Does Intragastric Balloon Treatment for Obesity in Chronic Kidney Disease Heighten Acute Kidney Injury Risk?

Helen L. MacLaughlin\textsuperscript{a, b}, Iain C. Macdougall\textsuperscript{a}, Wendy L. Hall\textsuperscript{b}, Tracy Dew\textsuperscript{a}, Konstantinos Mantzoukis\textsuperscript{c}, Jude A. Oben\textsuperscript{c, d}

\textsuperscript{a}Department of Nutrition and Dietetics, King’s College Hospital NHS Foundation Trust; \textsuperscript{b}Diabetes and Nutritional Sciences Division, King’s College London; \textsuperscript{c}Institute for Liver Health, Royal Free Hospital, University College London, and \textsuperscript{d}Guy’s and St. Thomas’ NHS Foundation Trust, London, UK

Abstract

\textbf{Background:} The outcomes of intragastric balloon (IGB) placement to achieve weight loss in obese patients with chronic kidney disease (CKD) have not been reported to date. This study aimed to assess the safety and efficacy of the IGB as a weight-loss treatment among this patient population. \textbf{Methods:} A prospective, single-arm, ‘first in CKD’ interventional study was conducted in patients with a body mass index >35 kg/m\textsuperscript{2} and CKD stages 3–4, referred for weight loss. After clinical assessment, the IGB was endoscopically inserted into the stomach and kept in place for 6 months. Complications, adverse events, acceptability, weight loss and metabolic responses were monitored over 6 months. \textbf{Results:} Eleven participants were recruited over 18 months. Two patients withdrew (1 prior to IGB insertion and 1 early removal after 3 days due to persistent vomiting) from the study; 9 patients completed the study. There were 5 episodes of acute kidney injury (AKI), occurring in 3 patients. After 6 months, the mean body mass decreased by 9.6% (SD ±6.8). Median waist circumference and total cholesterol decreased significantly (−7.7 cm; interquartile range (IQR) −15.3 to −3.9; and −0.2 mmol/l; IQR −0.6 to −0.05, respectively), with no changes in estimated glomerular filtration rate, blood pressure, triglycerides, adipokines, inflammation, or arterial stiffness measured by carotid-femoral pulse wave velocity. At IGB removal, there was 1 new case each of gastritis and esophagitis. \textbf{Conclusions:} Treatment with IGB has only moderate efficacy on weight loss; yet it results in a high rate of complications in obese patients with established CKD. The risk of AKI may be raised due to increased risk of dehydration secondary to gastrointestinal symptoms associated with IGB placement and reduced baseline kidney function.

Introduction

Obesity is an independent contributory factor to the development and progression of chronic kidney disease (CKD) [1–3]. We have previously demonstrated the efficacy and effectiveness of lifestyle interventions in obese patients with CKD [4, 5]. However, it may be desirable that greater weight loss be achieved in some patients, possibly due to higher body mass index (BMI), comorbidities and/or personal preferences. Several studies have demonstrated that weight loss can be achieved successfully by performing bariatric surgery in obese patients with CKD [6–8]. However, there are challenges with all 3 common surgical procedures (gastric banding, Roux-en-Y gastric bypass, and sleeve gastrectomy) in patients with CKD due to reports of band erosion, nutrient malabsorption, increased risk of hyperoxaluric nephrolithiasis, dehydration and acute kidney injury (AKI) [9–11]. Large studies of registry data indicate that patients with CKD have an increased risk of adverse events following weight loss surgery, which advances as kidney function declines [12, 13]. Patients with stages 3–4 CKD, or moderate to advanced kidney impairment, are 1.5 times more likely to experience a complication after undergoing weight loss surgery than patients without CKD [12].
Intragastric balloon (IGB) treatment is safe and results in significant weight loss over the short term in patients with normal kidney function [14–16]. This may therefore be considered an alternative weight loss treatment method for obese patients with CKD. The IGB is a fluid-filled silicone device that is inserted into the stomach endoscopically and inflated to a fixed volume; this is done in order to restrict intake of food while still retaining the normal stomach function without malabsorption. Complications such as nausea are common and almost 1/3 of patients experience some gastric erosion [17]; however, rare but hazardous complications such as gastric perforation and AKI have been reported [18, 19]. This study assessed the efficacy and safety of the IGB to facilitate weight loss in obese patients with CKD and to explore the effects of weight loss on metabolic syndrome and cardiovascular disease risk factors.

