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Long-term impact of bariatric surgery in diabetic nephropathy

LeAnne Young[1] · Zubaidah Nor Hanipah[2,3] · Stacy A. Brethauer[1,2] · Philip R. Schauer[1,2] · Ali Aminian[1,2]

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Abstract

Background Bariatric surgery has been shown to improve and resolve diabetes. However, limited literature about its impact on end-organ complications of diabetes is available. The aim of this study was to examine the long-term effect of bariatric surgery on albuminuria.

Methods We studied 101 patients with pre-operative diabetes and albuminuria [defined as urine albumin:creatinine ratio (uACR) > 30 mg/g] who underwent bariatric surgery at an academic center from 2005 to 2014.

Results Fifty-seven patients (56%) were female with a mean age of 53 (± 11) years. The mean pre-operative BMI and glycated hemoglobin (HbA1c) were 43.1 (± 7.6) kg/m^2 and 8.4 (± 1.8)%, respectively. The median pre-operative uACR was 80.0 (45.0–231.0) mg/g. Bariatric procedures included Roux-en-Y gastric bypass (n = 75, 74%) and sleeve gastrectomy (n = 26, 26%). The mean follow-up period was 61 (± 29) months. At last follow-up, the mean BMI was 33.8 (± 8.3) kg/m^2. The overall glycemic control improved after bariatric surgery. At last follow-up, 73% had good glycemic control (HbA1c < 7%) and 27% met diabetes remission criteria. The mean HbA1c at last follow-up was 6.7 (± 1.0)% and the median uACR was 30 (IQR 7–94) mg/g. Albuminuria improved in 77% and resolved in 51% of patients at long-term.

Conclusions Bariatric surgery has a significantly positive impact on albuminuria in patients with obesity and type 2 diabetes. Our data showed almost an 80% improvement in albuminuria at the short- and long-term period after bariatric surgery.

Keywords Bariatric surgery · Diabetes · Albuminuria · Nephropathy · Gastric bypass · Sleeve gastrectomy

Diabetic nephropathy affects about 20–30% of patients with type 2 diabetes (T2D) [1] and is one of the leading causes of end-stage renal disease (ESRD) [2]. ESRD causes significant morbidity and mortality, increases health care costs [3], and decreases quality of life [4]. Unfortunately, as the number of patients with T2D rises, the number of patients with ESRD is also expected to rise [5].

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✉ Ali Aminian
AMINIAA@ccf.org

1 Cleveland Clinic Lerner of College of Medicine, Cleveland, OH, USA

2 Department of General Surgery, Bariatric and Metabolic Institute, Cleveland Clinic, Mail Code M61, 9500 Euclid Avenue, Cleveland, OH 44195, USA

3 Department of Surgery, Faculty of Medicine and Health Sciences, University Putra Malaysia, Serdang, Selangor, Malaysia

Obesity is a major risk factor for developing T2D [6]. Besides T2D, obesity is also associated with the development of chronic kidney disease and ESRD [7–9]. Obesity independently causes kidney disease via the proposed mechanisms of increased glomerular filtration, increased sodium handling in the proximal tubule, and increased sympathetic activation of the renin–angiotensin system [10]. Studies have also demonstrated a relationship between obesity and markers of kidney damage such as the urinary albumin:creatinine ratio (uACR) [11].

Bariatric surgery is proven to induce sustainable weight loss [12–14] and improvements in T2D [15–19]. However, the impact of bariatric and metabolic surgery on end-organ complications of T2D is less characterized. Long-term glycemic control has shown to decrease the risk of ESRD [20]. However, achieving glycemic control with lifestyle changes and medications is difficult. Currently, only about half of patients are able to reach the American Diabetes Association's (ADA) glycemic control goal of HbA1c < 7% [21]. Accordingly, diabetes and surgery organizations including the ADA have recently incorporated bariatric surgery as a part of the treatment algorithm for T2D patients with obesity [22, 23].

Weight loss can decrease microalbuminuria and proteinuria through a variety of proposed mechanisms such as blood pressure (BP) control, improved insulin sensitivity, and decreased activation of the renin–angiotensin–aldosterone system [24]. Recent studies have begun to examine the effect of bariatric surgery on renal function [25]. Upala et al. [26] conducted a meta-analysis of 15 studies examining the effect of bariatric surgery on urinary albumin excretion in diabetic nephropathy. Bariatric surgery was shown to significantly improve albuminuria as measured by the uACR. However, these studies were limited by small sample size or by shorter-term follow-up period [26]. Thus, the primary aim of our study was to look at the long-term renoprotective effects of bariatric surgery in T2D patients with pre-operative albuminuria.

