2-y	41	225 ± 58*	35.8 ± 7.9*	46.8 ± 21.9	109 ± 22*	116 ± 23*

*Values represent mean ± SD, $p < 0.001$ versus preoperative, paired *t*-test.
BMI, body mass index; EWL, excess weight loss.

tient was disqualified because of low Tanner stage before device implantation. Fifty patients underwent operation. Of those, three patients were dropped from the study because of noncompliance, and two patients were removed from the study after adverse events. These two patients underwent band removal after a gastric perforation as a complication of band revision for eccentric pouch dilation (band slip), and band removal because of patient request after band slip. Forty-five patients remained with complete 1-year followup, and 41 had complete 2-year followup. There was no difference in demographics, initial weight, or BMI between the patients who were removed from the study or were lost to followup and those who remained in the study. Of the original 45 patients, 30 were female and 15 were male. There were 7 Hispanic, 5 African-American, and 1 Native-American patients. The remaining patients were Caucasian. Their mean age was 16.1 ± 1.2 years, preoperative weight was 299 ± 57 lb, and BMI was 48 ± 6.4 kg/m^2. No patients experienced intraoperative complications, including converting to an open approach. The EWL at 6 months, 1 year, and 2 years postoperatively were 31 ± 16%, 46 ± 21%, and 47 ± 22%, respectively. The BMI at these times were 40.2 ± 6.8, 36.3 ± 7.5, and 35.8 ± 7.9 kg/m^2, respectively. Both hip circumference and waist circumference were significantly lower 1 year and 2 years after LAGB (Table 2).

There were 36 patients who had preoperative and 1-year postoperative dual-energy x-ray absorptiometry (DEXA) scans at our institution. The others were either too large to fit on our scanner or had their preoperative DEXA at an outside institution. The subgroup of 36 was representative of the entire cohort (Table 3). After LAGB, patients lost significant fat mass and lean mass, but the fat mass loss was more substantial than the lean mass loss (Table 3). The fat mass loss was significant only in the percentage of android fat. We also used DEXA to evaluate bone density in our cohort. One year after LAGB, patients demonstrated an age-appropriate increase in bone density as measured by DEXA scan (Table 4).

Six of the 45 patients included in the initial analysis did not have any comorbidity at the time of operation. The remaining 39 had a total of 85 comorbidities. The distribution and outcomes for these at 1 and 2 years is shown in Table 5. There was a small amount of greater benefit seen 2 years after the procedure when compared with 1 year postprocedure (Table 5). All patients with impaired glucose tolerance and gastroesophageal reflux were completely free of disease 1 and 2 years after operation. Patients with hypertension, osteoarthritis, and dyspnea also significantly benefitted from LAGB. The condition with the least favorable result was dyslipidemia. Six of 17 patients were free from dyslipidemia after operation, but 4 patients had worsening dyslipidemia 1 year postoperatively. Two years after operation, an additional four patients had resolution of their dyslipidemia.

There were a number of asymptomatic patients with anemia (n = 19) 1 year after LAGB despite no significant difference in the hemoglobin or hematocrit levels in the cohort at either 1- or 2-year followup (data not shown). There was a small but significant decrease in mean RBC count (5.01 ± 0.38 versus 4.88 ± 0.44 and 4.80 ± 0.51 × 10^6/mcL, $p = 0.005$ and $p < 0.001$, respectively) and median red cell distribution width (12.8 versus 12.0 and 12.1%, $p < 0.001$) at 1 and 2 years, and a slight increase in mean corpuscular volume (82.1 versus 84.6 mcm^3 at both time points, $p < 0.001$). The hemogram also demon-

Table 3. Body Composition in Morbidly Obese Pediatric Patients 1 Year after Laparoscopic Adjustable Gastric Banding

Variable	Age, y	Weight, lbs	BMI, kg/m^2	% EWL	Fat mass, lb	Lean mass, lb	Android fat, %	Gynecoid fat, %
Preoperative (n = 36)	16.0 ± 1.2	314 ± 99	48 ± 7	N/A	144 ± 31	138 ± 26	57 ± 4	53 ± 4
1 y (n = 36)	17.1 ± 1.2	226 ± 53	36 ± 7	45 ± 20	105 ± 36	111 ± 23	52 ± 10	51 ± 9
p Value (paired *t*-test)		<0.0001	<0.0001		<0.0001	<0.0001	0.001	0.09

Values represent mean ± SD.
BMI, body mass index; EWL, excess weight loss.

