Original article

Diverted Mini Gastric Bypass Surgery in Obese Population: Out-turn Over Hepatic and Nephrological Parameters

Bariatric Surgery Series: Paper II

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Abstract

Introduction: Bariatric surgery is the best possible option for reducing weight when lifestyle changes and medication have not brought about lasting success and may reduce morbidity and mortality. Weight loss that may follow bariatric surgery in patients on calory restricted diet would result in reduced inflammation and therefore lowering inflammation-related organ damage, including that of the kidney and liver. The study aimed to observe the consequences of BMI change on Hepatic and Nephrological parameters in patients after One Anastomosis Gastric Bypass surgery. Method: This study was done at a bariatric center with 150 individuals (both male and female) aged 20 to 60 years with obesity grades II and III who were selected randomly. Hepatic and renal function tests were carried out at baseline visit, then 3 months and 6 months following surgically. Result: Bilirubin level significantly increased from baseline to visit 1; Serum glutamic-pyruvic transaminase (SGPT) levels significantly decreased at visits 1 and 2. The albumin to Globulin ratio was significantly increased at visit 2 . Blood Urea level and serum creatinine level reduced considerably at visit 1 and decreased more at visit 2 from baseline. Conclusion: Bariatric surgery may be related to improvement in both hepatic and renal function. The improvement may be attributed to reduced inflammatory organ damage related to obesity. More such studies must be performed to highlight the possible health benefits of bariatric surgery for obese patients whose lifestyle modification and medication have not aided in weight loss.

Keywords: Bariatric surgery, Obesity, inflammation, organ damage, weight loss, health benefit, renal function, liver function

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1. Introduction

Obesity is on the rise around the globe. It poses an imminent danger for concurrent medical conditions such as type 2 diabetes mellitus (T2DM), high blood pressure, coronary artery disease, stroke, lung disease, and other malignancies [1]. The only method of treating morbid obesity that reliably works is surgery, where considerable weight loss is attained and maintained, and the comorbidities and standard lifestyle factors associated with obesity

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are improved [2,3]. Continued T2DM treatment and sustainable weight reduction have been achieved with outstanding feasibility by bariatric surgery. For the treatment of obese patients with T2DM, various laparoscopic techniques for bariatric surgery have been reviewed [4]. The Roux-en-Y gastric bypass (RYGBP) and the biliopancreatic diversion with or without the duodenal switch (BPD/BPD-DS) have shown promising outcomes regarding weight reduction and controlling blood glucose levels [5,6].

A novel technique called the mini gastric bypass or one anastomosis gastric bypass (MGB/OAGB), invented by Rutledge in 1997, is a modified form of the traditional RYGBP [7]. Regardless of skepticism, various researchers have documented admirable reduction in weight and correction of obesity-related multiple medical conditions, including T2DM and fertility issues, as well as other benefits [8]. Laparoscopic mini gastric bypass (MGB) is a comparatively recent and preferred surgery at various hospitals because of short operating times, relatively more straightforward procedures, excellent weight reduction, and fewer post-operative complaints [9] (Figure 1). Nonetheless, weight reduction occasionally comes at the expense of malnourishment and the difficulties that accompany it [10]. Furthermore, its impact on liver function, particularly potential effects on non-alcoholic fatty liver disease (NAFLD), has not yet been extensively examined [11]. Up to 90% of these individuals have NAFLD, which is very common and varies from moderate fatty liver alterations and steatosis to non-alcoholic steatohepatitis (NASH), with the potential to proceed to liver fibrosis with persistent disease [12].

Obesity is a distinct susceptibility indicator for developing chronic kidney disease (CKD), which causes altered hormone levels and growth factors resulting in hyperfiltration, focal segmental glomerulosclerosis, inflammatory processes, and change in renal hemodynamics [13]. Additionally, studies have shown that bariatric surgery is associated with advantageous impacts on CKD by reducing its risk factors through reducing body weight and lowering insulin resistance, proteinuria, and hemoglobin A_1C (HbA_1C) [14]. Management with bariatric surgery is the most effective treatment option for morbid obesity [15]. In individuals with obesity and kidney dysfunction, bariatric surgery has been linked to a 58% decline in the risk of proteinuria and albuminuria and a 54% decrease in the risk of hyperfiltration [16]. Moreover, because of the longer biliopancreatic limb (BPL),

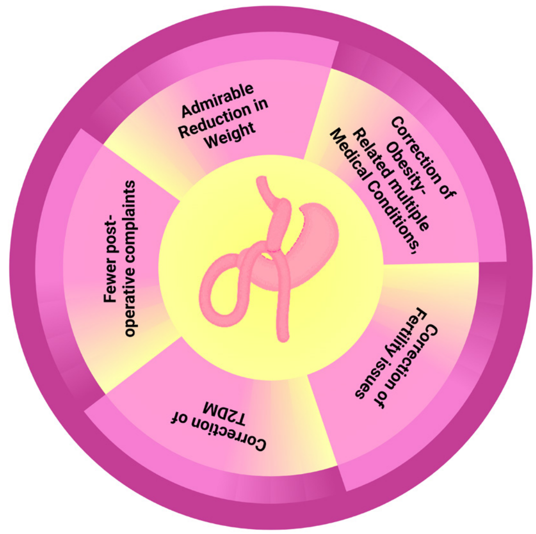

Figure 1: Schematic diagram showing beneficial effects of mini gastric bypass (MGB). This figure has been drawn with the premium version of BioRender [https://www.biorender.com/ Accessed on 27 July 2023] with license number OL25NPDHYE. Image credit: Susmita Sinha.

diverted mini gastric bypass surgery patients might encounter a more acute progression of losing weight than RYGBP patients, which may seldom cause altered liver function, nutritional deficiency and renal problems [17]. Therefore, Mini Gastric Bypass Surgery in Obese Population efficiently enhances kidney function parameters regardless of weight loss and reduces related medical issues [18].

