

Original article

## Effects of bariatric surgery on diabetic nephropathy after 5 years of follow-up

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### Abstract

**Background:** Studies have reported that the benefits of bariatric surgery extend beyond durable weight loss and include significant improvement in glycemic control. We hypothesized that improving diabetes control may have positive effects on end-organ complications of this disease, such as diabetic nephropathy (DN).

**Methods:** We identified all patients with type 2 diabetes mellitus (T2DM) who underwent bariatric surgery at our institution and had completed a 5-year follow-up. Patients' current diabetes status (remission, improvement, or no change) was determined by biochemical analyses and medication review. The presence of DN, preoperatively and postoperatively, was determined by urinary albumin/creatinine ratio (uACR).

**Results:** Fifty-two T2DM patients underwent bariatric surgery and had completed 5-year follow-up, including serial uACR measurements (25% male; age  $51.2 \pm 10.1$  years). Preoperative body mass index (BMI) was  $49 \pm 8.7$  kg/m<sup>2</sup>, mean duration of T2DM was 8.6 years (range .3–39), and baseline HbA<sub>1c</sub> was  $7.7\% \pm 1.4\%$ . DN, as indicated by microalbuminuria (30–300 mg/g) or macroalbuminuria (> 300 mg/g), was present in 37.6% preoperatively. Of these, DN resolved in 58.3% at a mean follow-up of 66 months (range 60–92). Among those with no evidence of DN preoperatively, albuminuria proceeded to develop 5 years later in only 25%. The 5-year remission and improvement rates for T2DM were 44% and 33%, respectively. Mean reductions in fasting glucose and glycosylated hemoglobin (HbA<sub>1c</sub>) were 36.6 mg/dL and 1.2%, respectively.

**Conclusion:** Bariatric surgery can induce a significant and sustainable improvement in T2DM and improve or halt the development of microvascular complications such as nephropathy. Considering that diabetes is often a progressive disease, these results are clinically important and warrant further investigation. (Surg Obes Relat Dis 2013;9:7-14.) © 2013 American Society for Metabolic and Bariatric Surgery. All rights reserved.

### Keywords:

Diabetic nephropathy; Bariatric surgery; Urinary ACR; Renal function; Type 2 diabetes mellitus

Diabetic nephropathy (DN) affects approximately 40% of the entire diabetic population, including patients with type 1 or type 2 diabetes mellitus (T2DM). It is the leading cause of end stage renal disease requiring dialysis and/or renal transplantation [1]. A systematic review of predictors of

diabetic complications, by Newman et al., identified that tight glycemic control had the most pronounced benefit on the development of microvascular diabetes complications, specifically nephropathy and retinopathy [2]. Such tight control of hyperglycemia is difficult to achieve using current maximal medical therapy. In fact, <50% of diabetic patients in the United States reach the American Diabetes Association's (ADA's) glycosylated hemoglobin (HbA<sub>1c</sub>) goal of <7% [3]. In addition to diabetes, obesity

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is independently associated with chronic kidney disease. Obese individuals develop microalbuminuria and overt proteinuria independently of underlying co-morbidities that also predict the development of chronic kidney disease (CKD) and progression to end stage renal disease [4]. The degree of protein excretion has been reported to be directly proportional to the severity of obesity and the metabolic syndrome [5], and a tight relationship has been found between obesity and changes in urine albumin-creatinine ratio (uACR) in patients with diabetes specifically [6]. Furthermore, albuminuria is an independent risk factor for cardiovascular disease and associated with markers of cardiac damage such as abnormal left ventricular geometry and mass [7,8].

The pathophysiology of diabetic and obesity-related nephropathy is complex and multifactorial. In the former, hyperglycemia initiates a cascade of events that are injurious to the kidney, including the production of vasodilatory prostaglandins, inflammatory cytokines, advanced glycosylation products, and reactive oxygen species. In obesity-related nephropathy, the increase in intra-abdominal pressure causes an increase in renal venous pressure, systemic blood pressure, and vascular resistance, all of which impair renal perfusion and activate the juxtaglomerular apparatus (JGA) and the renin-angiotensin-aldosterone system (RAAS), causing increased release of renin, angiotensin, and aldosterone. This, in turn, causes hypertension, glomerulopathy, and proteinuria [9,10]. Additionally, the metabolic syndrome features of insulin resistance and inflammation also play a role in the development of nephropathy in the setting of obesity. Low levels of adiponectin, an anti-inflammatory adipokine, have also been linked to the increased urinary protein in obese patients [11]. Common to both diabetic and obesity-related nephropathies are the effects of renal vasodilation, hyperfiltration, and ultimately renal injury [12,13].