Methods

After institutional review board (IRB) approval, a retrospective study was conducted at a single academic center from 2005 to 2014. All patients with T2D and pre-operative albuminuria were included and followed for at least 12 months after the bariatric procedures.

Patients' weight, body mass index (BMI), BP, glycated hemoglobin (HbA1c), serum creatinine, uACR, and diabetes medications were documented from the pre-operative period through the most recent follow-up.

Albuminuria was defined as uACR of > 30 mg/g [27]. Resolution of albuminuria was defined as uACR of 30 mg/g or less at the follow-up period. Macroalbuminuria was defined by uACR > 300 mg/g [27].

Diabetes remission was defined as HbA1c level < 6.5% or FBG < 7.0 mmol/L (126 mg/dL) without the use of any diabetes medications [28]. Glycemic improvement was defined as a reduction in HbA1c by > 1%, reduction in fasting blood glucose (FBG) by > 1.4 mmol/L (25 mg/dL), or reduction in HbA1c and FBG accompanied by a decrease in diabetic medication requirements as has previously been published [29–31]. Glycemic control was defined as HbA1c < 7% with or without medication use according to the ADA's guidelines [22]. Hypertension control was defined as BP < 140/90 mmHg.

Percentage total weight loss (%TWL) was calculated as [(operative weight − follow-up weight)/(operative weight)] × 100. The %EWL was calculated as [(operative weight − follow-up weight)/(operative weight − ideal weight)] × 100, with ideal body weight based on a BMI of 25 kg/m^2. Short-term follow-up was defined as post-bariatric surgery follow-up between 1 and 3 years. Long-term follow-up was defined as post-bariatric surgery follow-up between 4 and 10 years.

Data were summarized as mean with standard deviation and median with interquartile range (IQR) for continuous variables, and counts with percentages for categorical variables. Means were compared with the paired *t* test, medians were compared with the Wilcoxon-signed rank, and percentages were compared with the 2-sample test for binomial proportions. In all tests, $p < 0.05$ was considered statistically significant.

Results

Out of 1392 diabetic patients, 101 (7.2%) patients had pre-operative albuminuria with at least 12 months of uACR follow-up for albuminuria after bariatric surgery. Of this group, 57 (56%) were female with a mean age of 53 (± 11) years. The majority of patients were white (78%), followed by African American (20%), and Hispanic (2%). The mean pre-operative weight and BMI were 124.0 (± 24.6) kg and 43.1 (± 7.6) kg/m^2, respectively. The mean pre-operative HbA1c was 8.5% (± 1.8) and 76% had poor glycemic control with HbA1c ≥ 7%. Ninety-four patients (93%) had hypertension before surgery. Bariatric procedures performed were Roux-en-Y gastric bypass (RYGB, $n = 75$, 74%) and sleeve gastrectomy (SG, $n = 26$, 26%). The baseline demographics are summarized in Table 1.

The mean follow-up period was 61 (± 29) months. While all patients had short-term data (1–3 years after surgery), the long-term uACR data were available on 71 patients (71%). At short-term follow-up, the mean BMI decreased from 43.1 (± 7.6) kg/m^2 at baseline to 33.6 (± 7.7) kg/m^2. These changes were maintained at long-term follow-up (33.8 ± 8.3 kg/m^2). The RYGB patients had better %TWL and %EWL at last follow-up compared to the SG patients. The changes in body weight and BMI based on bariatric procedures (RYGB and SG) are summarized in Table 2.

The overall glycemic control improved during the short-term follow-up period and was maintained over the long-term follow-up period (Table 3; Fig. 1). The mean HbA1c of the whole cohort was 6.8 (± 1.4)% at short-term and 6.7 (± 1.0)% at long-term follow-up. The number of diabetes medications improved after bariatric surgery. Sixty-two (61.4%) patients required insulin at baseline but almost 50% of these patients were not on insulin after bariatric surgery at the last follow-up. Overall, the percentage of patients who were not taking any diabetes medications (including insulin) was 44% in the short-term follow-up period and 33% in the long-term follow-up period.