1.a. Effects of Obesity on Renal Pathophysiology

One of the significant risk factors of kidney disease globally has been noted to be obesity. This risk factor contributes to about 20-25% of chronic kidney disease (CKD) worldwide [19]. Between 1999-2009 the percentage of the obese population receiving renal transplants for end-stage kidney disease (ESKD) increased by around 44% [20]. After adjusting for hypertension and diabetes (major obesity-related CKD causes), obesity was observed to be an independent risk factor contributing to renal pathophysiology [21,22]. Non-diabetic obese individuals were noted to be more prone to develop progression of CKD when compared to non-obese patients [23]. There are various immunological and endocrine dysregulations in adipocytes that may lead to renal tissue damage [24].

Impairment of differentiation of adipocyte progenitors to form insulin-sensitive functional adipocytes occurs in obesity, which eventually may result in lipotoxicity with lipid deposition on organs like skeletal muscle, kidney, and liver. This leads to insulin signaling impairment and resistance to insulin in these organs [25]. Adipogenesis impairment also may cause hypertrophy of adipocytes which in turn promotes the production of inflammatory cytokines like interleukin 6 (IL 6) and tumor necrosis factor-α (TNF-α), promoting insulin resistance. The cytokines and signaling molecules from the adipocytes can mediate immunological and inflammatory phenotypes of organs like the kidneys and cause deterioration of renal functions. It was noted that in obese subjects, independent of diabetes mellitus, there were raised levels of IL 6 and TNF α in plasma, which were associated with the incidence and severity of CKD [26,27]. Loss of weight or bariatric surgery may reduce hyperfiltration of the glomerulus by lowering inflammatory cytokines [28].

Hypertrophy of adipocytes may lead to its outgrowth from its blood supply, causing hypoxia, inflammation, and death of cells [29]. There is the activation of hypoxia-inducible factor 1α (HIF-1α), which induces pro-inflammatory transcription promoting inflammation [30,31]. Hypoxia also decreases insulin sensitivity by reducing insulin receptors via destabilization of mRNA, which encodes for insulin receptors [32].

The relationship between obesity and CKD is bidirectional. CKD promotes lipotoxicity by decreasing the subcutaneous fat volume and redistributing the fat to viscera—ectopic fat deposition on organs like the kidney causing further inflammation [33]. Exposure of adipose tissue to serum containing urea has been reported to enhance activation of HIF-1α and NFκB that promote inflammation in adipocytes. In patients undergoing dialysis, adipose tissue exhibited more significant levels of markers of inflammation [34]. CKD also increases macrophage infiltration into adipocytes, promoting inflammation, glucose intolerance, and insulin resistance [33,35,36]. Uremia also causes the alteration of adipokines in adipocytes. Adipocytes incubated with serum containing urea displayed a rise in leptin secretion [37-39]. In CKD patients, accumulation of urea also promoted oxidative stress in adipocytes leading to the formation of adipokines like retinol-binding protein-4 and resistin, which induce resistance to insulin [36]. Adipokine changes also affect sodium handling by the kidney, which may lead to hypertension which in turn causes kidney injury. High leptin levels in obesity also increase sympathetic activity and thus promote the renin-angiotensin-aldosterone mechanism, thus increasing sodium retention by the kidney [40-42].

The proximal convoluted tubule of the kidney remains insulin sensitive, and hyperinsulinemia in obese patients increases sodium reabsorption in obesity. However, podocytes become resistant to insulin, promoting podocyte dedifferentiation and reducing the selectivity of the basement membrane of glomerulus [43-45].

In morbidly obese individuals failing to reduce weight and are unresponsive to weight loss medications may opt for bariatric surgery. There is reduced inflammation, hyperfiltration of glomerulus, and proteinuria in CKD patients with obesity following bariatric surgery [33]. There is also a reduction of about 79% in the 5-year risk of mortality in pre-dialysis CKD subjects with obesity [46].

1. b. Effects of Obesity on Hepatic Pathophysiology

The development of simple steatosis (SS) and nonalcoholic steatohepatitis (NASH) appear to be influenced by obesity [47]. Hepatocytes are thought to have an adipocyte-like activity when adipose tissue cannot preserve extra calories, as in disorders like lipodystrophies or typical obesity [48]. In these circumstances, the hepatic cells primarily store the additional lipids as triglycerides, which results in basic steatosis. Abnormal fat accumulation (for example, in skeletal muscle and the liver) due to increased lipolysis and decreased fatty acid intake in subcutaneous fat deposits may cause insulin resistance (IR) in different organs [49]. Since adipose tissue lipolysis accounts for around 60% of the primary substrate for intra-hepatic triglycerides free fatty acids (FFAs), the remaining 25% comes from de novo lipogenesis within the hepatocyte from nutrients like carbohydrates. This causes a redistribution of fat from regular to abnormal storage [50].