Optimal therapy of DN continues to evolve. Guidelines from the ADA and the National Kidney Foundation highlight the benefits of tight glycemic and blood pressure control, as well as inhibition of the renin-angiotensin system, in retarding the progression of diabetic nephropathy. In the Diabetes Control and Complications Trial, intensive treatment of diabetes reduced the incidence of microalbuminuria by 39% [14]. Managing other risk factors for cardiovascular disease, such as dyslipidemia, is also critical because of the high rate of cardiovascular death in this population. The effect of weight loss on the progression of kidney disease in the obese and of diabetic reduction through diet and lifestyle modifications can result in reduction of urinary protein excretion [15]. However, weight loss achieved in this way has limited efficacy and durability, and hence, the effects on albuminuria may not be sustained. Furthermore, some high-protein diets prescribed for weight loss may even be injurious to the kidney [16]. Bariatric surgery has revolutionized the treatment of obesity

in recent years. In addition to substantial weight loss, it leads to dramatic improvements in glycemic control, insulin sensitivity, and cardiovascular disease risk. To date, 3 randomized, controlled trials have directly compared medical versus surgical treatment for T2DM. The first of these, by Dixon et al., compared the 2-year outcomes of conventional medical treatment with gastric banding for the management of T2DM in 60 obese patients [17]. More recently, Schauer et al. and Mingrone et al. evaluated the 12- and 24-month effects, respectively, of bariatric surgery (gastric bypass or sleeve gastrectomy) compared with intensive medical therapy on diabetes management [18,19]. All 3 groups reported that weight loss surgery was far more effective than medical therapy at inducing remission or improvement of diabetes.

This postsurgical improvement in glycemic control may be responsible for the reduction in urinary protein excretion, which has also been reported after bariatric surgery. Navaneethan et al. have reported that urinary albumin excretion decreases in obese patients with diabetes after Roux-en-Y gastric bypass (RYGB). This reduction in albuminuria correlated with an improvement in insulin sensitivity and was related to a significant improvement in the anti-inflammatory adipokine adiponectin. Interestingly, these effects were independent of the degree of weight loss [20]. On the basis of these observations, we hypothesized that improving diabetes control with surgically induced weight loss may have positive effects on end-organ complications of this disease, such as DN, and that these effects are sustainable over medium- to long-term follow-up. The primary aim of this study was to determine the 5-year postoperative outcomes of morbidly obese diabetic patients who underwent bariatric surgery, with regard to the presence or absence of DN, glycemic control, and weight loss.

## Methods

### *Study cohort*

The Institutional Review Board at the Cleveland Clinic approved this retrospective review of a prospectively maintained bariatric surgery database. We identified all obese patients with a biochemically confirmed diagnosis of T2DM who had undergone bariatric surgery in our unit >5 years ago. These patients underwent surgery on the basis that they met the criteria for bariatric surgery outlined by the National Institutes of Health Consensus Development Panel report of 1991 [21]. The choice of procedure was determined on an individual case basis, influenced by surgeon and patient preferences, patient's body habitus, and past medical/surgical history. Thirty-six patients (69%) underwent RYGB, and 16 patients (31%) underwent restrictive procedures, including sleeve gastrectomy (n = 3) and adjustable gastric banding (LAGB; n = 13). These

procedures were performed as previously described [22–24]. All patients received similar preoperative preparation and were routinely followed up with at 1 week and 1 month postoperatively, then every 3 months for the first year and annually thereafter. Biochemical assessment was performed preoperatively and at annual clinic visits. Primary endpoints included the status of patient's diabetes at 5 years, the presence of DN (determined by the degree of albuminuria as measured by the uACR), weight loss, and other metabolic outcomes, including blood pressure and lipid profile.

