Impact of Sleeve Gastrectomy on Renal Function in Patients With Morbid Obesity: A One-Year Prospective Cohort Study

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Abstract

Background. Obesity is an independent risk factor for renal injury. A more favorable metabolic environment following weight loss may theoretically lead to improved renal function. We aimed to evaluate the evolution of renal function one year after sleeve gastrectomy in a large prospective cohort of patients with morbid obesity and assess the influence of fat-free mass (FFM) changes.

Patients and Methods. We prospectively included 563 severely obese patients admitted for sleeve gastrectomy. Patients were systematically evaluated one year after surgery. The estimated glomerular filtration rate (eGFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation. The FFM was estimated by analyzing computerized tomography (CT) scan sections from CT systematically performed two days and one year after sleeve gastrectomy to detect surgery complications.

Results. The mean age was 41.2±0.52 years. The mean body mass index was 43.5±0.3 kg/m^2 and 20.4, 30.5, and 30.7% of the included patients had type 2 diabetes, hypertension, and dyslipidemia, respectively. One hundred and fifteen patients were lost to follow-up at one-year post-surgery. The eGFR was significantly lower one year after sleeve gastrectomy than before surgery (87.8±0.9 versus 86.1±0.9, $p<0.01$). There was no difference in terms of post-surgery FFM loss between patients with an improved eGFR and those without (6.7±0.3 kg versus 6.76±0.5 kg, $p=0.9$). Furthermore, post-surgery changes in the eGFR did not correlate with the amount of FFM loss ($r=0.1$, $p=0.18$).

Conclusion. Renal function assessed by eGFR is significantly improved at one-year post-sleeve gastrectomy, independent of changes in skeletal muscle mass.

Introduction

The prevalence of obesity is increasing worldwide and it is estimated that one quarter of the world's population will be overweight or obese by 2045. Among the complications of obesity, diabetes and hypertension account for half of kidney-failure cases. Nevertheless, obesity itself is an independent risk factor for renal injury [1,2]. Obesity-related glomerulopathy is characterized by segmental and focal glomerulosclerosis associated with glomerulomegaly [3]. Chagnac et al. [4] showed that glomerular filtration and renal plasma flow are 51% higher in obese patients than healthy subjects. This may be due to vasodilatation of the afferent arteriole caused by insulin, together with vasoconstriction of the efferent arteriole favored by insulin resistance [5]. Thus, an increase in the glomerular filtration rate is an early manifestation of obesity-related renal injury.

A more favorable metabolic environment and a decrease in profibrotic factors following weight loss may theoretically lead to an improvement in renal function [6,7]. Recent studies suggest that renal function may improve in morbidly obese patients undergoing bariatric surgery [8,9], but data from large homogenous prospective cohorts are lacking. In this context, we aimed to evaluate the evolution of renal function one

year after sleeve gastrectomy in a large prospective cohort of patients with morbid obesity. The secondary aims were to assess 1) the influence of baseline renal function and changes in fat-free mass (FFM) on the post-surgery evolution of renal function and 2) the effect of the renal function status on weight loss at one-year post-surgery.