There were significant changes in uACR after bariatric surgery (Table 3). The median pre-operative uACR was 80 (IQR 45–240) mg/g. During the short-term follow-up period,

Table 1 Baseline characteristics of patients with albuminuria before bariatric surgery

Variable	Whole cohort ($n=101$)	Gastric bypass ($n=75$)	SG ($n=26$)
Female, *n* (%)	57 (56%)	42 (56%)	15 (58%)
Age (years)	53.3 (±10.9)	53.5 (±11.1)	53.0 (±10.2)
BMI (kg/m^2)	43.1 (±7.6)	42.6 (±6.6)	44.6 (±9.7)
Duration of diabetes (years), median (IQR)	9.0 (6.0–14.0)	9.0 (5.5–14.0)	10.5 (6.3–15.0)
Number of diabetes medications, median (IQR)	1 (1–2)	1 (1–2)	1 (0–2)
On insulin therapy, *n* (%)	62 (61%)	46 (61%)	16 (62%)
HbA1c (%)	8.4 (±1.8)	8.4 (±1.7)	8.7 (±2.1)
Poor glycemic control, *n* (%)	77 (76%)	59 (79%)	18 (69%)
Serum creatinine (mg/dL), median (IQR)	0.9 (0.7–1.2)	0.9 (0.7–1.2)	1.0 (0.8–1.1)
UACR (mg/g), median (IQR)	80.0 (45.0–231.0)	91.0 (50.5–272.0)	57.0 (39.3–167.3)
Macroalbuminuria (%)	22 (22%)	18 (24%)	4 (15%)
On ACEI or ARB, *n* (%)	88 (87%)	67 (90%)	21 (81%)

Poor glycemic control defined as HbA1c ≥ 7%; Macroalbuminuria defined as uACR > 300 mg/g

Means reported as mean ± SD; medians reported as median (IQR)

HbA1c glycated hemoglobin, *ACEI* angiotensin converting enzyme blocker, *ARB* angiotensin receptor blocker, *IQR* interquartile range

Table 2 Weight loss data by procedure type in patients with albuminuria before bariatric surgery

Variable	Whole cohort ($n=101$)	Gastric bypass ($n=75$)	SG ($n=26$)
BMI (kg/m^2)			
Baseline	43.1 (±7.6)	42.6 (±6.7)	44.6 (±9.9)
Short-term	33.6 (±7.7)	32.3 (±6.2)	37.6 (±10.2)
Long-term	33.8 (±8.3)	32.4 (±6.9)	37.6 (±10.7)
TWL (%)			
Short-term	22.1 (±10.1)	24.1 (±9.1)	16.3 (±10.9)
Long-term	22.2 (±11.9)	24.5 (±11.0)	16.0 (±12.2)
EWL (%)			
Short-term	59.6 (±35.7)	65.3 (±34.4)	43.3 (±34.7)
Long-term	58.4 (±36.1)	64.1 (±33.2)	42.5 (±39.7)

All values are mean ± SD

Short-term: 1–3 years after surgery; Long-term: 4–10 years after surgery

the median uACR decreased to 31 (IQR 9–31) mg/g. These changes were maintained during the long-term follow-up period, as the median uACR at last follow-up was 30 (IQR 7–93.5) mg/g. At last follow-up, 51% patients had a resolution of the albuminuria (uACR < 30 mg/g) (Fig. 1). These changes were observed even with a significant reduction in the number of patients who were taking angiotensin converting enzyme inhibitor or angiotensin II receptor blocker medications after bariatric surgery. There was no significant difference in uACR changes between RYGB and SG patients.

Twenty-two patients had baseline macroalbuminuria (RYGB $n=18$, SG $n=4$). In this subgroup at short-term follow-up, 17 (77%) patients had resolution or improvement of uACR. At long-term follow-up ($n=12$), 10 patients had resolution or improvement of albuminuria. All the SG patients had improvement of uACR at both short- and long-term follow-up.

There were no significant changes in the median serum creatinine of the whole cohort after bariatric surgery. Twenty-four (24.4%) patients had high baseline serum creatinine, as defined by creatinine > 1.2 mg/dL in males and > 1.1 mg/dL in females [32]. In the short-term follow-up of this subgroup ($n=23$), serum creatinine was normalized or improved in 13 patients. In the long-term follow-up of this subgroup ($n=16$), serum creatinine was normalized or improved in six patients.

Discussion

To date, this is the largest series on improvement in albuminuria in diabetic patients after bariatric surgery, with a mean follow-up period of 61 (±29) months. There was improvement of uACR after bariatric surgery in 77.1% of patients with pre-operative albuminuria. Resolution of albuminuria was seen in 51% of patients in the long-term follow-up period after bariatric surgery.