#### Clinical and analytical measurements

A patient's diabetes status at each time point was determined by biochemical analyses (fasting blood glucose, HbA<sub>1c</sub>) and review of medications. Postoperatively, diabetes was ascertained to be in 1 of 3 states: remission, improvement, or no change/worse. Remission was defined according to recent ADA criteria [25]: normal glycemic measurements (HbA<sub>1c</sub> <6.5%; fasting blood glucose [FBG] <100 mg/dL) for at least 1 year's duration without the use of any oral hypoglycemic medications or insulin. Partial remission or improvement was defined as hyperglycemia below the diagnostic thresholds for diabetes, for at least 1 year's duration, with no active pharmacologic therapy, or as a decrease in the amount of medication required to achieve euglycemia compared with preoperative doses. The presence of DN was determined by uACR, where microalbuminuria was defined by a ratio between 30–300 mg/L and macroalbuminuria as >300 mg/L. Presence of the latter is considered overt nephropathy. Urine measurements of albumin/creatinine ratio were routinely performed on a first-morning-void midstream urine sample. The urine specimens were refrigerated at 2–8 °C immediately after collection and were analyzed the same day. Albuminuria was expressed as uACR in milligram per liter.

#### Statistical analysis

All statistical analyses were performed using PASW Statistics program version 18 (SAS Institute, Cary, NC). Descriptive statistics were computed for all variables; data regarding patient characteristics and outcomes are outlined in numbers and percentages. Normal distributions of the data were checked using the Kolmogorov-Smirnov D test. Parametric data are presented as mean  $\pm$  SD and analyzed using Student's 2-sample *t* test for any 2 sample comparisons and ANOVA, followed by Tukey HSD post hoc test, as appropriate. Differences between proportions and categorical variables were determined using the  $\chi^2$  test. Associations between variables were determined using Pearson's correlation analyses. In all tests, *P* < .05 was considered statistically significant.

## Results

### Patient characteristics

Fifty-two obese patients with T2DM underwent bariatric surgery in our institution between May 2004 and December 2006 and met the inclusion criteria for this study. This represents a 50.5% follow-up rate of all eligible patients. Baseline demographic characteristics and clinical characteristics of the study cohort are outlined in Table 1. In brief, 75% were female, with a mean age of 51.2  $\pm$  10.1 years and mean preoperative body mass index (BMI) of 49  $\pm$  8.7 kg/m<sup>2</sup>. All patients had at least 1 other obesity-related co-morbidity, predominantly hypertension (84.3%) and/or hyperlipidemia (70.6%). On average, these patients' duration of diabetes was 8.6 years (range .3–39). Preoperative glycemic control was poor, with a mean HbA<sub>1c</sub> of 7.7 % (range 4.8–11.4 %) and nearly 1 in 3 (29%) were taking insulin before undergoing surgery. Of these T2DM bariatric patients, 37.6% already had albuminuria at the time of surgery. Mean postoperative follow-up was 66 months (range 60–92).

### Weight loss at 5 years

Weight and BMI remained significantly decreased 5 years after surgery. The decreases in weight and BMI, after a mean follow-up of 66 months, were 66.4  $\pm$  44 pounds and 11.2  $\pm$  7 kg/m<sup>2</sup>, respectively. The percent of excess weight loss (%EWL) among the entire cohort was 50.6%  $\pm$  28%. Patients who underwent RYGB experienced significantly greater EWL than those who underwent LAGB or sleeve gastrectomy (56.9% versus 34.4% versus 46.3%, respectively; *P* = .038).

### Diabetes control

Five years postoperatively, the remission rate of T2DM was 44% and the improvement rate was 33%. The remaining 23% had no change or worsening glycemic control

Table 1

Baseline characteristics of obese T2DM patients who are >5 yr post-bariatric surgery

N	52
Mean age, yr	51.2 $\pm$ 10.1
Gender, female/male	75%/25%
Preoperative BMI, kg/m <sup>2</sup>	49.0 $\pm$ 8.7
Other co-morbidities:	
Hypertension	84.3%
Hyperlipidemia	70.6%
Duration of T2DM, mo (range)	102.6 (3–468)
% taking insulin preoperatively	29%
Mean preoperative HbA <sub>1c</sub>	7.7% (4.8–11.4)
Diabetic nephropathy (albuminuria)	37.6%
Follow-up postoperative, mo (range)	66.0 (60–92)

Data presented as means  $\pm$  SD or as percentages of the total study cohort. T2DM = type 2 diabetes mellitus; BMI = body mass index; HbA<sub>1c</sub> = glycosylated hemoglobin.

despite undergoing surgery. This latter group had the longest duration of T2DM before undergoing surgery and had the lowest EWL after 5 years. The mean reduction in HbA<sub>1c</sub> was 1.2% ± 1.6% (average preoperative HbA<sub>1c</sub>: 7.7% ± 1.4%; postoperative HbA<sub>1c</sub>: 6.5% ± 1.1%), and FBG fell from 147.2 ± 55 mg/dL to 111.0 ± 37 mg/dL.