When hepatocytes are exposed to high carbohydrate and lipid levels, glucotoxicity and lipotoxicity are initiated, respectively, which plays a vital role in the progression of SS and NASH [51]. The pathophysiological pathways that link SS and NASH to lipotoxicity and glucotoxicity include oxidative stress, endoplasmic reticulum stress, and mitochondrial abnormalities [52,53]. Furthermore, the release of FFAs from insulin-resistant and malfunctioning fat cells causes lipotoxicity, brought on by the abnormal collection of harmful substances derived from triglycerides and the resulting stimulation of the inflammatory paths, cellular dysfunction, and lipoapoptosis. Besides, lipoapoptosis is a crucial component of NASH and is caused by the inability of hepatocytes to eliminate excess FFAs [54,55].

An intra-hepatic inflammatory response begins if obesity is not effectively managed at the stage of SS, probably as an ineffective opposing attempt to stop SS [56]. The liver's innate immune cells, such as Kupffer cells, dendritic cells, and hepatic stellate cells (HSCs), are activated during this process, and immune cells, primarily macrophages, neutrophils, monocytes, and T-lymphocytes, gradually invade the liver. As a result, Immune cells in the liver release cytokines that exacerbate the inflammatory response and aid in the fibrotic process, which often occurs when the inflammation persists [57].

Adipokines (such as leptin and adiponectin), hormones generated from adipose tissue that may have a role in SS, NASH, cirrhosis, and carcinogenesis, are an additional manner that obesity affects the liver [58]. Adipokines are adequately regulated in healthy people of average weight. However, this equilibrium is thrown off in obese people [59]. The released adipokines change, heading towards an additional steatogenic, inflammatory, and fibrogenic character as the adipose tissue enlarges [60]. Adipokines interact with immune cells (macrophages, B-lymphocytes, T-lymphocytes, and neutrophils), which invade adipose tissue during its expansion and produce interleukins (ILs) and classical cytokines such as IL-1, IL-6, and tumor necrosis factor (TNF alpha) [61].

Additionally, hypoadiponectinemia is linked to a more excellent ratio of normal to abnormal fat storage. Adiponectin functions as an anti-steatosis, anti-inflammatory, and anti-fibrotic adipokine [62]. In this way, adiponectin reduces the production of pro-inflammatory cytokines like TNF alpha. It promotes the production of anti-inflammatory cytokines like IL-10, suppressing macrophage activity and reducing oxidative stress and fibrogenesis [63]. In humans, higher amounts of adiponectin were found in controls, lower levels in SS patients, and even lower levels in NASH patients [64].

2. Objectives of the Study

The study is taken up with the objective of the consequences of BMI change on Hepatic and Nephrological parameters in patients after OAGB surgery.

3. Materials and Methods

3a. Study Details

Study Type: Longitudinal observational study.

Study Period: This study was conducted from January 2021 to January 2022

Sampling Type: Universal sampling was done with the patient's consent, and patients were informed about the possible benefits, side effects, and risks associated with the surgical interventions.

Study Subject: The subjects of this research were recruited from Asian Bariatrics Hospital, SG Highways, Ahmedabad, Gujarat, India

Methods of Enrollment and Randomization: Patients were enrolled between February 2021 and July 2021, and a 6-month follow-up was completed by January 2022.

Project Details: This is the second paper of the principal author of the Bariatric Surgery project. The earlier Paper-I was published in the Bangladesh Journal of Medical Science (https://www.banglajol.info/index.php/BJMS/article/view/66965) [65].

3b. Sample Size Calculation Asian Bariatrics Plus Hospital is a wide-ranging and sizeable center of obesity and metabolic surgery in India, where approximately 20-25 patients are operated on monthly. Thus, to meet the required sample size of 120-150, subjects will be enrolled for 6 months and followed up for another 6 months post-bariatric surgery.

Sample Size Estimation with Single Group Mean: $N = (Z\alpha/2)2\ s2 / d2$

$Z\alpha/2$ = standard deviation for the two-tailed alternative hypothesis at a significance level.

S = the standard deviation obtained from the previous study or pilot.

D = the estimate's accuracy or how close to the true mean.

$Z\alpha/2=3.29$; $s=6$; $d=1.5$.

The calculated sample size would be 130. If the allowance of 10% for missing, losses to follow-up, and withdrawals are assumed, then the corrected sample will be 143 subjects. The corrected sample size thus obtained is 130/ (1.0-0.10) □ 130/0.9 = 145; for 20% allowances, the corrected sample size will be 156. So, the estimated sample size preferred for this study would be 130-156 (Reference: https://pubmed.ncbi.nlm.nih.gov/29346210/) [66].

Inclusion Criteria: The age range included was between 20–50 years, belonging to both genders, with a mean BMI of 45.63±6.54 (male) and 41.81± 5.93kg/m^2 (female). Exclusion criteria: Any previous weight loss surgery history, severe cardio-respiratory disease, cancer, oral steroid treatment, and psychiatric medications, as per the recommendations indicated for bariatric surgery.

3c. Anthropometric Evaluation

Weight, height, and BMI were used for anthropometric evaluation. The patients were weighed on a Bioelectrical Impedance Machine In Body 770, and BSM 170 in body measuring scale was used for Height measurements.

3d. Surgical Intervention

Laparoscopic technique was used for the surgery. A 5 mm Endopath instrument at Palmer's point was used to create the Pneumoperitoneum and the remaining 3 ports –11 mm supra-umbilical port, 12mm right of right Rectus muscle port, and 5 mm port on right hypochondrium was created. Gastroesophageal junction dissection was done by retracting the fundus and dividing the peritoneum overlying the GE junction using a Goldfinger instrument. Further, the greater omentum was divided vertically to the upper line of the transverse colon and later divided transversely. DJ flexure was identified, and a loop of small bowel was traced to 150 cm. The loop was then pulled up in ante colic fashion and anchored to the greater curvature opposite the incisura. Dissection for the gastric pouch was started by creating a window in the lesser curve near the incisura. Subsequent stapler firing made the required stomach pouch of around 100ml. 36 Fr Gastric Calibration Tube was used. Alimentary and biliopancreatic limb measurements were 150 cm.