Subjects and Methods

Screening and Recruitment
This was a single-arm, prospective ‘first in CKD’ interventional study in obese patients with CKD. It was designed to establish the efficacy of IGB insertion to achieve weight loss over 6 months, to monitor IGB-related complications and adverse events, and to examine patient acceptability of the procedure. The planned sample size of this initial safety and efficacy study was 12 patients. Obese patients (BMI >35 kg/m²), aged >18, with CKD stages 3–4 (4 variable Modification of Diet in Renal Disease (MDRD) study equation estimated glomerular filtration rate (eGFR) 15–59 ml/min/1.73 m²), referred to dietetics services for weight loss, were eligible for inclusion in the study. The criteria for exclusion were as follows: patients who had undergone previous bariatric surgery or major abdominal surgery, patients who were pregnant or with acute malnutrition identified by Subjective Global Assessment (SGA) [20], or those who were unable to provide written informed consent. The study was conducted with the approval of the National Research Ethics Service North West Greater Manchester South Committee (12/NW/0811) and in accordance with the Helsinki Declaration of 1975, as revised in 2013 [21]. An independent study-monitoring committee oversaw the procedures followed in conducting this study.

Once a 2–5% loss of body mass was achieved through a combination of diet and exercise in our renal weight management clinic [5], patients were referred to the study gastroenterologist for assessment for IGB insertion. Prerequisite weight loss was required to enter the study to demonstrate changes to lifestyle factors, prior to insertion of the IGB. Prior to consenting to participate in the study, all patients were thoroughly informed about the possible benefits and known risks associated with IGB insertion.

Intervention
Patients underwent endoscopic insertion of the soft silicone IGB (Orbera, Allergan) through a procedure performed by an experienced gastroenterologist. Once the IGB was in position, it was filled with 500–700 ml of saline and 10 ml methylene blue dye. After 6 months, the IGB was deflated and removed during endoscopy. At 1 and 2 weeks post IGB placement, serum creatinine was monitored to detect acute changes in kidney function, including episodes of AKI. AKI was defined on the basis of AKIN criteria of at least a 50% increase from baseline, or an acute rise of at least 26.4 μmol/l in 48 h, in serum creatinine [5].

All patients received from the study dietitian verbal and written communication for dietary education for weight loss with the IGB, prior to IGB insertion, and at 2–3 days post IGB insertion through telephone calls, and at 1, 6 and 12 weeks during study visits. Information provided included sample meal plans, portion size guidance, reduction in snacking behaviour, and the importance of maintaining an adequate fluid intake. For the initial 3 days post IGB insertion, only liquids were consumed, followed by a soft diet for 4 days. All patients were advised to drink 2 litres of fluids each day for the first week. From 1 week onwards, normal foods were eaten in very small portions, that is, quantity enough to fit a side plate (with a diameter of 200 mm), plus at least 1.5 litres of non/very low energy containing fluids (includes fat-free milk) consumed in 150–200 ml volumes, in between meals. Eating protein foods (lean meat, fish, pulses, eggs, and reduced fat dairy foods) at every meal was recommended, with the addition of vegetables at main meals, and a small portion of rice,
pasta, bread, potatoes or other starchy vegetables, only if needed to reach satiety.

All patients continued with appropriate medical treatment under the care of their nephrologist and general physician, including the use of angiotensin converting enzyme inhibitors, angiotensin receptor blockers and other blood pressure (BP)-lowering medications, statins, erythropoietin, iron, vitamin D, phosphate binders, and treatment of diabetes and other pre-existing conditions.

Outcomes and Measures
The primary efficacy outcome was defined as at least a 10% reduction in body mass 6 months after IGB insertion. Safety outcomes included complications associated with the IGB, adverse events and acceptability, measured by the early IGB removal rate. Early cessation of the study was planned if consistent inadequate weight loss or major early or late post IGB insertion complications occurred prior to the completion of the study. Events were reviewed by an independent safety-monitoring committee throughout the study. Expected minor adverse events were nausea and vomiting, constipation, reflux and gastrointestinal discomfort. Major adverse events included, but were not limited to AKI, gas-trointestinal obstruction, spontaneous balloon deflation, epigastric pain and reflux.