Table 4. Bone Density in Morbidly Obese Pediatric Patients 1 Year after Laparoscopic Adjustable Gastric Banding

Variable	Bone mineral density	Z-score
Baseline (n = 36)	1.21 ± 0.19	0.41 ± 1.1
1-year (n = 36)	1.28 ± 0.16	0.74 ± 1.2
p Value (paired *t*-test)	0.0004	0.0003

Values represent mean ± SD.

strated a small but significant decrease in WBC 1 and 2 years after surgery (8.53 ± 2.27 versus 7.86 ± 2.07 and 7.43 ± 1.95 × 10^9/L, $p = 0.047$ and $p < 0.001$, respectively).

There was no statistical difference in mean serum chloride, blood urea nitrogen, glucose, or total protein between baseline and 1 or 2 years after surgery (data not shown). Of these, only total protein was different from baseline 2 years after surgery (7.7 ± 0.6 versus 7.4 ± 0.4 g/dL, $p < 0.001$). There was a small but significant increase in serum sodium (140.7 ± 1.7 versus 141.6 ± 2.1 mEq/L, $p = 0.028$) and bicarbonate (25.2 ± 1.6 versus 26.8 ± 2.6 mEq/L, $p < 0.001$) after 1 year, but it was not significant after 2 years, and a small but significant decrease in serum potassium after 1 year (4.4 ± 0.3 versus 4.3 ± 0.4 mEq/L, $p = 0.015$), which also was not significant after 2 years. But there was a larger and significant decrease in mean alkaline phosphatase level at both 1 and 2 years (Table 6). Nonparametric analysis revealed no difference in serum creatinine, albumin, calcium, or total bilirubin levels between baseline and 1-year after operation (data not shown). Serum albumin and total bilirubin remained no different from baseline 2 years after surgery (data not shown). Median creatinine (0.75 versus 0.7 mg/dL, $p = 0.002$) and median calcium (9.7 versus 9.6 mg/dL, $p = 0.01$) levels were slightly lower 2 years after surgery. There were significant decreases in median aspartate aminotransferase (AST) and alanine aminotransferase (ALT) both 1 and 2 years after operation (Table 6). Mean hemoglobin A1c and fasting insulin levels were also significantly improved 1 and 2 years after surgery (Table 6), although the levels did not differ between years 1 and 2.

There was no difference in mean serum cholesterol or LDL levels 1 year after surgery (data not shown), although a decrease in LDL was noted 2 years after surgery that nearly reached statistical significance (106 ± 27 versus 98 ± 29 mg/dL, $p = 0.052$). But there was a significant decrease in serum triglyceride levels and a concomitant increase in serum HDL levels at both time points (Table 6). Similarly, there was no difference in mean T4 level or median free T4 1 and 2 years after surgery (data not shown). But T3 (1.5 ± 0.33 versus 1.4 ± 0.21 ng/dL and 1.3 ± 0.22 ng/dL, $p = 0.002$ and $p < 0.001$, respectively) and thyroid stimulating hormone (TSH) (2.45 ± 1.49 versus 2.00 ± 1.53 and 1.9 ± 1.4 mIU/L, $p = 0.037$ and $p = 0.026$, respectively) levels were significantly decreased 1 and 2 years after LAGB.