The presence of albuminuria is a strong predictor of renal and cardiovascular risk in patients with T2D and hypertension [33]. Multiple studies have shown that decreasing the level of albuminuria (mainly by inhibition of the renin–angiotensin–aldosterone system) can reduce the risk of adverse renal and cardiovascular outcomes. For example, in the Prevention of Renal and Vascular End-Stage Disease

Table 3 Long-term and short-term outcomes of bariatric surgery in patients with albuminuria

Variable	Baseline	Short-term	Long-term	p value	
				Short-term versus baseline	Long-term versus baseline
Cardiometabolic and renal outcomes					
Mean HbA1c (%)					
Whole cohort	8.4 (± 1.8)	6.8 (± 1.4)	6.7 (± 1.0)	< 0.0001	< 0.0001
Gastric bypass	8.4 (± 1.7)	6.8 (± 1.4)	6.7 (± 0.9)	< 0.0001	< 0.0001
Sleeve gastrectomy	8.7 (± 2.1)	7.1 (± 1.4)	6.8 (± 1.4)	0.0034	0.0011
UACR (mg/g), median (IQR)					
Whole cohort	80.0 (45.0–231.0)	31.0 (9.0–31.0)	30.0 (7.0–93.5)[a]	< 0.0001	< 0.0001
Gastric bypass	91.0 (50.5–272.0)	34.0 (9.3–113.0)	35.5 (7.0–94.0)	< 0.0001	< 0.0001
Sleeve gastrectomy	57.0 (39.3–167.3)	24.5 (7.0–110.0)	26.0 (9.0–75.5)	0.016	0.024
Serum creatinine (mg/dL), median (IQR)	0.9 (0.7–1.2)	0.9 (0.8–1.2)	1.0 (0.8–1.3)	0.82	0.15
Systolic BP (mmHg)	133 (± 19)	130 (± 15)	132 (± 17)	0.17	0.91
Diastolic BP (mmHg)	75 (± 10)	75 (± 12)	72 (± 10)	0.51	0.041
Medications					
No. of diabetes drugs, median (IQR)	1 (1–2)	0 (0–1)	1 (0–2)	< 0.0001	< 0.0001
No. on insulin therapy/no. available (%)	62/101 (61%)	28/101 (28%)	23/84 (27%)	< 0.0001	< 0.0001
No. without diabetes medication/no. available (%)	4/101 (4%)	44/101 (44%)	28/84 (33%)	< 0.0001	< 0.0001
No. on ACEI or ARB/no. available (%)	88/101 (87%)	54/101 (53%)	49/84 (58%)	< 0.0001	< 0.0001

Means reported as means ± SD

Short-term: 1–3 years after surgery; Long-term: 4–10 years after surgery

HbA1c glycated hemoglobin, *ACEI* angiotensin converting enzyme blocker, *ARB* angiotensin receptor blocker, *IQR* interquartile range

[a]71 out of 101 patients had long-term UACR data

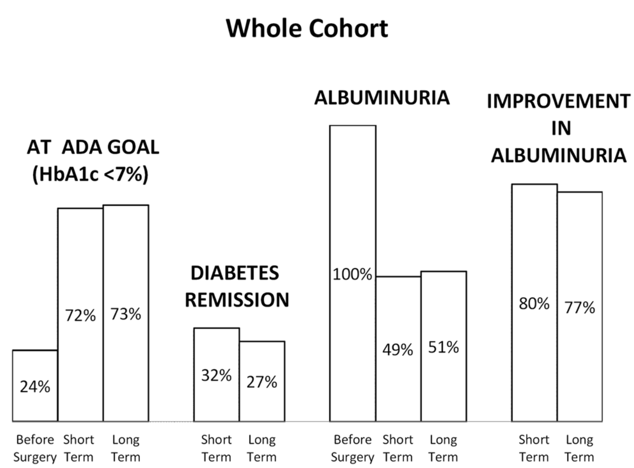

Fig. 1 Short- and long-term glycemic and renal outcomes of bariatric surgery in patients with albuminuria based on the ADA goals for glycemic (HbA1c < 7%) and albuminuria (< 30 mg/g)

Intervention Trial (PREVEND IT), treatment with fosinopril (an angiotensin converting enzyme inhibitor) in patients with albuminuria led to a significant reduction in urinary albumin excretion (26%) and a trend toward less cardiovascular events (40%) [34]. In the Losartan Intervention for Endpoint Reduction in Hypertension (LIFE) study, patients who were able to significantly decrease their uACR at 1 year experienced a reduced risk of myocardial infarction, stroke, and cardiovascular mortality compared with patients who were not able to decrease their uACR [35]. Furthermore, in the Irbestartan in Patients with Type 2 Diabetes and Microalbuminuria (IRMA-2) study, administration of irbesartan (an angiotensin receptor blocker) in patients with T2D, hypertension, and microalbuminuria, could reduce the risk of progressing to overt nephropathy (overt proteinuria) [36].