The secondary outcomes were changes in BP, blood lipids, waist circumference, insulin resistance measured by the homeostasis model of assessment of insulin resistance method (HOMA-IR) [22], proteinuria measured as the urinary protein to creatinine ratio, markers of inflammation, leptin, adiponectin and arterial stiffness, measured by carotid to femoral pulse wave velocity (PWV). Kidney function is reported as eGFR and is calculated using the MDRD study equation; however, it was not selected as a study outcome due to the underestimation of kidney function by eGFR in obesity, and the error associated with measuring changes in kidney function as body size and body composition change [23–26]. So kidney function must be interpreted bearing all these constraints in mind.

Baseline demographic data including date of birth, ethnicity, gender, medical history, current medications, and cause of CKD were recorded at the initial study visit. Using a wall-mounted stadiometer, the height was measured to the nearest 1 cm without the patients wearing shoes. Weight was measured to the nearest 0.1 kg on a calibrated electronic scale with the patient wearing light clothing and no shoes. Absolute weight loss was defined as the difference between baseline weight and weight at the time of measurement; it was calculated as an absolute value and as a percentage of baseline weight.

Sitting BP was measured using an automated sphygmomanometer with an appropriate-sized upper arm cuff, 3 times, and the average of the closest 2 readings was calculated. Waist circumferences were measured to the nearest 0.1 cm at the level of the umbilicus with a calibrated plastic tape measure.

Laboratory Measures
Morning venous blood samples were collected in standardized blood collection tubes (BD Vacutainer, New Jersey) after an overnight fast, for serum glucose, creatinine, leptin, adiponectin, inter-leukin-6 (IL-6), lipids, high-sensitivity C-reactive protein (hs-CRP), and insulin. Quantitative sandwich enzyme-linked immunosorbent assays were used to measure serum IL-6, leptin and adiponectin (R&D systems, USA), and insulin (Insulin Centaur, Siemens, UK). hs-CRP was measured using an immunoturbidimetric assay (PZ Cormay, Poland). Standard enzymatic techniques were used to measure lipid fractions, total cholesterol, and glucose (Siemens Advia 2400, UK). The index of insulin resistance was calculated using the HOMA-IR equation [22], previously validated in patients with CKD [27]. Urine samples were analysed for total protein and creatinine using standard analytical methods and the protein-to-creatinine ratio was calculated.

Arterial Stiffness
Arterial stiffness was measured by carotid to femoral PWV by recording arterial pulse waveforms using the Vicorder oscillometric device (Skidmore Medical, UK), validated against the recognised gold standard SphygmoCor system [28]. To measure PWV, cuffs were placed around the upper thigh and at the level of the carotid artery around the neck of the supine patient. The distance from the sternal notch to the top of the femoral cuff, in a straight line, was measured with a calibrated plastic tape measure, and PWV was calculated from this distance and the pulse transit time between the 2 points. The measurement was performed 3 times, and the average of the closest 2 measures was calculated.
Statistical Analyses
Due to the exploratory nature of this study, the statistical analysis was limited to tests suitable for small samples (SPSS version 22.0: IBM). Descriptive statistics included the calculation of median (interquartile range (IQR)) values over time for continuous outcome variables to measure trends. Changes in the distribution for continuous variables between baseline and 6 months were determined with Friedman’s 2-way analysis of variance by ranks; and the median (with 95% CIs) of the differences between the pre IGB and 6 months post IGB values was calculated using the Hodges- Lehman median difference. Early balloon removals, complications, adverse events, study withdrawals and reasons for withdrawal were collected and analysed descriptively.