3e. Nutritional Intervention

The hepatic function test (HFT) and Renal function test (RFT) were examined at the baseline visit, and the same tests were repeated at 3 months and 6 months after surgery. The dietary recommendation was a Very low-calorie diet (VLCD) with higher protein intake (1-1.2g per kg IBW). The Diet progressed in texture over 1 month, and later the diet was revised as per the weight loss observed over time. A balanced diet consisting of all food groups was included.

All patients in the postoperative period received commercially available mineral and vitamin supplements, per American Society of Metabolic & Bariatric Surgery (ASMBS) guidelines, 2016-18, after a week of surgery. At follow-up, the patients were asked about supplement compliance, dietary concerns, and complaints after surgery like gastric reflux, constipation, diarrhea, nausea, vomiting, etc.

3f. Biochemical Assays

Various biochemical tests were done to see the hepatic function (SGPT, SGOT, Total Protein, Albumin, Globulin, and A: G ratio) and renal function tests like creatinine, urea, etc.

3g. Statistical Method

We used descriptive statistics to summarize and describe our collected data in our analysis. We calculated the mean and standard deviation for continuous variables, such as biomarker measurements, to measure central tendency and dispersion, respectively. Categorical variables, on the other hand, were summarized using percentages to represent the proportion of each category within the sample.

To investigate the changes in biomarkers before and after follow-up, we employed a paired sample t-test. This statistical test allowed us to compare the means of the biomarkers within the same individuals, thereby assessing the mean difference between the pre-operative and post-operative measurements.

We considered time (pre and post-operative follow-up) as the primary predictor variable in our regression model to examine the effects of Anastomosis Gastric Bypass Surgery on the biomarkers. We were interested in understanding how the biomarkers changed over time due to the surgical procedure. To analyze the impact of time, we used a mixed-effects model. This model allowed us to account for having repeated measurements within the same individuals (pre and post-operative measurements). This model treated time as a fixed effect, representing its overall effect on the biomarkers across all individuals.

Additionally, we included the within-subject difference as a random effect, capturing the individual-specific variation in the biomarker changes. Our regression model also incorporated other covariates, including age, sex, BMI, and comorbidities. However, upon analyzing the data, we found that comorbidities did not significantly affect the biomarker changes. As a result, we decided to remove comorbidities from the final regression model, focusing solely on age, sex, and BMI as potential predictors of the biomarker changes.

To determine statistical significance, we considered p-values less than 0.05. We utilized STATA 15 (StataCorp, LP, College Station, Texas, USA) for our statistical analyses. Additionally, we used GraphPad Prism 8.3.0.538 to generate figures that visually represented the results of our analyses (Figure 2).

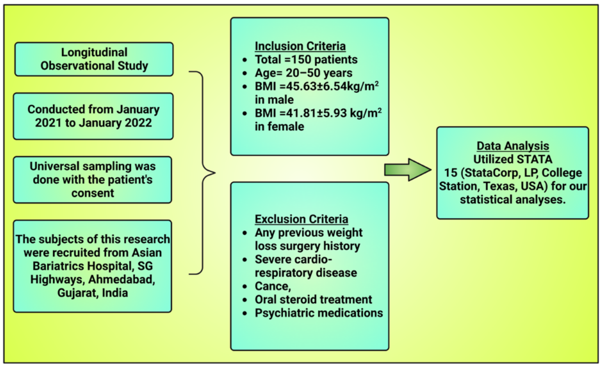

Figure 2: Flow chart showing the materials and methods. This figure has been drawn with the premium version of BioRender [https://www.biorender.com/ Accessed on 27 July 2023] with license number IZ25NPYR7N. Image credit: Susmita Sinha.

3h. Ethical Approval

This study was approved by the Institutional Review Board of Asian Bariatrics Hospital, SG Highways, Ahmedabad, Gujarat, India, with Reference number IECHR-AB/2021/11 dated 19/05/2021. All the study subjects verbally explained the study's intention, motive, and future scientific publication. The written informed consent was obtained before data collection commenced.

4. Results

This observational study involved the inclusion of 150 patients who were scheduled to undergo Anastomosis Gastric Bypass Surgery. The mean age of the patients was 41.7 years, with a standard deviation of 14.7 years (mean±SD: 41.7±14.7). Among the study participants, 56 individuals (37.3%) were male, while 94 (62.7%) were female. This distribution highlights a higher representation of female patients in the study cohort.

Regarding comorbidities, hypertension was identified as the most prevalent condition among the enrolled patients, affecting 49.3% of them. This was followed by obstructive sleep apnea syndrome (OSAS) at 32.7% and a history of diabetes at 29.3%. Additional comorbidities, such as dyslipidemia and hypothyroidism, were present in 20.7% and 16.7% of the patients, respectively (Table 1).

Table 1: Demographic characteristics of the study participants

Factors	Observation
Age	41.7±14.7
Sex	
Male	56(37.3%)
Female	94(62.7%)
H/O Diabetes	44(29.3%)
H/O Hypertension	74(49.3%)
H/O OSAS	49(32.7%)
H/O Dyslipidemia	31(20.7%)
H/O Hypothyroidism	25(16.7%)

Notes: Data were presented as mean±SD or number with percent in the parenthesis. History of= H/O.