*Change in prevalence of diabetic nephropathy 5 years postoperatively*

More than 1 in 3 (37.6%) of these obese patients with diabetes had nephropathy at the time of surgery—31.3% had microalbuminuria, and 6.3% had overt nephropathy (macroalbuminuria). Preoperatively, 21.6% were taking a renoprotective antihypertensive (an angiotensin-converting enzyme inhibitor [ACEi] or angiotensin receptor blocker ARB). The prevalence of albuminuria/DN after >5 years was 32.4%. This reflects an absolute decrease in the prevalence of DN of 5.2% over 5 years (*P* = .456). Of those who had albuminuria preoperatively, however, 58.3% experienced remission and had normal uACR >5 years after bariatric surgery. Of those with normal uACR preoperatively (i.e., no evidence of albuminuria or DN), microalbuminuria developed after 5 years in 25%. The other 75% continued to have normal uACRs, and no patient developed overt nephropathy during the postoperative follow-up period. When postoperative uACR measurements were evaluated according to T2DM remission status at 5 years, those patients who experienced remission or improvement also had the lowest postoperative

uACRs (21.8 ± 45.6 mg/g and 27.5 ± 33.6 mg/g, respectively), indicating that improvement in glycemic control was clearly associated with remission or non-progression to DN (Fig. 1). Conversely, patients who did not experience remission or whose glycemic control worsened over time continued to have elevated urinary protein excretion, with a mean uACR of 147.4 ± 188.8 of mg/g.

*Relationship between the effects of bariatric surgery on hypertension, DN, and the use of renoprotective antihypertensives*

The proportions of patients who experienced remission or improvement in hypertension 5 years after bariatric surgery were 10% and 46%, respectively. The mean changes in systolic and diastolic pressures were from 143 mm Hg to 134 mm Hg and from 82 mm Hg to 75 mm Hg, respectively (*P* = .015 and *P* = .001, respectively). Preoperatively, 23.1% of the study cohort was prescribed a renoprotective antihypertensive (an ACEi or ARB). After 5 years, the number of patients taking either of these medications almost doubled to 55.8%. Preoperatively, uACR was equivalent regardless of whether or not patients were taking an ACEi or ARB. At 5 years postoperatively, those who were not taking 1 of these antihypertensives had significantly lower uACR (which was within normal range, on average) compared with patients who continued to take an ACEi or ARB after 5 years (Fig. 2).