Patients And Methods

Study population

We conducted an observational prospective cohort study that included consecutive patients admitted to our institution to undergo sleeve gastrectomy between January 2013 and November 2016. As previously described [21], patients fulfilling the following criteria were considered to be eligible for this study: (i) severe obesity [body mass index (BMI) ≥ 35 kg/m^2] with at least one comorbid condition or morbid obesity alone (BMI ≥ 40 kg/m^2) not responsive to medical treatment, (ii) no medical or psychological contraindication for bariatric surgery, (iii) not currently an excessive drinker, as defined by a mean daily consumption of more than 20 g of alcohol per day for women or more than 30 g of alcohol per day for men, (iv) no long-term consumption of hepatotoxic drugs, and (v) negative screening results for chronic liver disease unrelated to obesity. All patients had a pre-operative evaluation including: (i) a biological assessment of renal function, lipid profile, nutritional status, and liver function tests, and (ii) liver ultrasound and transient elastography [21]. All patients underwent a laparoscopic single-port sleeve gastrectomy. A liver biopsy was performed during the sleeve gastrectomy as previously described [22]. The indications for liver biopsy were: (i) ultrasound results suggestive of liver steatosis or liver dysmorphia and/or (ii) abnormal liver tests and/or (iii) a macroscopically abnormal liver, as observed by the surgeons [21]. A computerized tomography (CT) scan was systematically performed two days and one year after sleeve gastrectomy to detect early and late complications of surgery. The clinical interest of routine postoperative CT scans has been previously proven [23]. It was shown that a combination of clinical surveillance and early imaging allowed prompt management of complicated cases, avoiding further morbidity. In our patients, CT scans were performed on a targeted zone of interest using a limited number of phases, thus considerably decreasing radiation exposure. Written informed consent was obtained from all participants. The study did not include minors and was conducted in accordance with the national law concerning medical investigations (Huriet Law) and the 1964 Helsinki declaration. The study protocol and consent procedure were approved by the ethics committee of the Bicêtre Hospital.

Follow-up

All patients systematically received a convocation for an evaluation one year after the bariatric surgery during a one-day hospital stay. Clinical parameters included the amount of weight loss [percentage of total weight loss (%TWL), percentage of excess weight loss (%EWL)] and persistence of comorbidities (hypertension, diabetes, dyslipidemia). Biological assessment included all parameters tested at inclusion (renal function, lipid profile, nutritional status, and liver function tests).

The primary outcome was the change in estimated glomerular filtration rate (eGFR), calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation at one-year post-surgery. A subgroup analysis of patients with hypofiltration (eGFR < 90 ml/min/1.73 m^2) and hyperfiltration (eGFR > 120 ml/min/1.73 m^2) was also performed. The secondary outcome was the evolution of the eGFR as a function of changes in FFM at one-year post-sleeve gastrectomy.

Renal function evaluation

Renal function was assessed by calculating the eGFR. The use of eGFR equations that rely on total body weight, such as the Cockcroft and Gault formula, is not appropriate for extremely obese patients, as they overestimate the eGFR. In our study, we used CKD-EPI equations, which do not include total body weight:

For women: ≤ 62 (≤ 0.7) eGFR (ml/min/1.73 m^2) = 144 x (creatinine/0.7) $^{-0.329}$ x $(0.993)^{age}$

> 62 (> 0.7) eGFR (ml/min/1.73 m^2) = 144 x $(creatinine/0.7)^{-1.209}$ x $(0.993)^{age}$

For men: ≤ 80 (≤ 0.9) eGFR (ml/min/1.73 m^2) = 141 x $(creatinine/0.9)^{-0.411}$ x $(0.993)^{age}$

> 80 (> 0.9) eGFR (ml/min/1.73 m^2) = 141 x $(creatinine/0.9)^{-1.209}$ x $(0.993)^{age}$

Normal eGFR values are considered to be between 90 and 120 ml/min/1.73 m^2. In our study, an eGFR value < 90 ml/min/1.73 m^2 was considered to indicate impaired renal function (hypofiltration). An eGFR value > 120 ml/min /1.73 m^2 was considered to be representative of glomerular hyperfiltration.

Fat-free mass (FFM) evaluation

The skeletal muscle cross-sectional area at the third lumbar vertebrae (SMA, cm^2) was measured on the CT scan, performed two days and one year after sleeve gastrectomy, by a single reader, as previously described [20]. The FFM was estimated using the following regression equation previously validated by Moutzarkis *et al.*[24]: FFM (kg) = 0.3×SMA (cm^2) + 6.06.