Table 2: Difference in outcome biomarkers level before and after one Anastomosis Gastric Bypass Surgery.

	Baseline	Visit-1	p-value	Visit-2	p-value
Bilirubin (mg/dl)	0.55±0.22	0.68±0.26	<0.001	0.58±0.29	0.373
ALP [IU/L]	102.7±47.6	104.2±39.0	0.758	103.0±53.3	0.969
SGPT (IU/L)	25.8±11.0	18.9±7.51	<0.001	16.6±7.62	<0.001
Total Protein (gm/dl)	6.97±1.01	6.87±0.49	0.299	6.87±0.71	0.326
Ratio A/G	1.18±0.63	1.29±0.28	0.090	1.51±0.67	<0.001
Serum Urea (mg/dl)	26.2±7.66	23.2±6.81	<0.001	22.9±6.77	<0.001
Serum Creatinine (mg/dl)	0.92±0.26	0.86±0.23	<0.001	0.78±0.21	<0.001

Notes: Data was presented as mean±SD. Paired sample t-test was used to estimate the p-value, and the comparison was between the baseline with visit-1 and visit-2. Milligrams per deciliter = mg/dL. Alkaline phosphatase = ALP. Serum glutamic pyruvic transaminase = SGPT. Grams per deciliter = gm/dL. Albumin/Globulin = A/G.

The analysis revealed several significant findings. Bilirubin levels increased significantly from baseline (0.55±0.22 mg/dl) to Visit-1 (0.68±0.26 mg/dl, p<0.001). However, at Visit-2, there was no significant change in the levels (0.58±0.29 mg/dl). Serum glutamic-pyruvic transaminase (SGPT) levels decreased significantly from baseline (25.8±11.0 IU/l) to Visit-1 (18.9±7.51 IU/l, p<0.001) and further reduced at Visit-2 (16.6±7.62 IU/l, p<0.001). The A/G ratio did not significantly change from baseline (1.18±0.63) to Visit-1 (1.29±0.28, p=0.090). However, there was a significant increase in the ratio

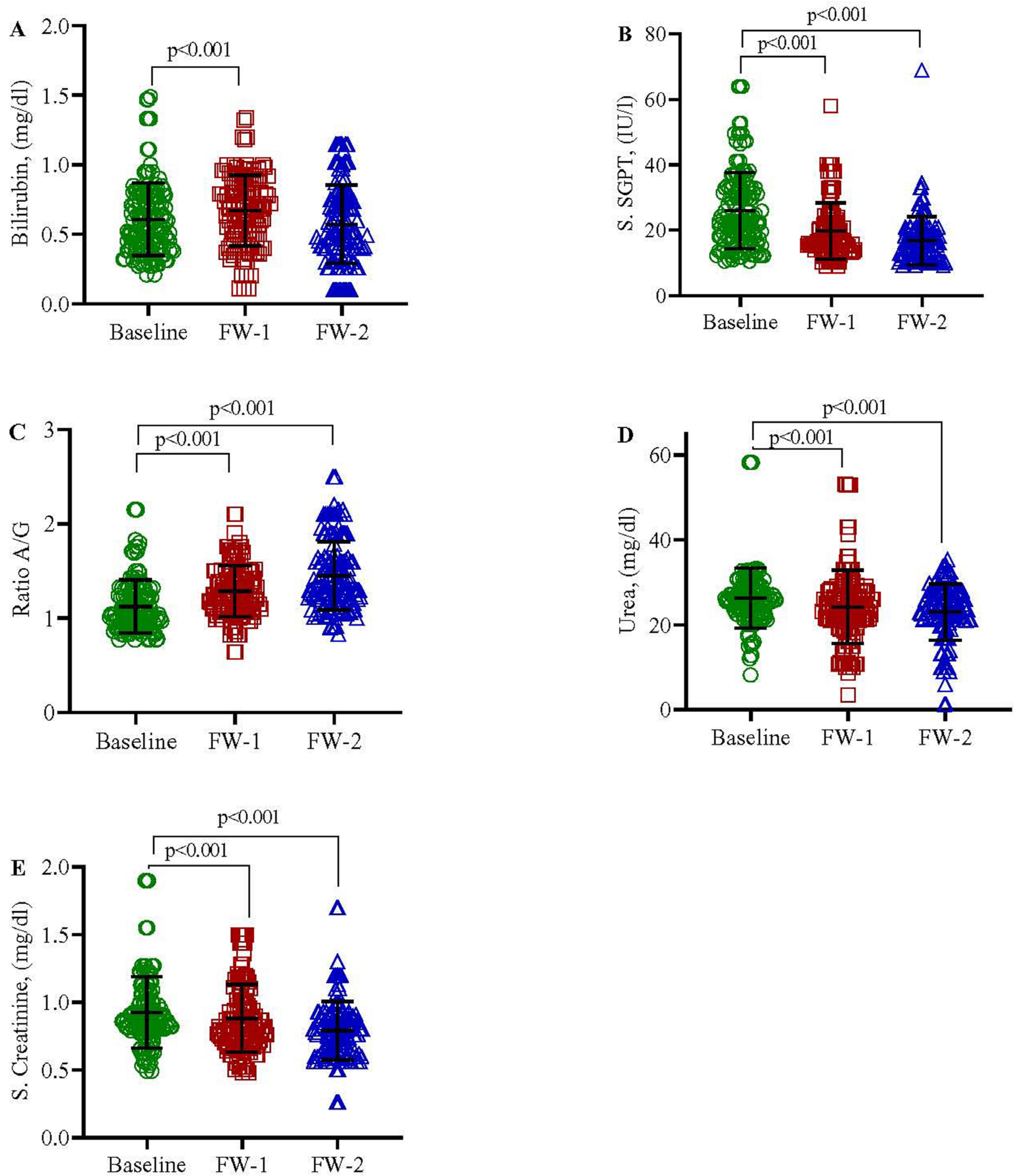

Figure 3: Difference in studied biomarkers after one Anastomosis Gastric Bypass Surgery. Paired sample t-test was applied to see the difference between baseline with follow-ups 1 & 2.