Current literature on albuminuria after bariatric surgery is limited by small sample sizes and/or short-term follow-up. Sample sizes are often less than 100 patients [37, 38] and follow-up is often limited to 12–24 months [39–41]. Amor et al. [39] found that uACR decreased from 85.7 mg/g at baseline to 42.2 mg/g 1 year after bariatric surgery. Out of the 44 patients with pre-operative albuminuria, 59% (at 12 months) and 77% (at 24 months) had resolution of albuminuria. Heneghan et al. [25] showed a 58% reduction in albuminuria 5 years after bariatric surgery. Miras et al. [42] compared diabetic microvascular complications between bariatric surgery and medical treatment groups. Of the patients with pre-operative albuminuria, the surgical group patients ($n = 30$) had improvements in albuminuria at

12 months while the medical group patients ($n=10$) did not have significant changes in albuminuria at 1 year follow-up. The median uACR of the surgical group decreased from 8.5 mg/mmol (IQR 5.7–17.4) at baseline to 1.7 mg/mmol (IQR 1.0–6.1) at 12 month follow-up.

Bariatric surgery can also prevent the development of albuminuria. One of the largest studies with a median follow-up of 10 years (2010 bariatric surgery patients and 2037 non-surgery patients) showed that bariatric surgery patients had a decreased incidence of developing albuminuria (9.4/1000 person-years vs 20.4/1000 person-years) as compared to the nonsurgical group [43]. This study excluded patients with pre-operative albuminuria.

There are likely many mechanisms underlying the improvements in albuminuria in patients with T2D after bariatric surgery, one of which may be the improved glycemic control after bariatric surgery. Our findings showed significant improvements in HbA1c. Only 24% of patients achieved the ADA goal of HbA1c < 7% before surgery, but 73% achieved the ADA goal in the long-term follow-up period. There was also a significant decrease in diabetes medications usage after bariatric surgery. 61% of patients were on insulin at baseline but only 27% were on insulin at long-term follow-up. While the RYGB group achieved greater weight loss than the SG group in our cohort, we did not observe any significant differences in HbA1c and uACR between surgical groups. Of note, only 26 patients underwent SG which may have impacted the statistical analysis.

The UK Prospective Diabetes Study assessed risk factors for renal dysfunction in patients with T2D, and found that development of albuminuria and renal impairment were independently associated with increased systolic BP and HbA1c [44]. We did not observe significant changes in BP in our cohort during the short-term and long-term follow-up periods.

Despite being the largest series with long-term follow-up on diabetic patients with albuminuria before bariatric surgery, this study has several limitations due to its retrospective single center design and lack of a medical control group. Only patients with pre-operative uACR and post-operative follow-up data were included in this study. Thus, there is the possibility that we did not capture all patients with albuminuria. Follow-up data are also limited in some patients, especially data on HbA1c, BP, uACR, and serum creatinine. However, 71 patients (71%) in this cohort had long-term uACR follow-up. In our small series, in the subset of patients with abnormal creatinine at baseline, serum creatinine was normalized or improved in 13 (out of 23) patients at short term follow up. In the long-term follow-up of this subset ($n=16$), serum creatinine was normalized or improved in six patients. While our series clearly showed the positive impact of bariatric surgery on albuminuria (80% improvement and 50% resolution), we would need a large sample size to study the impact of post-surgical reduction in albuminuria on the renal function and future cardiovascular events. In addition, future prospective studies, preferably randomized clinical trials comparing albuminuria resolution in bariatric surgery and medical control groups are warranted.

Conclusion

Findings of this study suggest that bariatric surgery in diabetic patients with pre-existing nephropathy can improve albuminuria. Additionally, 30% of patients had diabetes remission and 70% of the patients achieved the ADA goal for HbA1c < 7% at last follow-up after bariatric surgery. Finally, our long-term data showed almost a 50% resolution and 80% improvement of albuminuria after bariatric surgery.

Compliance with ethical standards

Disclosures Authors Young, Nor Hanipah, Brethauer, Schauer, and Aminian have no conflicts of interest or financial ties to disclose.