Statistical analysis

The χ^2 test was used for comparisons between qualitative variables, which are described by the frequency and percentage of each class. Student T tests were used to compare normally distributed quantitative variables and Mann-Whitney and Wilcoxon tests for those for which the distribution did not follow a normal distribution. The distributions of the quantitative variables are described by the mean ± the standard error of the mean (SEM). All comparisons were performed using bilateral tests with an alpha risk of 5%. Simple correlations between variables were evaluated using Spearman Correlation Tests. An analysis of the different groups of patients was performed before and after the bariatric surgery to

evaluate: 1) the evolution of renal function according to the initial eGFR and 2) the weight loss and evolution of the comorbidities based on whether or not there was pre-existing renal insufficiency.

Results

Patient characteristics

In total, 563 patients with an indication for sleeve gastrectomy fulfilled the inclusion criteria. The mean age of the patients was 41.2 ± 0.52 years and a large majority were women (77.1%). The mean BMI was 43.5 ± 0.3 kg/m^2 and 20.4, 30.5, and 30.7% of the included patients had type 2 diabetes, hypertension, or dyslipidemia, respectively. The characteristics of the study population are described in Table 1.

Among the qualifying patients, 115 did not return for the follow-up visit at one-year post-surgery. The patients lost to follow-up were younger (37.3 ± 1.3 versus 42.2 ± 0.6, $p < 0.0001$), had a lower frequency of dyslipidemia (23.5% versus 32.6%, $p = 0.05$) and a higher baseline eGFR (94.3 ± 2.3 versus 86.1 ± 2.3, $p < 0.0001$) than those returning for the one-year post-surgery evaluation. The baseline characteristics of patients who returned for the one-year follow-up and those who did not are reported in Table 2.

Evolution of renal function one year after sleeve gastrectomy

The eGFR, as estimated by the CKD-EPI equation, significantly improved one year after sleeve gastrectomy relative to the value before surgery (87.8 ± 0.9 versus 86.1 ± 0.9, $p < 0.01$). The mean weight lost at the one-year follow-up was 27.1 ± 0.4% of total body weight. The evolution of comorbidities and metabolic parameters are presented in Table 3.

We then assessed the post-operatory evolution of the eGFR as a function of the baseline renal status. The eGFR significantly increased for patients with impaired renal function at baseline by one-year post-sleeve gastrectomy (78.8 ± 1 ml/min/1.73 m^2 versus 74.3 ± 0.9, $p < 0.001$), where it decreased for those with baseline renal hyperfiltration (114.1 ± 2.8 ml/min/1.73 m^2 versus 124.7 ± 1.2, $p < 0.01$) (Figure 1). Furthermore, the %TWL was lower for patients with a low eGFR at baseline than for those with a normal baseline eGFR (26.3 ± 0.6% versus 28.2 ± 0.6%, $p = 0.03$).

Evolution of the fat-free mass and estimated glomerular filtration rate one year after sleeve gastrectomy

FFM values two days and one-year post-sleeve gastrectomy, estimated from the SMA measured by CT, were available for 184 patients. FFM was significantly lower one-year post-surgery (43.6 ± 0.7 kg versus 50.3 ± 0.8 kg, $p < 0.0001$). However, there was no difference in terms of post-surgery FFM loss between patients for whom the eGFR improved and those for whom it did not (6.7 ± 0.3 kg versus 6.76 ± 0.5 kg, $p = 0.9$). The post-surgery changes in eGFR did not correlate with the amount of FFM loss ($r = 0.1$, $p = 0.18$). Furthermore, the absolute weight loss (32.6 ± 0.7 kg versus 32.4 ± 0.9 kg, p=0.9) and %TWL (27.4 ± 0.6% versus 26.8 ± 0.7%, $p = 0.48$) were not significantly different between patients with an improved eGFR and those for whom it did not improve one-year post-surgery.

Discussion

In this homogenous prospective cohort of 563 morbidly obese patients undergoing sleeve gastrectomy, renal function assessed by eGFR globally improved one-year post-surgery. When stratified by baseline renal status, eGFR was significantly higher by the one-year follow-up only for patients with impaired renal function (eGFR < 90 ml/min/1.73 m^2) before surgery.