at Visit-2 (1.51±0.67, p<0.001). Urea levels decreased significantly from baseline (26.2±7.66 mg/dl) to Visit-1 (23.2±6.81 mg/dl, p<0.001) and were further reduced at Visit-2. The mean serum creatinine levels decreased significantly from baseline (0.92±0.26 mg/dl) to Visit-1 (0.86±0.23 mg/dl, p<0.001) and were further reduced at Visit-2 (0.78±0.21 mg/dl, p<0.001) (Table 2 and Figure 3).

After the surgery, there is a significant decrease in S. SGPT levels were noted at both follow-ups compared to pre-operation by 7.74 IU/L (95% CI=-10.2, -5.30;

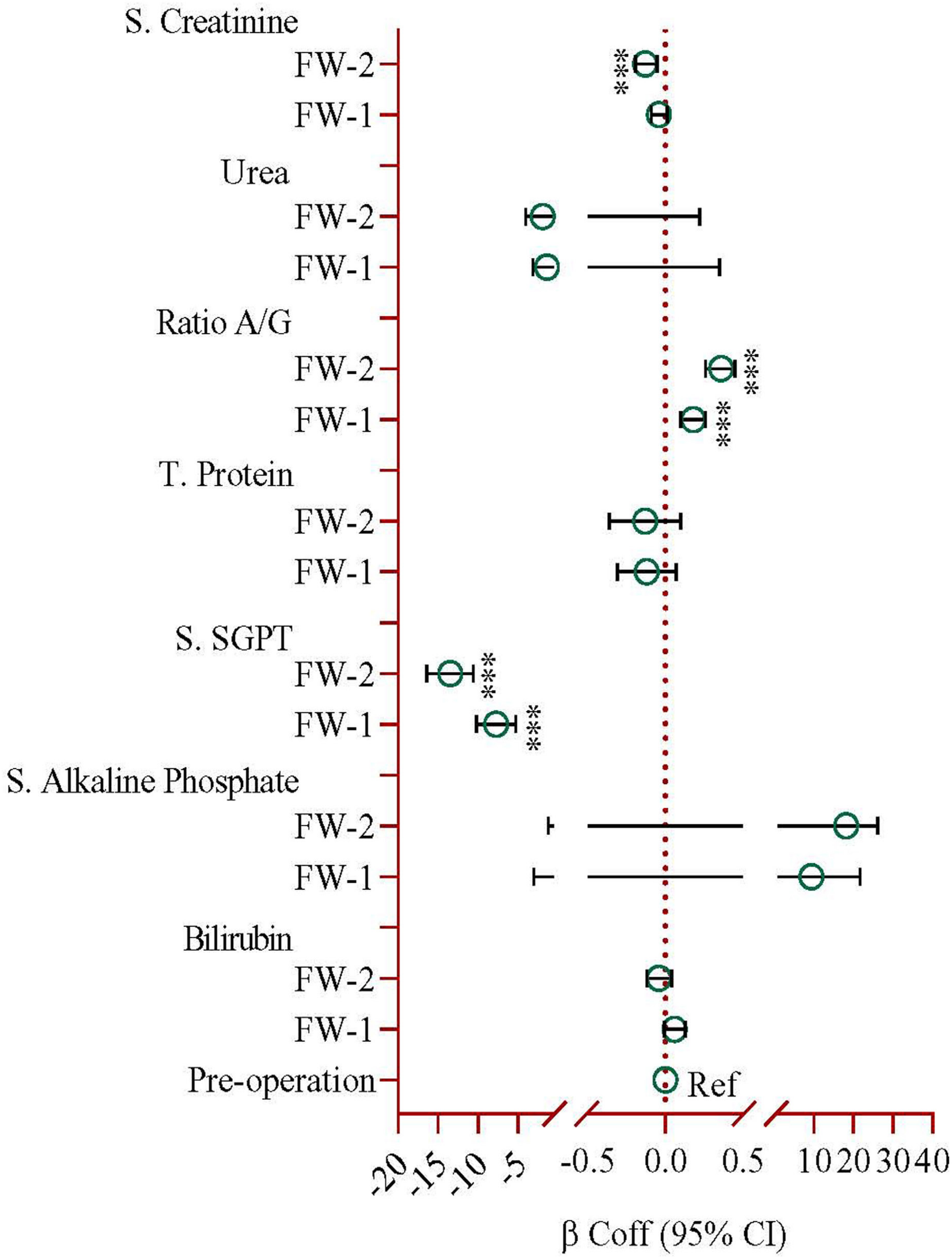

Figure 4: The effect of one Anastomosis Gastric Bypass Surgery on biomarkers between follow-up and pre-operation. The estimates and significant differences were calculated using a subject-specific mixed effects model controlling for time, age, BMI, and Sex. Note, *** indicates $p<0.001$ and * $p<0.05$.