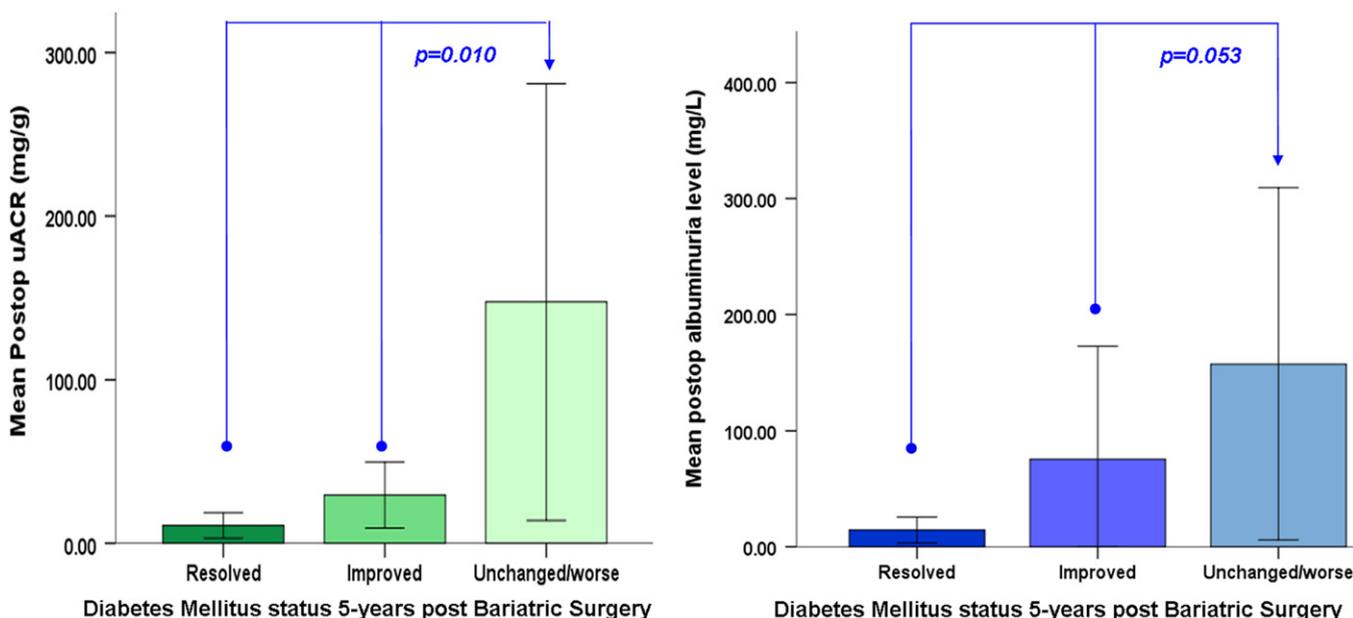


Fig. 1. (A) Mean uACR values according to T2DM remission status >5 years after bariatric surgery. Those patients who experienced remission or improvement of their diabetes had significantly lower uACR after a mean postoperative follow-up period of 66 months, compared with patients who had no improvement or progression of diabetes (*P* = .010). (B) Mean albuminuria level (mg/L) according to T2DM remission status >5 years after bariatric surgery. Mirroring the uACR data, urinary excretion of albumin was also lowest in patients who experienced remission of their diabetes compared with patients who had no improvement or progression of diabetes (*P* = .053). uACR = urinary albumin/creatinine ratio; T2DM = type 2 diabetes mellitus.

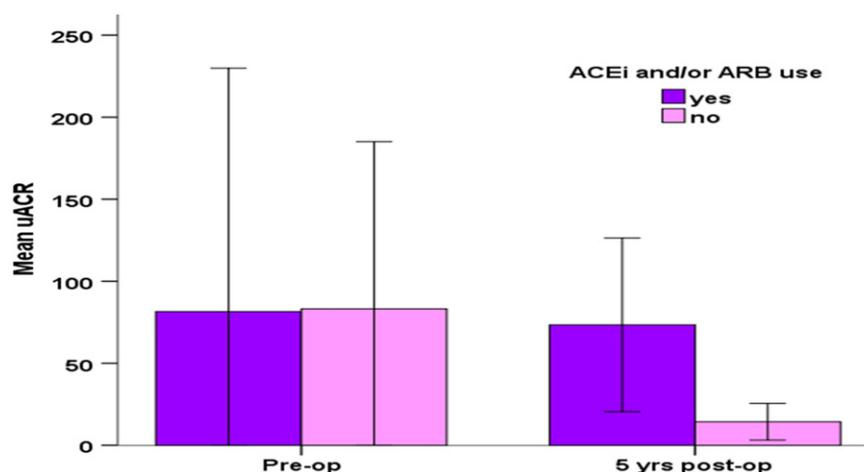


Fig. 2. Mean uACR values according to whether or not patients were taking an ACEi or ARB preoperatively and postoperatively. Preoperatively, uACR level was equivalent in patients taking and not taking renoprotective antihypertensives. Postoperatively, however, the uACR was significantly lower and within normal range in patients who were no longer prescribed an ACEi or ARB. uACR = urinary albumin/creatinine ratio; ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker.

#### Correlation of postoperative uACR with weight loss, metabolic and renal parameters

Postoperative uACR correlated significantly with the amount of weight loss (expressed in absolute terms and as the %EWL), and with systolic blood pressure at 5 years (Table 2). There was no significant correlation between postoperative uACR measurement and individual glycemic measures preoperatively or postoperatively. The uACR at 5 years postoperatively did correlate with the preoperative uACR (Pearson's correlation coefficient .426;  $P = .013$ ). There was no significant decrease in blood urea nitrogen (BUN) or creatinine levels after surgery: BUN preoperatively and postoperatively was 19.4 mg/dL and 17.8 mg/dL, respectively ( $P = .397$ ), and creatinine level preoperatively and postoperatively was 1.0 mg/dL and 1.0 mg/dL, respectively, ( $P = .899$ ). Estimated glomerular filtration rate (eGFR) was not routinely measured in our institution >5

years ago, so it was not possible to compare preoperative and postoperative eGFR levels in this group of patients.