Our results are in accordance with those of previously published studies showing that the glomerular filtration rate improves mainly for obese patients with impaired baseline renal function [2,6,10,11]. In a retrospective study of 149 obese patients undergoing gastric by-pass or sleeve gastrectomy, Holcomb et al. [6] showed a post-surgery increase of the eGFR for patients with a baseline eGFR < 90ml/min/1.73 m^2. A significant increase in eGFR was also found during the first year following bariatric surgery by Neff et al. [10] in a prospective cohort of 461 patients with morbid obesity. In their study, changes in eGFR correlated with TWL and EWL. The authors also showed progressive improvement of the eGFR over a five-year follow-up in a subgroup of 19 patients with impaired renal function at baseline.

In our study, the eGFR was lower at one-year post-surgery for patients with baseline glomerular hyperfiltration (eGFR > 120 ml/min/1.73 m^2). Our results confirm those of the study of Clerte et al. [2] who showed a post-bariatric surgery decrease in the measured GFR, using the plasma iohexol clearance test, for a small group of morbidly obese patients with baseline renal hyperfiltration [2]. Previous biopsy studies showed that nephron hypertrophy and an increased number of glomerular capillaries are early events in obesity-related renal injury [12,13]. Rebelos et al. [14] recently reported that morbidly obese patients without chronic kidney disease show higher renal blood flow and cortical perfusion than lean controls, which reverses six months after bariatric surgery. Obesity-associated systemic hypertension increases glomerular blood flow through afferent arteriole dilation, thus partially explaining glomerular hyperfiltration in obese patients [15]. This phenomenon promotes arteriosclerotic changes and further progressive renal damage. Glomerular hyperfiltration can therefore be considered as an early signal of renal injury in obese patients. Thus, the decrease of the eGFR observed following sleeve gastrectomy in our subgroup of obese patients with baseline glomerular hyperfiltration translates into an improvement in renal function and probably slower degradation of the renal reserve. In accordance with this hypothesis, a recent study showed that obesity is associated with degradation of the eGFR during a three-year follow-up of kidney transplant recipients [16].

Accurate evaluation of renal function by measuring eGFR is problematic in obese patients. Radiolabel- and non-radiolabel-based methods for measuring GFR are the gold standard but are complicated to perform in routine practice and are expensive [17]. Plasma creatinine alone is a poor marker of renal function impairment, as it depends on the rate of creatinine production by the muscles. Therefore, methods to estimate the GFR using plasma creatinine and anthropometric data, such as the Cockcroft and Gault formula, have been developed. However, morbidly obese patients have a disproportional amount of fat mass relative to lean mass [18] and these methods overestimate the GFR. The use of

equations that do not rely on body weight may theoretically overcome this issue. Friedman et al. [19] showed that the CKD-EPI creatinine-cystatin C equation accurately estimates the GFR of morbidly obese patients. However, the CKD-EPI creatinine equation also performed adequately in this patient group and we therefore used it in our study in the absence of routine cystatin C level assessment. Aside from the loss of fat mass, sleeve gastrectomy also induces a decrease in skeletal muscle mass, as previously shown using CT cross-sections to estimate FFM [20]. Muscle mass loss influences plasma creatinine levels and may therefore falsely increase the eGFR in the absence of a true modification. In our study, patients whose eGFR increased one-year post-sleeve gastrectomy showed similar absolute weight loss and %TWL as those whose eGFR did not. Furthermore, post-surgery changes in the eGFR did not correlate with the amount of skeletal muscle loss as evaluated on CT-scan images. Overall, these data suggest that the post-bariatric surgery changes of eGFR described in our cohort of morbidly obese patients is not related to FFM loss and may reflect a true improvement in renal function.

Our study had a number of strengths, including its prospective design and the high number of included patients undergoing sleeve gastrectomy as the only bariatric surgery procedure. Another strength was the availability of FFM evaluation before and after surgery, which allowed us to assess the relationship between FFM loss and evolution of the eGFR.