$p<0.001$ and by 13.5 IU/L (95% CI=-16.4, -10.6; $p<0.001$) respectively at follow-up 1 & 2 (Figure 2). Whereas there is a significant increase in the Ratio A/G by 0.18 units (95% CI=0.10, 0.26; $p<0.001$) and 0.36 units (95% CI=0.26, 0.45; $p<0.001$), respectively, at follow-up 1 and follow-up 2 compared to pre-operation. There is also a significant decrease in S. Creatinine levels observed at follow-up 2 (β=-0.04, 95% CI=-0.09, 0.01; $p<0.001$ (Figure 4).

5. Discussion

Bilirubin levels increased significantly from baseline (0.55±0.22 mg/dl) to Visit-1 (0.68±0.26 mg/dl, $p<0.001$). However, at Visit-2, there was no significant change in the levels (0.58±0.29 mg/dl). A study reported similar findings where the bilirubin levels increased after one anastomosis gastric bypass surgery [67]. There is also a significant decrease in S. SGPT levels after the surgery. According to an analysis of 25 OAGB patients, SGPT levels were significantly more critical in the OAGB group at 1-year follow-up. In contrast, this study found SGPT levels to be considerably lower after OAGB [68,69]. Besides, the primary tool for diagnosing and monitoring the development of liver disease is the level of hepatocyte destruction as measured by liver enzymes (Figure 5).

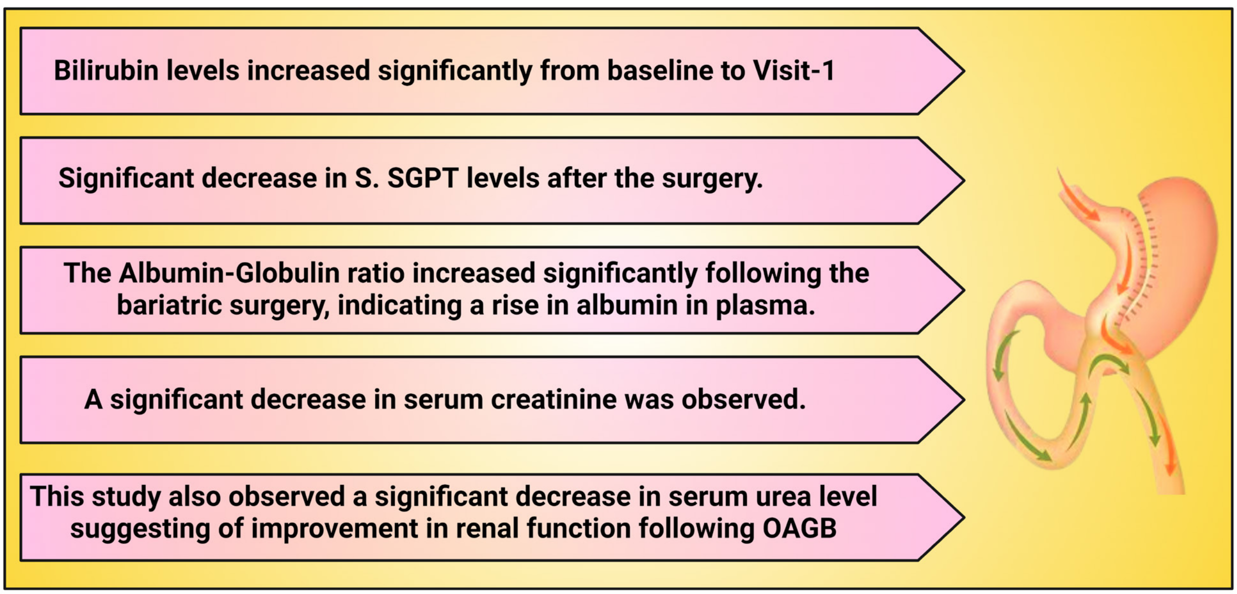

Figure 5: Chart showing study findings. This figure has been drawn with the premium version of BioRender [https://www.biorender.com/ Accessed on 27 July 2023] with license number VY25NQ1C5L. Image credit: Susmita Sinha.

The Albumin-Globulin ratio increased significantly following the bariatric surgery, indicating a rise in albumin in plasma. A study done to observe if Globulin -Albumin ratio is a good predictor for gastric carcinoma following curative resection noted that a low Globulin – Albumin ratio pointed towards a good outcome [70]. They suggested that a raised globulin level in serum reflects tumor progression and antitumor immunity suppression. At the same time, a rise in albumin causes suppression of cancer cell growth by DNA replication stability and antioxidant release [71]. Malnutrition, such as obesity (a state of chronic inflammation), causes albumin production suppression [72,73]. Since bariatric surgery promotes weight loss and improved BMI and reduces inflammation, there may be an improvement in the Albumin-Globulin ratio [70,74,75]. Also, following bariatric surgery, toll-like receptors (TLR-2 and TLR-4) expression, nuclear factor kappa β (inflammatory transcription factor), matrix metallopeptidase, and C-reactive protein decrease leading to lowering of systemic inflammation [76,77].

In this study, a significant decrease in serum creatinine was observed. Similar findings were noted by several studies performed previously [78-84]. The decline in serum creatinine level indicates an improvement in renal function following bariatric surgery. Obesity-related inflammation leads to hyperfiltration due to kidney structure damage and eGFR increase. Bariatric surgery stabilizes eGFR and improves ultrafiltration as well as creatinine clearance [85,86]. Serum creatinine level is a crude indication of eGFR and depends on muscle mass. Therefore, a reduction in serum creatinine level following surgery may be due to trauma from surgery, leading to an overestimation of GFR. Formula using both serum creatinine and cystatin C may give a more accurate renal function estimation [87]. Another study by Schuster *et al.*, [88] found an increase in serum creatinine levels which may be due to a severe form of kidney disease that cannot be reversed by surgical intervention.