#### Discussion

This study finds that bariatric surgery has a sustainable beneficial effect not only on glycemic control but also on end-organ complications of the disease such as DN. We found that more than half of the patients who already had albuminuria at the time of surgery (58.3%), experienced durable remission of albuminuria >5 years after their weight loss surgery. Furthermore, there was a notable lack of expected progression to DN in those obese patients with diabetes who had normal uACR preoperatively. Considering that according to the ADA, the rate of progression to nephropathy among all patients with type 2 diabetes is 10%–20% per year, it may be expected that without intervention and weight loss, albuminuria would have developed in well over 50% of our obese diabetic cohort after 5 years. The fact that microalbuminuria developed in only 25% of these patients after a mean follow-up of 66 months shows that bariatric surgery may halt or dramatically reduce the development of nephropathy in obese patients with diabetes.

A small number of studies have reported an improvement in renal function after bariatric surgery. Iaconelli et al. evaluated the effect of severe weight loss surgery on renal function, among other complications of diabetes, 10 years after the malabsorptive biliopancreatic diversion (BPD) procedure [26]. The authors used a less stringent threshold for defining nephropathy than in the present study, with a definition of microalbuminuria in the range 50–300 mg/L, so they may have slightly underestimated the prevalence of albuminuria preoperatively and postoperatively. We used the uACR range 30–300 mg/g to define albuminuria, which

Table 2

Correlation of postoperative uACR with preoperative and postoperative metabolic parameters

	Pearson's correlation coefficient	$P$
Correlation with uACR		
Weight loss, lb	-.401	.015
%EWL	-.326	.049
Preoperative HbA <sub>1c</sub> , %	.298	.078
Postoperative HbA <sub>1c</sub> , %	.199	.238
Fasting glucose, mg/dL	.020	.905
Systolic blood pressure, mm Hg	.348	.037
Diastolic blood pressure, mm Hg	.111	.520
BUN, mg/dL	.305	.070
Creatinine, mg/dL	.385	.020
Preoperative uACR, mg/g	.426	.013

EWL = excess weight loss; HbA<sub>1c</sub> = glycosylated hemoglobin; BUN = blood urea nitrogen; uACR = urinary albumin/creatinine ratio.

is also the reference range recommended by the ADA [27]. Only 22 patients who had BPD were available for 10-year follow-up, but all 7 patients who had microalbuminuria preoperatively experienced remission after BPD. In addition to the small study number, a limitation of this study is the lack of generalizability to current bariatric surgery practice, because BPD is uncommonly performed worldwide. Navarro-Diaz et al. examined renal function preoperatively and 12 and 24 months postoperatively in 61 morbidly obese patients who had undergone RYGB [28,29]. The authors found that many obesity-related renal alterations improved after surgery and that the decrease in albuminuria persisted at 24 months.

Our group recently reported that RYGB in severely obese T2DM patients was associated with a significant reduction in albuminuria which correlated with an improvement in insulin sensitivity (as measured by the Matsuda index) and with a beneficial increase in the anti-inflammatory adipokine adiponectin [20]. The present study had extended follow-up in a purely diabetic obese population, and the results, similar to those of Iaconelli et al. [26], are clear that bariatric surgery induces a significant and sustainable decrease in urinary albumin excretion. These improvements in albuminuria occurred despite the fact that patients in both studies remained obese or overweight after surgery. In the present study, we demonstrate that postoperative uACR correlated significantly with both the amount of weight lost in absolute terms (pounds) and with %EWL. The postoperative urinary albumin excretion was inversely proportional to the degree of weight loss. Interestingly, there was no direct correlation between postoperative uACR or albuminuria levels and glycemic indices. These observations suggest a potential mechanism for the improvement in nephropathy after bariatric surgery other than improved glycemic control. One potential explanation for these findings is the decrease in intra-abdominal pressure that occurs with significant weight loss. Bloomfield et al. have previously reported that decompression of the abdomen leads to decreases in mean arterial pressure, central venous pressure, and renal venous pressures [10]. Doty et al. then reported, in an animal model, that decreasing renal venous pressure can reverse the pathophysiology associated with elevated pressures; the authors noted that after an intervention to lower renal venous pressures in swine, there was an increase in GFR and a decrease in plasma renin activity, serum aldosterone, and urinary protein excretion [30]. One could hypothesize that the beneficial change in renal venous pressure that occurs after weight loss surgery, likely due to a reduction in intra-abdominal pressure after loss of visceral fat, leads to improved renal perfusion with consequent deactivation of the RAAS via negative feedback pathways.