References

1. Molitch ME, DeFronzo RA, Franz MJ, Keane WF, Mogensen CE, Parving H-H, Steffes MW, American Diabetes Association (2004) Nephropathy in diabetes. Diabetes Care 27(Suppl 1):S79–S83. https://doi.org/10.2337/DIACARE.27.2007.S79
2. Postma MJ, de Zeeuw D (2009) The economic benefits of preventing end-stage renal disease in patients with type 2 diabetes mellitus. Nephrol Dial Transplant 24:2975–2983. https://doi.org/10.1093/ndt/gfp352
3. Damien P, Lanham HJ, Parthasarathy M, Shah NL (2016) Assessing key cost drivers associated with caring for chronic kidney disease patients. BMC Health Serv Res 16:690. https://doi.org/10.1186/s12913-016-1922-4
4. Romagnani P, Remuzzi G, Glassock R, Levin A, Jager KJ, Tonelli M, Massy Z, Wanner C, Anders H-J (2017) Chronic kidney disease. Nat Rev Dis Prim 3:17088. https://doi.org/10.1038/nrdp.2017.88
5. Burrows NR, Hora I, Geiss LS, Gregg EW, Albright A (2017) Incidence of end-stage renal disease attributed to diabetes among persons with diagnosed diabetes—United States and Puerto Rico, 2000–2014. MMWR Morb Mortal Wkly Rep 66:1165–1170. https://doi.org/10.15585/mmwr.mm6643a2
6. González-Muniesa P, Mártinez-González M-A, Hu FB, Després J-P, Matsuzawa Y, Loos RJF, Moreno LA, Bray GA, Martinez JA (2017) Obesity. Nat Rev Dis Prim 3:17034. https://doi.org/10.1038/nrdp.2017.34
7. Wang Y, Chen X, Song Y, Caballero B, Cheskin LJ (2008) Association between obesity and kidney disease: a systematic review and meta-analysis. Kidney Int 73:19–33. https://doi.org/10.1038/sj.ki.5002586
8. Hsu C, McCulloch CE, Iribarren C, Darbinian J, Go AS (2006) Body mass index and risk for end-stage renal disease. Ann Intern Med 144:21. https://doi.org/10.7326/0003-4819-144-1-200601030-00006
9. Garofalo C, Borrelli S, Minutolo R, Chiodini P, De Nicola L, Conte G (2017) A systematic review and meta-analysis suggests