Our study also had several limitations. First, we used the CKD-EPI equation to estimate the GFR rather than cystatin C, as it was not routinely performed at our institution. However, the use of the CKD-EPI creatinine equation has already been validated in morbidly obese patients and post-surgery changes of skeletal muscle mass did not appear to influence the evolution of the eGFR in our cohort. Second, a measured GFR was not available in our cohort. Nevertheless, the available methods to measure the GFR are too laborious to be used in clinical practice. Third, we only had follow-up data at one-year post-surgery. However, the rate of loss to follow-up was only around 20%, similar to previous results from tertiary centers. Furthermore, improvement of the eGFR mainly occurs during the first year after bariatric surgery. It should also be mentioned that measurement of albuminuria is not systematically performed in patients requiring bariatric surgery and therefore was not available in our study.

In conclusion, our results show that renal function of morbidly obese patients is significantly improved at one-year post-sleeve gastrectomy. This improvement was defined by an increase in the eGFR in the sub-group with a low baseline GFR and a decrease in the eGFR of those with baseline renal hyperfiltration. The post-sleeve evolution of the eGFR was independent of changes in skeletal muscle mass.

Declarations

Author contribution statement

CSV, SN and GP performed study conceptualization and design. DS, AL, SS, SN and CSV performed data collection. CV and SN performed data analysis. All authors were involved in the interpretation of the data.

DS and CSV edited the manuscript. PL, HT, KL, SP, MN, MG, MZ, AB and ID provided revision. All authors approved the final version to be published.

Conflict of Interest

The authors declare that they have no conflict of interest.

Compliance with Ethical Standards

The study was conducted in accordance with the national law concerning medical investigations and the 1964 Helsinki declaration and its later amendments or comparable ethical standards. The study protocol and the consent procedure were approved by the local ethics committee. Informed consent was obtained from all individual participants included in the study.

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Tables

Table 1. Characteristics of the study population.

	Included (N = 563)
Age (years)	41.2 ± 0.52
Male	124 (22.02%)
BMI (kg/m^2)	43.5 ±0.3
Hypertension	170 (3 0.5%)
Type 2 diabetes	115 (20.4%)
Dyslipidemia	171 (30.7%)
Urea (mmol/L)	5.1 ± 0.1
Creatinine (µmol/L)	83.1 ± 2.5
Uric acid (mmol/L)	350.1 ± 3.5
Fasting blood glucose (mmol/L)	6 ± 0.1
HbA1c (%)	5.9 ± 0.1
Insulin (mmol/L)	25.3 ± 0.7
AST (IU/L)	30.7 ± 0.9
ALT (IU/L)	43.4 ± 1.5
GGT (IU/L)	46.4 ± 2.3
Albumin (g/L)	40.7 ± 0.1
Cholesterol (mmol/L)	5.32 ± 0.07
Serum triglycerides (g/L)	1.47 ± 0.04
HDL (mmol/L)	1.29 ± 0.02
LDL (mmol/L)	3.38 ± 0.06
CRP (mg/L)	12.4 ± 0.4
Ferritin (ng/L)	145.5 ± 6.20
TE (kPa)	7.7 ± 0.3
CAP (dB/m)	309.78 ± 4.43
Steatosis	376 (81%)
NASH	177 (38.3%)
Fibrosis	
0 - No fibrosis	84 (18.3%)

1 - peri-sinusoidal or periportal fibrosis	276 (60%)
2 - Peri-sinusoidal and periportal fibrosis	78 (20%)
3 - Septal fibrosis	17 (3.7%)
4 – Cirrhosis	5 (1.1%)

Results are shown as the mean ± standard error of the mean or n (%). (SEM, standard error of the mean) or n (%).

Abbreviations: BMI, body mass index; HbA1c, glycated hemoglobin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; GGT, γ-gamma glutamyl transferase; HDL, high density lipoprotein; LDL, low-density lipoprotein; CRP, C-reactive protein; TE, transient elastometry; CAP, controlled attenuation parameter; NASH, nonalcoholic steatohepatitis.