There was no difference in mean serum B12 or parathyroid hormone levels at 1 year (data not shown), although B_{12} levels were significantly lower by year 2 (564 ± 203 versus 503 ± 246 pg/mL, $p = 0.037$). The only significant decrease in nutritional values at 1 year was seen in folate levels (13.9 ± 7.7 versus 8.8 ± 5.1 ng/mL, $p = 0.001$), but this difference was no longer significant at 2 years. There were small but significant increases in phosphorus (4.0 ± 0.6 versus 4.2 ± 0.6 mg/dL, $p = 0.047$) and magnesium (1.9 ± 0.2 versus 2.0 ± 0.2 mEq/L, $p = 0.036$) levels at 1 year, but these values were also no longer significant 2 years postprocedure. Larger increases in iron (60 ± 31 versus 73 ± 35 μg/dL, $p = 0.048$) and Vitamin D (22 ± 10 versus 26 ± 12 pg/mL, $p = 0.025$) levels were seen 1 year after operation, but similarly, were no longer significant 2 years after surgery. A summary of the most significant laboratory values is provided in Table 6.

Table 5. Comorbid Conditions Associated with Morbid Obesity in Pediatric Patients

Comorbid conditions	Patients preoperatively, n (%) (total n = 39)	1 y postoperatively, n (total n = 21)	2 y postoperatively, n (total n = 13)
Dyslipidemia	17 (44)	11 (4 improved, 3 unchanged, 4 aggravated)	5 (1 improved, 3 unchanged, 1 aggravated)
Back pain	12 (31)	6 (3 improved, 3 unchanged)	4 (2 improved, 2 unchanged)
Hypertension	11 (28)	3 (2 improved, 1 unchanged)	0
Depression	10 (26)	6 (5 improved, 1 unchanged)	6 (5 improved, 1 unchanged)
Impaired glucose tolerance	9 (23)	0	0
Dyspnea at rest	8 (21)	4 (4 improved)	1 (1 unchanged)
Osteoarthritis	8 (21)	3 (3 improved)	2 (2 improved)
Obstructive sleep apnea	5 (13)	4 (3 improved, 1 unchanged)	4 (4 improved)
Asthma	3 (8)	3 (3 improved)	2 (2 improved)
Gastroesophageal reflux	2 (5)	0	0

Table 6. Summary of the Most Significant Laboratory Values 1 and 2 Years after Laparoscopic Adjustable Gastric Banding

Laboratory test	Baseline (n = 44)	1-y (n = 44)	2-y (n = 41)	p Value
Alkaline phosphatase, U/L	102 ± 28	82 ± 21	75 ± 16	< 0.001
Aspartate aminotransferase, U/L	27	19	20	< 0.001
Alanine aminotransferase, U/L	35.5	23.5	22	< 0.001
Hemoglobin A1c, %	5.6 ± 0.4	5.3 ± 0.3	5.3 ± 0.3	< 0.001
Fasting insulin, μIU/mL	36.3 ± 20.0	13.8 ± 7.6	13.9 ± 11.1	< 0.001
Triglycerides, mg/dL	117 ± 36	89 ± 38	76 ± 31	< 0.001
HDL, mg/dL	38.6 ± 8.7	45.3 ± 11.6	46.4 ± 13.1	< 0.001

*Values represent mean ± SD when the data were distributed normally, and median if the data were skewed. Appropriate parametric or nonparametric analysis was performed to determine p value. The p values are for both 1- and 2-year values compared with baseline.

DISCUSSION

It is approaching 20 years since the National Institutes of Health consensus development conference on gastrointestinal surgery for severe obesity in 1991,[7] and it appears that weight loss surgery will continue to be part of the armamentarium in the fight against obesity as potential indications for bariatric procedures are expanding.[1] Our center began using LAGB to treat adolescents with morbid obesity in 2001 off-label, when the device was initially approved for use in this country, and subsequently, in 2005, obtained an investigation device exemption from the FDA to better evaluate the safety and efficacy. Initial results demonstrated durable short-term weight loss, resolution of comorbidities, few issues with compliance, with little morbidity and no mortality.[5,6] But with the increased emphasis on the metabolic consequences of weight loss surgery, questions remain whether LAGB can be expected to produce significant health benefits in the adolescent population. So we analyzed our prospective cohort to determine their body composition changes, the status of their comorbid illnesses, their markers of metabolic health, and their overall nutritional status.