Results
Between April 2013 and October 2014, 72 patients satisfied the inclusion criteria and were contacted to participate in the study. Eleven consented to participate, while 1 withdrew from the study prior to IGB insertion. Ten patients had an IGB placed and there was 1 early removal after 3 days due to persistent vomiting; 9 patients (3 male) completed the study. The median (IQR) age was 58 (49–68), with median eGFR being 23 (17–40) ml/ min/1.73 m², and median BMI being 40.2 (36.9–43.4) kg/ m². The majority of patients belonged to the white ethnic group (8 White, 1 Black). Diabetes, with or without co-existing hypertension, was the major cause of CKD in 8 out of 11 patients; 2 patients developed CKD secondary to drug toxicity and 1 patient had lupus nephritis. All patients were hypertensive and prescribed antihypertensive medication. Eight of the 9 patients also had diabetes and all of them required anti-hyperglycaemic therapy. All patients were well nourished at the time of entry into the study, as assessed by SGA, and they all remained well nourished throughout the study.

Primary Efficacy Outcome
The a priori efficacy criterion was at least a 10% reduction in body mass 6 months after IGB insertion. The mean and median percentage weight loss after IGB insertion were 9.6% (SD 6.8%) and 9.1% (IQR 13.6–9%), respectively, after 6 months (fig. 1). Pre-IGB weight loss and the change in weight from this point to the baseline assessment pre-IGB placement is also included in figure 1.

Safety – Complications and Adverse Events
A summary of the complications and adverse events is presented in table 1. In 5 out of 10 (50%) patients, the initial IGB insertion was abandoned due to gastritis. All patients with gastritis were treated with proton pump inhibitors for 6 weeks and all had successful IGB insertion at the second attempt. The IGBearly removal rate was 9% (1/11 patients). There were 5 episodes of AKI post IGB insertion, occurring in 3 patients during the study (prevalence rate 30%). Two patients required hospitalization for severe vomiting, 1 received intravenous rehydration fluids and anti-emetics, and in the other, the IGB was removed 3 days post insertion. AKI occurred due to dehydration secondary to nausea and vomiting and/or ongoing use of diuretics. At the time of IGB removal, there was 1 case each of gastritis and esophagitis, diagnosed during endoscopy, neither of which was present at IGB insertion in these patients. There were no other adverse events directly related to IGB placement in this study.

Post study, after removal of the initial balloon, 4 patients had a second balloon inserted and there were 2 cases of AKI secondary to dehydration in these patients, both requiring hospitalization for dehydration and intractable vomiting; they were treated with intravenous rehydration and anti-emetics. Overall, there was a 50% prevalence rate of AKI, with 5 of the 10 patients having at least 1 AKI event, and there were a total of 7 AKI events from 14 IGB placements, including 2 severe adverse events during the study, defined as unexpected hospitalization related to a study procedure.

Secondary Outcomes
Serial measures and the median of the differences in between baseline and 6 months for the anthropometric, haemodynamic and metabolic outcomes are listed in table 2. After 6 months of IGB treatment, the Hodges Lehman median difference in body weight was −10.4 kg (95% CI −15.9 to −5.2; table 2). BMI, waist circumference, total cholesterol and leptin also decreased significantly after 6 months, and the reduction in low density lipoprotein cholesterol reached statistical significance (p=0.059). BP, proteinuria, insulin resistance, arterial stiffness, markers of inflammation, other lipid fractions and adiponectin did not change in this cohort of obese patients with CKD, following treatment with IGB for 6 months.
Discussion

This study was the first to examine the safety and efficacy of IGB treatment for obesity in patients with moderate to advanced CKD. A priori efficacy of IGB treatment for obesity was almost reached with 9.6% weight loss, but there was a high rate of AKI following IGB insertion. The percentage of total body weight loss at 6 months was less than the reported 12.2–13.6% weight loss in pooled meta-analyses of weight loss following IGB treatment in obese patients with normal kidney function [15, 16]. However, IGB treatment resulted in greater weight loss at 6 months compared to lifestyle treatment with a combination of dietary and physical activity changes with pharmacotherapy in obese patients with CKD [5]. The incidence of gastric lesions before IGB treatment IGB insertion. Gastritis is thought to be common in patients with CKD, although there is a paucity of studies examining its incidence in patients with CKD not requiring renal replacement therapy. After IGB treatment, its incidence appears consistent with that reported in other studies in patients with normal kidney function. A 2007 Cochrane review of IGB treatment of reported a tenfold increased relative risk for gastric erosion compared to no IGB insertion [29], and 2 systematic reviews reported gastric erosion in 12–32% of patients post IGB treatment [15, 17]. This high rate of gastric erosion may also negatively impact the quality of life and medication burden, as treatment usually requires additional drug therapy. There was one case of early removal of the IGB due to poor tolerance. A 2008 systematic review and meta-analysis reported an early removal rate of 4.2% [16]. Due to the small sample size, it is not possible to compare the rate of early balloon removal to that in other studies.