Table 2. Baseline characteristics of patients with versus without a follow-up evaluation at one-year post-bariatric surgery.

	One-year evaluation (N = 448)	Lost to follow-up (N = 115)	p
Age (years)	42.2 ± 0.6	37.3 ± 1.3	< 0.0001
Male	92 (20.5%)	32 (27.8%)	ns
BMI (kg/m^2)	43.3 ± 0.3	44.4 ± 0.7	ns
Hypertension	137 (30.9%)	33 (28.7%)	ns
Type 2 diabetes	91 (20.5%)	22 (19.1%	ns
Dyslipidemia	144 (32.6%)	27 (23.5%)	0.05
Creatinine (µmol/L)	82.8 ± 2.2	84.2 ± 8.5	ns
Uric acid (mmol/L)	349.4 ± 7.5	352.7 ± 7.5	ns
eGFR (ml/min/1.73 m^2)	86.1 ± 2.3	94.3 ± 2.3	< 0.0001
Fasting blood glucose (mmol/L)	6 ± 0.2	5.9 ± 0.2	ns
HbA1c (%)	5.9 ± 0.1	5.9 ± 0.1	ns
Insulin (mmol/L)	24.9 ± 0.4	26.7 ± 0.4	ns
AST (IU/L)	31.1 ± 1.3	29.1 ± 1.3	ns
ALT (IU/L)	43.2 ± 2.8	44.3 ± 2.8	ns
GGT (IU/L)	46.7 ± 3.6	44.9 ± 3.6	ns
Cholesterol (mmol/L)	5.36 ± 0.09	5.12 ± 0.09	ns
Serum triglycerides (g/L)	1.48 ± 0.07	1.43 ± 0.07	ns
TE (kPa)	7.6 ± 0.4	7.8 ± 0.5	ns
CAP (dB/m)	306.3 ± 0.5	319.1 ± 7.5	ns
Steatosis	297 (81.2%)	79 (80.6%)	ns
NASH	144 (39.6%)	33 (33.7%)	ns
Significant fibrosis (F≥2)	79 (21.7%)	21 (21.9%)	ns

Results are shown as the mean ± standard error of the mean or n (%). (SEM, standard error of the mean) or n (%).

Abbreviations: BMI, body mass index; eGFR, estimated glomerular filtration rate; HbA1c, glycated hemoglobin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; GGT, gamma-glutamyl

transferase; TE, transient elastometry; CAP, controlled attenuation parameter; NASH, nonalcoholic steatohepatitis.

Table 3. Evolution of parameters at one-year post sleeve gastrectomy.

	Pre sleeve (N = 448)	Post sleeve (N = 448)	p
eGFR (ml/min/1.73 m^2)	86.1 ± 0.9	87.8 ± 0.9	< 0.01
BMI (kg/m^2)	43.3 ± 0.3	31.5 ± 0.3	< 0.0001
Hypertension	(30.9%)	(11.5%)	< 0,0001
Type 2 diabetes	(20.5%)	(10.6%)	< 0.0001
Dyslipidemia	(32. 6%)	(9.8%)	< 0.0001
Creatinine (µmol/L)	82.8 ± 2.2	81.2 ± 2.5	0.012
Uric acid (mmol/L)	349.8 ± 4	291.2 ± 3.8	< 0.0001
Fasting blood glucose (mmol/L)	6 ± 0.2	4.8 ± 0.1	< 0.0001
HbA1c (%)	5.9± 0.1	5.4 ± 0.1	< 0.0001
Insulin (mmol/L)	25 ± 0.8	8.9 ± 0.3	< 0.0001
Cholesterol (mmol/L)	5.36± 0.08	5.6 ± 0.17	ns
Serum triglycerides (g/L)	1.48 ± 0.05	0.99 ± 0.03	< 0.0001
CAP (dB/m)	307.3 ± 5.4	225.3 ± 5.2	< 0.0001

Results are shown as the mean ± standard error of the mean or n (%). (SEM, standard error of the mean) or n (%).