Other than the mean age being lower than in an adult cohort, the patient demographics in our study are not different than those in our previous reports or in adults.[5,6,9] The percent EWL at 6 months, 1 year, and 2 years postoperatively were the same or better than those reported in most adult series and adolescent series from overseas.[9-10] Adolescent LAGB cohorts from abroad have demonstrated resolution of comorbid conditions paired with weight loss.[11,12] Our results are similar, with 55% of comorbid conditions completely resolved at 1-year followup and 29% demonstrating improvement when compared with baseline. The results are slightly better 2 years after operation. The only comorbidity with mixed results was dyslipidemia, for which 4 of 17 patients had worsening lipid profiles after surgery. Large adult series of LAGB have also shown persistence of dyslipidemia despite significant weight reduction,[12] and it is possible that RYGB may result in superior control of this particular comorbidity, perhaps because of its malabsorptive component.[13] But this hypothesis would need to be tested in a prospective randomized fashion before RYGB could be recommended in adolescents with dyslipidemia.

Our patients underwent a battery of evaluations aimed at assessing metabolic and nutritional health. The results yielded only one significant nutritional deficiency in the cohort at 1 year (which was resolved at 2 years) and abundant data confirming the benefits of the procedure. Patients lost fat mass in excess of lean mass, although there was significant lean mass loss 1 year after operation. Fat mass loss was significant only in the android component. Similar results have been seen in five adolescent patients after RYGB.[14] Android fat has long been linked to development of obesity-related illnesses,[15] and the significant android fat loss coupled with improvement or resolution of the vast majority of comorbidities after LAGB in our cohort suggests LAGB is a viable strategy to treat morbid obesity and its related illnesses in the adolescent.

The presence of lean mass loss is not unique to our study. Some adult series have suggested that muscle mass is relatively preserved after LAGB or RYGB, although significant lean mass loss was seen after both types of operation in these studies.[16-18] It may be that bariatric surgery and its limited food intake results in diminished protein intake and subsequent lean mass loss. But the limited nutritional intake did not impair normal bone maturation as evaluated by bone mineral density on DEXA scan. Equally plausible is that decreased fat mass leads to a lesser load on the skeletal muscles with subsequent lean mass loss from a reduction of work being performed by these muscles. In either case, it may be important to follow adolescent patients with yearly DEXA scans to ensure that the lean mass loss plateaus with time. Certainly we and others have not seen any symptomatic muscle loss after weight loss surgery to date.

Laboratory evaluations demonstrated substantial improvement in metabolic parameters that one would expect with significant weight loss, without any significant nutritional consequences. The only nutritional deficiency seen in our patients as a group was a decrease in mean folate levels at 1 year after surgery, which corrected 2 years after the procedure. The cause of this deficit is unclear because all patients were prescribed a multivitamin with 100% of the recommended daily allowance of folate. We did find that approximately 40% of our patients had a decrease in hemoglobin and hematocrit, which could suggest noncompliance with the vitamin supplementation. But we also found small increases in mean magnesium and phosphorus levels and larger increases in mean iron levels that would argue against noncompliance. There was a small but significant increase in median red blood cell mean corpuscular volume that could be attributed to the folate deficiency, and it will be important to continue to follow nutritional values and hemograms in adolescent patients undergoing weight loss surgery moving forward.