Very few studies on IGB treatment report AKI as a complication, with reports of acute kidney failure, functional renal failure, or renal insufficiency at rates of 1.1–4.5% of patients studied [18, 19, 30, 31]. In one study, acute renal failure was reported in 2 patients with diabetes being treated with metformin [19]. Factors associated with AKI include older age, proteinuria, hypertension, diabetes, and pre-existing CKD [32, 33]. The incidence of AKI in our study may have been higher than previously reported due to the increased risk of AKI associated with CKD, but also due to the close monitoring of kidney function 1 and 2 weeks post IGB insertion, due to the anticipated risk of AKI, secondary to volume depletion caused by nausea and vomiting.

Interventions to reduce the risk of AKI, such as using sick day guidance [34] to temporarily cease ACE inhibitors and diuretics in patients with CKD at IGB insertion, until adequate fluid intake is established and post-procedure nausea and vomiting subsides, could potentially reduce complications following IGB placement in patients with CKD. The benefit of this strategy needs careful consideration against the competing risks of possible uncontrolled hypertension and hypervolemia with temporary cessation of these therapies. Additionally, prophylactic intravenous fluid therapy for several days may reduce the risk of dehydration; however, this is unlikely to be cost effective or practical if administering this therapy requires several days of hospitalization.

Weight loss is associated with a reduction in inflammatory cytokines, reduced insulin resistance and increased adiponectin [22, 23], but these relationships have not yet been well studied in obese patients with CKD. After IGB treatment, improvements in waist circumference, leptin and total cholesterol were evident, indicating a strong likelihood of a reduction in abdominal fat. Insulin resistance, CRP, and IL-6 all demonstrated a trend towards improvement in this study, also evident in previous studies of IGB in non-CKD participants [35–38]. Due to the small sample size in this ‘first in CKD’ proof of concept safety study, trends in changes in metabolic factors may be useful for developing a hypothesis for the mechanisms associated with weight loss that may contribute to a reduction in CVD risk in this high-risk group of patients. Arterial stiffness, measured by PWV, also demonstrated a trend towards improvement with weight loss in this study. PWV is a slow-changing marker of arterial vasculature health, and any beneficial effect may become more marked with longer term follow-up. A search of the literature did not identify any previous reports on the effect of weight loss with IGB treatment on arterial stiffness in any population. Increased PWV, indicating increased large vessel arterial stiffness, is associated with obesity [39], and is an independent predictor of cardiovascular events in patients with stages 4–5 CKD, even after adjustment for established cardiovascular risk factors [40]. A reduction in arterial stiffness with a therapeutic intervention such as weight loss represents a true reduction in arterial wall damage, rather than simply a change in cardiovascular risk scores [41]. These preliminary results indicate that weight loss in obese patients with CKD due to IGB treatment may contribute to reduced CVD risk, with the potential mechanism identified as a reduction in inflammation leading to the modification of medial arterial wall structure. This hypothesis remains to be tested in future studies if improved safety can be demonstrated with a refined protocol including preventative measures to reduce the risk of AKI, as discussed.

The major limitations of this study are the single-arm study design and small sample size. These limitations are true of most proof-of-concept studies, and the design mirrors that of phase 2 drug treatment studies to
establish safety and efficacy in a small number of patients. The strength of this study was the close monitoring of kidney function post IGB insertion to examine safety. Using this design, we have detected a potentially high rate of AKI in a small number of patients, and we acknowledge that the study was not designed to detect metabolic and haemodynamic effects of weight loss in obese patients with CKD.