obesity predicts onset of chronic kidney disease in the general population. Kidney Int 91:1224–1235. https://doi.org/10.1016/j.kint.2016.12.013
10. Bayliss G, Weinrauch LA, D'Elia JA (2012) Pathophysiology of obesity-related renal dysfunction contributes to diabetic nephropathy. Curr Diab Rep 12:440–446. https://doi.org/10.1007/s11892-012-0288-1
11. Rossi M, Nicolucci A, Pellegrini F, Comaschi M, Ceriello A, Cucinotta D, Giorda C, Pomili B, Valentini U, Vespasiani G, De Cosmo S (2010) Obesity and changes in urine albumin/creatinine ratio in patients with type 2 diabetes: the DEMAND study. Nutr Metab Cardiovasc Dis. https://doi.org/10.1016/j.numecd.2009.02.013
12. Buchwald H, Avidor Y, Braunwald E, Jensen MD, Pories W, Fahrbach K, Schoelles K (2004) Bariatric surgery. JAMA 292:1724. https://doi.org/10.1001/jama.292.14.1724
13. Carlsson LMS, Peltonen M, Ahlin S, Anveden Å, Bouchard C, Carlsson B, Jacobson P, Lönroth H, Maglio C, Näslund I, Pirazzi C, Romeo S, Sjöholm K, Sjöström E, Wedel H, Svensson P-A, Sjöström L (2012) Bariatric surgery and prevention of type 2 diabetes in Swedish obese subjects. N Engl J Med 367:695–704. https://doi.org/10.1056/NEJMoa1112082
14. Maciejewski ML, Arterburn DE, Van Scoyoc L, Smith VA, Yancy WS, Weidenbacher HJ, Livingston EH, Olsen MK, Olsen MK (2016) Bariatric surgery and long-term durability of weight loss. JAMA Surg 151:1046–1055. https://doi.org/10.1001/jamasurg.2016.2317
15. Schauer PR, Bhatt DL, Kirwan JP, Wolski K, Aminian A, Brethauer SA, Navaneethan SD, Singh RP, Pothier CE, Nissen SE, Kashyap SR (2017) Bariatric surgery versus intensive medical therapy for diabetes—5-year outcomes. N Engl J Med 376:641–651. https://doi.org/10.1056/NEJMoa1600869
16. Aminian A (2018) Sleeve gastrectomy: metabolic surgical procedure of choice? Trends Endocrinol Metab 29:531–534
17. Aminian A, Brethauer SA, Andalib A, Nowacki AS, Jimenez A, Corcelles R, Hanipah ZN, Punchai S, Bhatt DL, Kashyap SR, Burguera B, Lacy AM, Vidal J, Schauer PR (2017) Individualized metabolic surgery score. Ann Surg 266:650–657. https://doi.org/10.1097/SLA.0000000000002407
18. Ardestani A, Rhoads D, Tavakkoli A (2015) Insulin cessation and diabetes remission after bariatric surgery in adults with insulin-treated type 2 diabetes. Diabetes Care 38:dc141751. https://doi.org/10.2337/dc14-1751
19. Aminian A, Daigle CR, Romero-Talamás H, Kashyap SR, Kirwan JP, Brethauer SA, Schauer PR (2014) Risk prediction of complications of metabolic syndrome before and 6 years after gastric bypass. Surg Obes Relat Dis 10:576–582. https://doi.org/10.1016/j.soard.2014.01.025
20. Wong MG, Perkovic V, Chalmers J, Woodward M, Li Q, Cooper ME, Hamet P, Harrap S, Heller S, MacMahon S, Mancia G, Marre M, Matthews D, Neal B, Poulter N, Rodgers A, Williams B, Zoungas S, ADVANCE-ON Collaborative Group (2016) Long-term benefits of intensive glucose control for preventing end-stage kidney disease: ADVANCE-ON. Diabetes Care 39:694–700. https://doi.org/10.2337/dc15-2322
21. Resnick HE, Foster GL, Bardsley J, Ratner RE (2006) Achievement of American Diabetes Association clinical practice recommendations among U.S. adults with diabetes, 1999–2002: the National Health and Nutrition Examination Survey. Diabetes Care 29:531–537. https://doi.org/10.2337/DIACARE.29.03.06.DC05-1254
22. American Diabetes Association (2018) Standards of medical care in diabetes—2018. Diabetes Care 41:S3. https://doi.org/10.2337/dc18-Sppc01
23. Rubino F, Nathan DM, Eckel RH, Schauer PR, Alberti KGMM, Zimmet PZ, Del Prato S, Ji L, Sadikot SM, Herman WH, Amiel SA, Kaplan LM, Taroncher-Oldenburg G, Cummings DE, Delegates of the 2nd Diabetes Surgery Summit (2016) Metabolic surgery in the treatment algorithm for type 2 diabetes: a joint statement by international diabetes organizations. Diabetes Care 39:861–877. https://doi.org/10.2337/dc16-0236
24. Morales E, Praga M (2012) The effect of weight loss in obesity and chronic kidney disease. Curr Hypertens Rep 14:170–176. https://doi.org/10.1007/s11906-012-0247-x
25. Heneghan HM, Cetin D, Navaneethan SD, Orzech N, Brethauer SA, Schauer PR (2013) Effects of bariatric surgery on diabetic nephropathy after 5 years of follow-up. Surg Obes Relat Dis 9:7–14. https://doi.org/10.1016/j.soard.2012.08.016
26. Upala S, Wijarnpreecha K, Congrete S, Rattanawong P, Sanguankeo A (2016) Bariatric surgery reduces urinary albumin excretion in diabetic nephropathy: a systematic review and meta-analysis. Surg Obes Relat Dis 12:1037–1044. https://doi.org/10.1016/j.soard.2015.11.019
27. de Jong PE, Curhan GC (2006) Screening, monitoring, and treatment of albuminuria: public health perspectives. J Am Soc Nephrol 17:2120–2126. https://doi.org/10.1681/ASN.2006010097
28. Buse JB, Caprio S, Cefalu WT, Ceriello A, Del Prato S, Inzucchi SE, McLaughlin S, Phillips GL, Robertson RP, Rubino F, Kahn R, Kirkman MS (2009) How do we define cure of diabetes? Diabetes Care 32:2133–2135. https://doi.org/10.2337/dc09-9036
29. Brethauer SA, Kim J, el Chaar M, Papasavas P, Eisenberg D, Rogers A, Ballem N, Kligman M, Kothari S, ASMBS Clinical Issues Committee (2015) Standardized outcomes reporting in metabolic and bariatric surgery. Surg Obes Relat Dis 11:489–506. https://doi.org/10.1016/j.soard.2015.02.003
30. Aminian A, Brethauer SA, Andalib A, Punchai S, Mackey J, Rodriguez J, Rogula T, Kroh M, Schauer PR (2016) Can sleeve gastrectomy "cure" diabetes? Long-term metabolic effects of sleeve gastrectomy in patients with type 2 diabetes. Ann Surg 264:674–681. https://doi.org/10.1097/SLA.0000000000001857
31. Brethauer SA, Aminian A, Romero-Talamás H, Batayyah E, Mackey J, Kennedy L, Kashyap SR, Kirwan JP, Rogula T, Kroh M, Chand B, Schauer PR (2013) Can diabetes be surgically cured? Long-term metabolic effects of bariatric surgery in obese patients with type 2 diabetes mellitus. Ann Surg 258:628–636. https://doi.org/10.1097/SLA.0b013e3182a5034b
32. Chernecky C, Berger B (2013) Creatinine—serum. In: Chernecky C, Berger B (eds) Laboratory tests and diagnostic procedures, 6th edn. Elsevier, St Louis, MO, pp 266–432
33. Basi S, Fesler P, Mimran A, Lewis JB (2008) Microalbuminuria in type 2 diabetes and hypertension: a marker, treatment target, or innocent bystander? Diabetes Care 31(Suppl 2):S194–S201
34. Asselbergs FW, Diercks GF, Hillege HL, van Boven AJ, Janssen WM, Voors AA, de Zeeuw D, de Jong PE, van Veldhuisen DJ, van Gilst WH, Prevention of Renal and Vascular Endstage Disease Intervention Trial (PREVEND IT) Investigators (2004) Effects of fosinopril and pravastatin on cardiovascular events in subjects with microalbuminuria. Circulation 110:2809–2816
35. Ibsen H, Olsen MH, Wachtell K, Borch-Johnsen K, Lindholm LH, Mogensen CE, Dahlöf B, Devereux RB, de Faire U, Fyhrquist F, Julius S, Kjeldsen SE, Lederballe-Pedersen O, Nieminen MS, Omvik P, Oparil S, Wan Y (2005) Reduction in albuminuria translates to reduction in cardiovascular events in hypertensive patients: losartan intervention for endpoint reduction in hypertension study. Hypertension 45:198–202
36. Parving HH, Lehnert H, Bröchner-Mortensen J, Gomis R, Andersen S, Arner P, Irbesartan in Patients with Type 2 Diabetes and Microalbuminuria Study Group (2001) The effect of irbesartan on the development of diabetic nephropathy in patients with type 2 diabetes. N Engl J Med 345:870–878
37. Zeve JL, de M, Tomaz, Nassif CAB, Lima PAN, Sansana JH, Zeve LRZ CH (2013) Obese patients with diabetes mellitus type 2