The remainder of the laboratory evaluations revealed improved metabolic status and general health. There was a small, but significant decrease in WBC count 1 and 2 years after LAGB. The difference is unlikely to be clinically significant, but it well known that obesity is a chronic inflammatory condition and WBCs can decrease after weight loss surgery.[17] Indicators of improved metabolic status include decreased serum liver enzyme levels, thyroid stimulating hormone, hemoglobin A1c and fasting insulin, serum triglyceride levels, and an increase in HDL levels. Weight-loss surgery has been shown to improve nonalcoholic fatty liver disease and nonalcoholic steatohepatitis in adults.[18] In our cohort, the aminotransferases and alkaline phosphatase levels had highly significant but small decreases. In the subset of patients (n = 9) that had elevated AST or ALT at baseline, the effect of weight-loss surgery on aminotransferase level was even more dramatic. Mean AST dropped 1 year after surgery from 68 ± 36 to 22 ± 7 U/L (p = 0.002, paired *t*-test) and mean ALT dropped from 121 ± 76 to 34 ± 14 U/L (p = 0.005, paired *t*-test). A trial specifically for morbidly obese teenagers with nonalcoholic fatty liver disease or nonalcoholic steatohepatitis would certainly be interesting to corroborate our results.

Also interesting was the effect of LAGB on thyroid function. We found a small difference in serum T_3 levels, but the difference of 0.1 ng/mL is smaller than the analytic variability of the test. We also found a significant decrease in mean TSH levels from baseline. Similar findings have been reported and the decrease in TSH may be related to circulating leptin levels.[19,20] The clinical significance of a decrease within the normal range is unclear and merits further study. In a subset of our cohort with a baseline TSH of >3 mcU/mL (n = 12), suggesting an element of subclinical hypothyroidism, LAGB resulted in a decrease in median TSH from 3.6 to 2.9 mcU/mL 1 year after surgery (p = 0.028, Wilcoxon signed ranks test).

Perhaps the most imperative finding in our study is the effect LAGB had on glucose homeostasis. Obesity in adults and in adolescents has a well-described association with increased insulin resistance, which is proportional to the patient's BMI.[21,22] We found that the mean hemoglobin A1c for the entire cohort dropped by a small but highly significant amount 1 year after surgery and stayed at that level for the second year. More importantly, mean fasting insulin levels fell to nearly one-third of their baseline values 1 year after surgery and remained at that level after year 2. The importance of insulin resistance is well known as it relates to the metabolic syndrome,[23] and insulin resistance is even implicated in the pathogenesis of nonalcoholic fatty liver disease.[24,25] There has been a great deal of interest in promoting the gastric bypass as a treatment for insulin resistance,[1] and some advocate gastric bypass as a reasonable option for the treatment of obese adolescents with type 2 diabetes mellitus.[26] So it is important to demonstrate that LAGB for morbidly obese adolescents improves glucose metabolism as well, reducing the risk of developing the potentially devastating consequences of insulin resistance.

Also included in the criteria for the diagnosis of metabolic syndrome is the presence of dyslipidemia and increased waist circumference.[27] The American Heart Association criteria for inclusion in the diagnosis of metabolic syndrome consists of a triglyceride level >150 mg/dL, or an HDL <40 mg/dL for men and <50 mg/dL for women.[31] Although our cohort did not reach the criteria in regard to triglyceride levels, the mean HDL level was below the cutoff. Significantly, the HDL levels were increased by approximately 20% 1 year after LAGB in our adolescent population, with an additional small increase after year 2. This was coupled with a mean triglyceride level decrease of about 15%. With regard to waist circumference, the least strict value for patients born in the US is a waist circumference greater than 102 cm in men and 88 cm in women.[28] Our patients met these criteria even after the significant decrease in waist circumference 2 years after LAGB.

In summary, we found that the LAGB provides an excellent option for weight loss in the morbidly obese adolescent population. The EWL after LAGB in morbidly obese adolescents is approximately 45% at 1- and 2-year followup, with the majority of weight loss consisting of android fat. With the decrease in android fat, resolution or improvement of comorbidities is seen for the vast majority of patients. Patients may expect improved metabolic function, as demonstrated by improvement in liver function enzymes, serum lipid levels, and measures of glucose homeostasis, without significant nutritional deficiencies. These data further support LAGB as an appropriate surgical option for pediatric patients with morbid obesity.

Author Contributions

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