This study also observed a significant decrease in serum urea level which again suggests an improvement in renal function following bariatric surgery. A study done by Seki *et al.*, found that higher Blood urea nitrogen level was associated with poor renal outcome [89]. Following bariatric surgery, if calory restricted diet is followed, there is weight loss, reducing inflammation [90,91]. Reducing inflammation helps lower kidney structural damage and improves renal tubular filtration [92]. Since urea is free-filtered, not secreted but reabsorbed by the renal tubule, a low urine flow rate would lead to more urea reabsorption, thus increasing blood urea level [93]. An improvement in renal filtration would therefore lower the blood urea level [94]. Renal function improvement may also be attributed to insulin sensitivity and blood pressure improvement in patients following bariatric surgery, which decreases tissue injury in the kidney [95]. An improvement in blood pressure may be due to the lowering of leptin levels which in turn lower sympathetic activity within the body, which may also lower renal structural damage [96]. Insulin sensitivity may increase after bariatric surgery due to a decrease in κB kinase β inhibitor activity and improvement in insulin signaling in muscle along with weight loss [97].

Therefore, the improvement in the parameters, including Albumin-Globulin ratio, serum creatinine, and blood urea, may be due to weight loss, increase in insulin sensitivity, improvement in blood pressure, and reduction in inflammation following surgery (Figure 6).

6. Conclusion

One Anastomosis Gastric Bypass surgery appears to positively impact hepatic function as evidenced by improvement in ALP, SGPT, Serum Bilirubin, and Albumin/Globulin ratio following surgery.

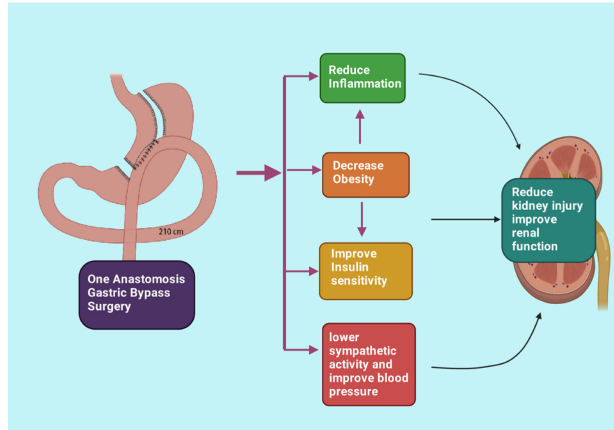

Figure 6: Demonstrates the changes that may follow One Anastomosis Gastric Bypass Surgery, like a decrease in obesity, Inflammation, increased insulin sensitivity, and a reduction in sympathetic activity with the improvement of blood pressure in patients leading to reduced damage to organs like a kidney with the recovery of its function. This figure has been drawn with the premium version of BioRender [https://biorender.com/ Accessed on 24 July 2023] with license number II25NADJEN. Image credit: Rahnuma Ahmad.

This seems to be also the case for renal function since Serum creatinine and blood urea levels decreased in patients after bariatric surgery. Such positive changes may be linked to weight loss and decreased obesity-related inflammation. Obesity is a chronic state of inflammation that leads to various inflammatory cytokine production. These cytokines, like TNFα, Interleukins may lead to tissue damage. Bariatric surgery followed by patients consuming a calory restricted diet may reduce BMI and lower inflammation in the body. Reducing inflammation may lead to decreased organ damage and improved organ functioning. For example, a decrease in serum creatinine level may reflect an improvement in hyperfiltration and lowering of GFR towards normal in these patients. Long-term follow-up of these patients may further reveal the eventual impact of this life-altering surgery on renal and hepatic physiology. More studies in the future to assess the inflammatory cytokines and other mediators of inflammation changes following bariatric surgery and its impact on various organs may help stress the significance of bariatric surgery, particularly in patients who suffer from morbid obesity showing no improvement with lifestyle changes and medication.

7. Recommendation

The International Federation for Surgery of Obesity

(IFSO) recommends the Mini Gastric Bypass as an excellent and efficient bariatric and metabolic procedure. However, it has specific problems, such as hypoalbuminemia, which is more commonly reported in vegetarians and people with alcoholic and nonalcoholic liver disease. In addition to being documented following biliopancreatic diversion, steatohepatitis, and hepatic failure are well-known side effects of jejunoileal bypass, a common type of bariatric surgery. More severe consequences could be avoided with early detection and effective therapy. Future research on this subject should consider hepatic elastography and biopsy as it would help to understand further how OAGB affects the structure and function of the liver.

Consent for Publication

The author reviewed and approved the final version and has agreed to be accountable for all aspects of the work, including any accuracy or integrity issues.

Disclosure

The author declares that they do not have any financial involvement or affiliations with any organization, association, or entity directly or indirectly with the subject matter or materials presented in this editorial. This includes honoraria, expert testimony, employment, ownership of stocks or options, patents, or grants received or pending royalties.

Data Availability

The data is exclusively available from the principal author for research purposes only.

Authorship Contribution

All authors contributed significantly to the work, whether in the conception, design, utilization, collection, analysis, and interpretation of data or all these areas. They also participated in the paper's drafting, revision, or critical review, gave their final approval for the version that would be published, decided on the journal to which the article would be submitted, and made the responsible decision to be held accountable for all aspects of the work.

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