Abbreviations: eGFR, estimated glomerular filtration rate; BMI, body mass index; HbA1c, glycated hemoglobin; CAP, controlled attenuation parameter.

Figures

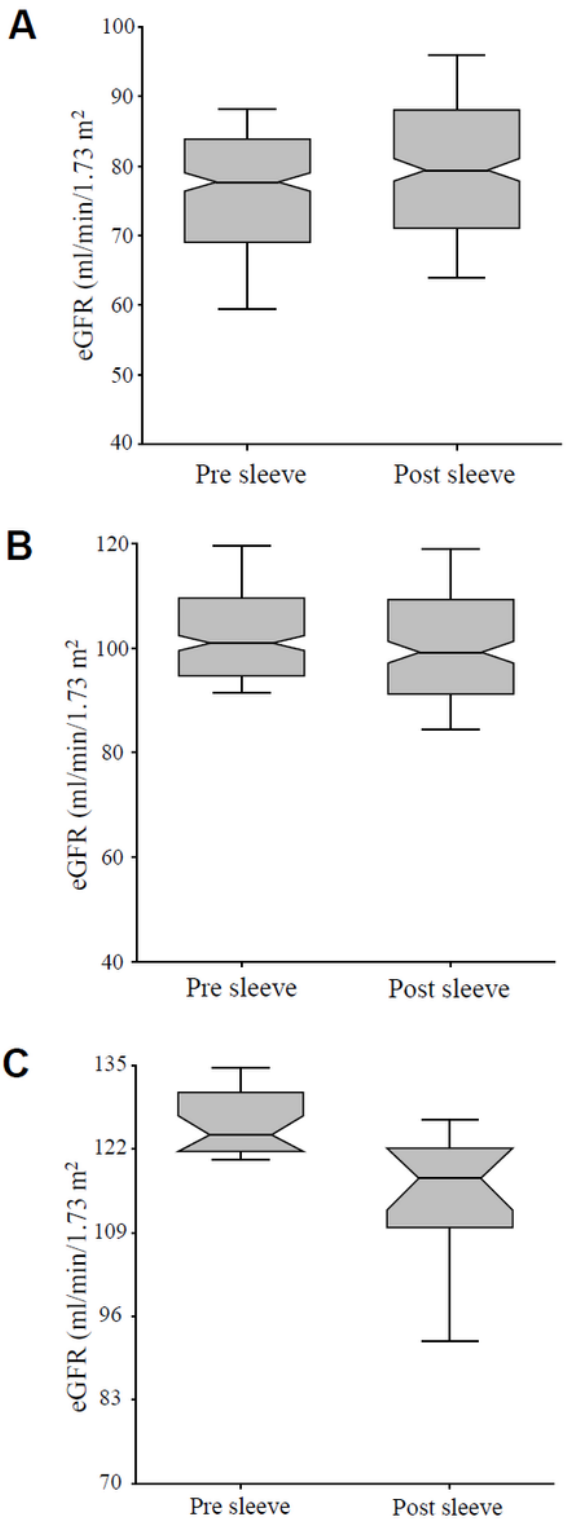

Figure 1

Estimated glomerular filtration rate (eGFR) before and after sleeve gastrectomy in the groups of patients with impaired baseline renal function (A), normal renal function (B), and glomerular hyperfiltration (C). Notched box plots showing the evolution of the estimated glomerular filtration rate (eGFR) at one-year post-sleeve gastrectomy as a function of baseline renal status. The line in the box indicates the median; the height of each box is the median ± 1.57 interquartile range/√n, used to assess the 95% confidence

interval around group medians. Differences are considered to be significant if the shaded boxes do not overlap ($p < 0.05$). The horizontal lines above and below each box indicate the interquartile range (from the 25th to 75th percentile) and the vertical lines at the ends of the boxes encompass the adjacent values: upper: 75th percentile + 1.5 times the interquartile range; lower: 25th percentile - 1.5 times the interquartile range.