The adverse event rate indicates that the IGB procedure, while moderately effective for weight loss, may carry a high rate of complications in obese patients with CKD. The risk of AKI may be raised due to a combination of increased dehydration secondary to gastrointestinal symptoms associated with IGB placement and reduced baseline kidney function. Studies including preventative measures to reduce the risk of AKI, including cessation of ACE inhibitors and diuretics for a defined period, are recommended.

Disclosure Statement
The results of this study have not been published previously in whole or part, except in the abstract form. The authors have no conflicts of interest to declare related to this work.

References


### Table 1

<table>
<thead>
<tr>
<th>Complications/adverse events</th>
<th>Occurred, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nausea and vomiting in first week</td>
<td>8 of 10 patients</td>
</tr>
<tr>
<td>AKI (5 episodes in 3 of 10 patients)</td>
<td></td>
</tr>
</tbody>
</table>

**Patient 5**

1st AKI: 1 week post IGB insertion, vomiting daily, creatinine rose from 226 to 279 μmol/l, returned to baseline 1 week later; 2nd AKI: 5 months post insertion, admitted with dehydration secondary to vomiting, AKI, (peak creatinine 650 μmol/l, potassium 3.3 mmol/l), diagnosis of viral gastroenteritis, IV fluids and anti-emetics given, creatinine fell to 310 μmol/l, 5 days later at discharge; 3rd AKI: 6 months post insertion, readmitted with dehydration secondary to vomiting, AKI, hyponatremia (Na 114 mmol/l), diagnosis of esophageal candidiasis, IV fluids and anti-emetics given, IGB removed, creatinine 179 μmol/l, 1-week post discharge

**Patient 7**

1-week post IGB insertion, creatinine rose from 460 to 538 μmol/l, nil vomiting reported, drinking 2 litres fluids/day, 2 weeks post IGB insertion creatinine 563 μmol/l, loperamide, furosemide and indapamide ceased, creatinine returned to baseline 4 weeks post IGB insertion

**Patient 9**

1 month post IGB insertion, creatinine rose from 280 μmol/l, 1 week post insertion to 474 μmol/l, furosemide and candesartan ceased, creatinine fell to 301 μmol/l, 1 week later

**Hospitalization for intravenous fluid therapy (2 of 10 patients)**

**Patient 5**

As detailed above

**Patient 10**

Admitted 1 day post IGB insertion with intractable vomiting, creatinine 144 μmol/l at baseline to 173 μmol/l, 2 days post insertion, severe vomiting and nausea continued until IGB removed on day 3 post insertion