Although the sustained remission of DN that occurred in 58.3% of patients in this study is remarkable, it is also noteworthy that there was a distinct lack of progression to nephropathy in a population at high risk of this diabetic

complication. No medical therapy has been as effective in achieving an effect of this magnitude on diabetic nephropathy. As alluded to in the introduction, strict glycemic control achieved through intensive medical therapy has a beneficial effect on urinary protein excretion, although it has not been reported to affect mortality or microvascular complications with the exception of a mild-moderate decrease in albuminuria (The Veterans Affairs Diabetes Trial [VADT] and the Action in Diabetes and Vascular Disease: Preterax and Diamicron MR Controlled Evaluation [ADVANCE] trial) [31,32]. This latter beneficial effect is also at the expense of significantly higher rates of severe hypoglycemia.

One of the most interesting findings from this study was not only that remission of albuminuria was associated with diabetes remission but that there was a significant correlation between postoperative uACR and systolic blood pressure. Even among patients who were not taking renoprotective antihypertensive medications postoperatively, the uACR remained in the low/normal range in this subgroup of patients. This probably reflects the fact that these patients experienced remission of preoperative hypertension, as well as improved glycemic control, after bariatric surgery, so all of their antihypertensive medications were discontinued, including an ACEi or ARB. Nonetheless, it reflects an independent beneficial effect of surgically induced weight loss on DN, and not just an effect of renoprotective medications that could reduce urinary protein excretion.

One of the major limitations of bariatric surgery literature with respect to its effect on T2DM is the lack of long-term follow-up. Many studies have reported excellent remission rates at 1–2 years [19,33]. Of the few publications reporting postsurgical diabetes remission rates after at least 2 years of follow-up [26,34], none have defined remission as strictly as is recommended by the ADA [25]. Sjostrom et al. reported a diabetes recovery rate of 36% at 10 years [34]. Even with the application of more stringent criteria to define diabetes remission, the present study found a 44% remission rate at 5 years and a further diabetes improvement rate of 33%. Further long-term prospective studies are needed to support these findings and to better identify the long-term effects of bariatric surgery on diabetes.

The strengths of this study are the study numbers, duration of follow-up, standards used to measure and define albuminuria, and the generalizability of this data to current bariatric practices given that RYGB was the most commonly performed procedure. Previous groups evaluating renal function after bariatric surgery have largely relied on a decrease in creatinine clearance or serum creatinine as markers of improvement in renal function. These are not sensitive indicators of nephropathy, however [35]. The uACR is a more sensitive and specific indicator of early nephropathy [36]. Currently, the National Kidney Foundation recommends the use of uACR, obtained under standardized conditions (first-voided, morning, midstream

specimen), to detect microalbuminuria. Given that it is also an indicator of subclinical cardiovascular disease [7], the findings from this study further emphasize the importance and benefits of reducing albuminuria through weight loss.

This study had some limitations, particularly the small number of participants, the low follow-up rate (50.5%), the retrospective nature of the data analysis, and the lack of a control group. The variability in weight loss among the different bariatric procedures used is also a factor that could be better controlled in a prospective study using a single procedure. The numbers in this series were too small to allow for comparisons of DN remission rates between different bariatric procedures and also did not allow for regression analyses. Two potential control groups would be informative in this setting: a cohort of obese patients with diabetes treated with intensive medical treatment and a cohort of obese patients without diabetes who also underwent bariatric surgery. The ability to compare nephropathy progression in these other cohorts would help to better determine the true effect of bariatric surgery on diabetic and obesity-related nephropathy.

## Conclusion

Although this is a pilot study, the findings suggest that bariatric surgery can induce a significant and sustainable improvement in T2DM and improve or delay the development of microvascular complications such as nephropathy. Considering that diabetes is often a progressive disease, these results are clinically important and warrant further investigation. Larger prospective studies, with longer follow-up, are necessary to further examine the effect of different bariatric procedures on renal parameters and other end-organ complications of diabetes.

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## Disclosure

*The authors have no commercial associations that might be a conflict of interest in relation to this article.*

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