undergoing gastric bypass in Roux-en-Y: analysis of results and its influence in complications. Arq Bras Cir Dig 26(Suppl 1):47–52
38. Kota SK, Ugale S, Gupta N, Modi KD (2012) Laparoscopic ileal interposition with diverted sleeve gastrectomy for treatment of type 2 diabetes. Diabetes Metab Syndr Clin Res Rev 6:125–131. https://doi.org/10.1016/j.dsx.2012.09.014
39. Amor A, Jiménez A, Moizé V, Ibarzabal A, Flores L, Lacy AM, Vidal J (2013) Weight loss independently predicts urinary albumin excretion normalization in morbidly obese type 2 diabetic patients undergoing bariatric surgery. Surg Endosc 27:2046–2051. https://doi.org/10.1007/s00464-012-2708-3
40. Zhang H, Di J, Yu H, Han X, Li K, Zhang P (2015) The short-term remission of diabetic nephropathy after Roux-en-Y gastric bypass in Chinese patients of T2DM with obesity. Obes Surg 25:1263–1270. https://doi.org/10.1007/s11695-015-1666-y
41. Saliba J, Kasim NR, Tamboli RA, Isbell JM, Marks P, Feurer ID, Ikizler A, Abumrad NN (2010) Roux-en-Y gastric bypass reverses renal glomerular but not tubular abnormalities in excessively obese diabetics. Surgery 147:282–287. https://doi.org/10.1016/j.surg.2009.09.017
42. Miras AD, Chuah LL, Khalil N, Nicotra A, Vusirikala A, Baqai N, Graham C, Ravindra S, Lascaratos G, Oliver N, le Roux CW (2015) Type 2 diabetes mellitus and microvascular complications 1 year after Roux-en-Y gastric bypass: a case–control study. Diabetologia 58:1443–1447. https://doi.org/10.1007/s00125-015-3595-7
43. Carlsson LMS, Romeo S, Jacobson P, Burza MA, Maglio C, Sjöholm K, Svensson P-A, Haraldsson B, Peltonen M, Sjöström L (2015) The incidence of albuminuria after bariatric surgery and usual care in swedish obese subjects (SOS): a prospective controlled intervention trial. Int J Obes 39:169–175. https://doi.org/10.1038/ijo.2014.72
44. Retnakaran R, Cull CA, Thorne KI, Adler AI, Holman RR, UKPDS Study Group (2006) Risk factors for renal dysfunction in type 2 diabetes: U.K. Prospective Diabetes Study 74. Diabetes 55:1832–1839. https://doi.org/10.2337/db05-1620