**Gastritis/esophagitis**

2 of 9 patients

**Delayed IGB insertion**

5 of 10 patients
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Baseline</th>
<th>3 months</th>
<th>6 months</th>
<th>Median of differences</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight, kg</td>
<td>111.5 (103.6 to 116.7)</td>
<td>105.4 (99.3 to 110.1)</td>
<td>100.0 (97.7 to 109.5)</td>
<td>-10.4 (-15.9 to -5.2)</td>
<td>0.003</td>
</tr>
<tr>
<td>BMI kg/m²</td>
<td>40.2 (36.9 to 43.1)</td>
<td>36.6 (33.9 to 42.0)</td>
<td>35.2 (32.4 to 42.5)</td>
<td>-3.6 (-5.6 to -2.0)</td>
<td>0.003</td>
</tr>
<tr>
<td>Waist, cm</td>
<td>123.9 (122.1 to 124.2)</td>
<td>120.6 (114.7 to 134.6)</td>
<td>118.5 (110.7 to 138.5)</td>
<td>-7.7 (-15.2 to -3.9)</td>
<td>0.003</td>
</tr>
<tr>
<td>Waist hip ratio</td>
<td>1.00 (0.96 to 1.06)</td>
<td>0.96 (0.95 to 1.05)</td>
<td>0.96 (0.92 to 1.02)</td>
<td>-0.03 (-0.06 to 0.01)</td>
<td>0.1</td>
</tr>
<tr>
<td>eGFR, ml/min/1.73 m²</td>
<td>23 (17 to 40)</td>
<td>32 (19 to 39)</td>
<td>30 (15 to 36)</td>
<td>0 (-4 to 4)</td>
<td>0.7</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>127 (118 to 146)</td>
<td>130 (116 to 140)</td>
<td>129 (111 to 135)</td>
<td>-8 (-20 to 5)</td>
<td>0.7</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>72 (65 to 76)</td>
<td>71 (64 to 75)</td>
<td>70 (64 to 73)</td>
<td>-1 (-16 to 3)</td>
<td>0.7</td>
</tr>
<tr>
<td>Cholesterol, mmol/l</td>
<td>3.7 (3.5 to 4.3)</td>
<td>3.1 (2.9 to 4.4)</td>
<td>3.6 (3.0 to 4.3)</td>
<td>-0.2 (-0.6 to -0.1)</td>
<td>0.03</td>
</tr>
<tr>
<td>HDL, mmol/l</td>
<td>1.1 (0.8 to 1.5)</td>
<td>1.1 (0.9 to 1.5)</td>
<td>1.0 (0.9 to 1.5)</td>
<td>0 (-0.2 to 0.1)</td>
<td>1.0</td>
</tr>
<tr>
<td>LDL, mmol/l</td>
<td>1.6 (1.4 to 2.4)</td>
<td>1.6 (1.2 to 2.2)</td>
<td>1.6 (1.1 to 2.0)</td>
<td>-0.3 (-0.6 to 0)</td>
<td>0.059</td>
</tr>
<tr>
<td>Triglycerides, mmol/l</td>
<td>2.1 (1.3 to 2.8)</td>
<td>1.6 (1.4 to 1.8)</td>
<td>1.9 (1.3 to 2.7)</td>
<td>0 (-0.6 to 0.6)</td>
<td>0.7</td>
</tr>
<tr>
<td>PWV, m/s</td>
<td>8.7 (6.7 to 9.6)</td>
<td>8.2 (6.5 to 9.0)</td>
<td>8.1 (7.8 to 9.4)</td>
<td>0.37 (-1.2 to 2.0)</td>
<td>1.0</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>8.7 (5.0 to 28.5)</td>
<td>7.5 (3.5 to 21.7)</td>
<td>6.1 (1.8 to 24.3)</td>
<td>-1.4 (-10.7 to 4.1)</td>
<td>1.0</td>
</tr>
<tr>
<td>hs-CRP, μg/l</td>
<td>7.0 (3.5 to 17.7)</td>
<td>5.1 (3.0 to 20.6)</td>
<td>4.4 (2.7 to 13.2)</td>
<td>-2.4 (-0.9 to 20)</td>
<td>0.3</td>
</tr>
<tr>
<td>IL-6, ng/l</td>
<td>7.2 (1.9 to 10.2)</td>
<td>8.3 (4.5 to 16.7)</td>
<td>5.7 (1.7 to 11.1)</td>
<td>0.3 (-4.1 to 2.0)</td>
<td>0.3</td>
</tr>
<tr>
<td>Leptin, μg/l</td>
<td>63.6 (46.3 to 100.0)</td>
<td>61.3 (38.2 to 73.9)</td>
<td>55.0 (30.7 to 78.9)</td>
<td>-22.7 (-47.2 to -0.9)</td>
<td>0.03</td>
</tr>
<tr>
<td>Adiponectin, μg/l</td>
<td>8.5 (4.5 to 15.3)</td>
<td>12.1 (4.6 to 12.6)</td>
<td>7.6 (4.0 to 20.5)</td>
<td>-0.18 (-13.0 to 2.8)</td>
<td>0.7</td>
</tr>
<tr>
<td>Urinary PCR, mmol/mg</td>
<td>45.3 (12.8 to 121.4)</td>
<td>27.9 (12.9 to 103.9)</td>
<td>48.5 (12.9 to 84.7)</td>
<td>-14.7 (-79.2 to 5.3)</td>
<td>0.48</td>
</tr>
</tbody>
</table>

1. Hodges-Lehman median of the intra-individual differences from baseline to 6 months and 95% CI.

HDL = high density lipoprotein; LDL = low density lipoprotein; PCR = plasma creatinine ratio.

Table 2: Anthropometric, haemodynamic and metabolic parameters (median and IQR) pre and post IGB placement in obese patients with CKD.
Figure 1. Body weight changes with diet and exercise only and after IGB insertion in obese patients with CKD (dashed lines – individual patient data, heavy line – median body weight change).