



## The Effect of Laparoscopic Sleeve Gastrectomy on Adiponectin Level



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### Abstract

**Background:** Obesity is accompanied with chronic low-grade inflammation contributing to obesity-related diseases. Sleeve Gastrectomy (SG) has been shown to be a very effective solo option for long-term weight loss and control of obesity-associated morbidity.

**Objective:** measuring Adiponectin, c-reactive protein (CRP), erythrocyte sedimentation rate (ESR), total leucocytes count (TLC) and hemoglobin A1C (HbA1C) before and after 4 months of SG to assess the effect of sleeve on the inflammatory profile of these patients.

**Settings:** one bariatric center Kasr Al-Ainy teaching Hospital in Egypt

**Patients and Methods:** a prospective study included 40 patients who underwent a laparoscopic sleeve gastrectomy (SG). Preoperative and 4 months postoperative Adiponectin, CRP, ESR, TLC and HbA1C levels were measured, the results were documented and analyzed then the results were correlated to the baseline results and to change in body mass index (BMI).

**Results:** Baseline tendency towards inflammation in our patients was represented with elevated mean CRP, ESR1 and ESR2, high normal mean TLC and decreased mean Adiponectin. 4 months after sleeve, mean adiponectin level rose from 1.16 mg/L preoperatively to 1.55 mg/L postoperatively, mean CRP dropped from 13.43 mg / L to 6.18 mg/L, mean ESR1 and ESR2 dropped from 33.97 mm / hr and 59.83 mm / hr to 23.27 mm / hr and 45.08 mm / hr respectively and mean TLC dropped from 7.86 103 / cm to 6.27 103/cm.

**Conclusion:** Obese patients have baseline low grade inflammation. SG causes significant improvement of obesity related inflammation on the short term, the effect derived by SG on improving inflammatory markers is not totally dependent on reduction of BMI.

**Keywords:** morbid obesity, sleeve gastrectomy, adiponectin, obesity related inflammatory status, CRP, ESR, TLC

### Introduction

Obesity is becoming a world pandemic that affects more than one third of the population around the globe, obesity related comorbidities are top listed as the leading causes of death.

Adipose tissue that was recognized simply as an inert storage organ is now considered as an endocrine organ and part of an innate immune system. Factors secreted from adipose tissue are very important contributors in regulation of metabolism and inflammatory responses and obesity is accompanied with various derangements in the Metabolic and inflammatory functions mediated by adipose tissue ends up with suffering of the obese patients from a chronic low-grade inflammatory state.

Bariatric surgeries are becoming the gold standard treatment of morbid obesity with dramatic effects regarding potent weight loss and prompt amelioration of obesity related comorbidities like diabetes, hypertension and others.

Sleeve gastrectomy is Gaining More reputation as Solo Surgery with perfect short and medium term effects.

### Aim of the work

The Aim of Study is to assess the Effect of sleeve gastrectomy on the inflammatory profile of our patients, and to deepen our understanding about the metabolic and inflammatory effects of obesity and how the surgical intervention with sleeve gastrectomy can reverse these changes.

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It also aims to emphasize about the metabolic effects derived by sleeve gastrectomy and if these effects are purely through decreasing body weight or other mechanisms involved to drive the metabolic actions of sleeve gastrectomy by assessing the relationship between changes in inflammatory markers after sleeve and changes in Body mass Index (BMI)

### Patients and Methods

This is a prospective randomized study that included 40 patients. All patients underwent a (SG) as a bariatric procedure over 6 month period in Kasr Al-Ainy teaching Hospital during the period from August 2017 to February 2018. Preoperative and postoperative evaluation followed the same standard protocol and included a thorough personal, medical and surgical history, complete labs including extra specific labs for this study, complete endocrinal workup, psychological testing, and counseling by a dietician and followed a low caloric diet in arrange of one to three weeks according to BMI. All comorbidities that increase perioperative risk were controlled before surgery as far as possible.

The study included 16–54 years old patients of both genders, who are given consent and comply with the evaluation and treatment schedule with BMIs of 40 kg/m<sup>2</sup> or more, or between 35 kg/m<sup>2</sup> and 40 kg/m<sup>2</sup> with other comorbidity that could be improved if they lost weight. All appropriate non-surgical measures have been tried but have failed to achieve or maintain adequate, clinically beneficial weight loss for at least six months. Patients are receiving or will receive management in a specialist obesity service. Patients are generally fit for anesthesia and surgery. Patients commit to the need for long-term follow up. Patients needed to demonstrate the absence of significant psychopathology that could limit their ability to understand the procedure and comply with the medical, surgical, and/or behavioral recommendations.

On the other hand, the study excluded patients with chronic inflammatory diseases, type 2 diabetic patient > 10 years duration, pregnancy or lactation at screening or surgery, patients with a documented history of toxic drug and/or steroid supplements within 30 days of the screening visit or during study participation, alcohol abuse within 2 years of the screening visit, previous mal-absorptive or restrictive procedures performed for the treatment of obesity, any condition that would preclude compliance with the study. Such conditions included: congenital or acquired anomalies of the gastrointestinal tract (e.g., atresia or stenosis), severe cardiopulmonary disease or other serious organic disease making the subject a high-risk surgical candidate, uncontrolled hypertension, and portal hypertension. Additional exclusion criteria included

chronic or acute upper gastrointestinal bleeding conditions (e.g., gastric or esophageal varices) cirrhosis, congenital or acquired intestinal telangiectasia, esophageal dysmotility, or Barrett's esophagus, hiatal hernia, previous surgery of the foregut (i.e., hiatal hernia repair or previous gastric surgery), pancreatitis, an immunocompromised status or autoimmune connective tissue disease.

### Preoperative preparation

Patients were consented about the nature of the research, and each patient understood and agreed to the procedure. All patients underwent a standard evaluation preoperatively.

Blood tests were requested in the form of: Complete blood picture, Fasting blood sugar, Lipid profile (cholesterol, LDL, HDL, triglycerides), Clinical chemistries (serum albumin, ALT, AST, GGT, Urea, Creatinine), Prothrombin time and concentration, Serum Adiponectin, quantitative CRP, ESR, HbA1c.

Also radiological investigations done including: abdominal ultrasonography, chest X-ray, pulmonary function tests and upper GI endoscopy.

Thromboembolic prophylaxis with subcutaneous low molecular weight heparin was administered on the evening prior to surgery and continued daily from the first postoperative day until the patient was ambulant for a maximum of 14 days postoperatively.

### Surgical procedure

#### Setting

All procedures were performed under general anesthesia with the patient in supine position and the surgeon positioned between the legs of the patient (French position) after applying compression stockings on the patient lower legs. The patients were firmly secured to the operating table to allow for placement in the anti-Trendelenburg position as required. Carbon dioxide insufflation was used to create pneumoperitoneum using the Veress needle in the left hypochondrium for all cases maintaining a 15 mmHg intra-abdominal pressure and flow rate between 2–2.5 liters/minute to be increased up to 10 Liters/minute after ports insertion. After creation of pneumoperitoneum, a five trocar approach was used. A 12 mm subxyphoid trocar serves as a liver retractor. One 12 mm trocar between the subxyphoid trocar and the umbilicus serves as the optical port (camera port), and an additional two: 12 mm and 15 mm working ports are placed 3–4 cm under the left and right costal margin. Another 5 mm left subcostal anterior axillary line trocar for stomach traction (assistant port).

### ***Surgical technique***

The separation of the greater gastric curvature is divided starting 3-6 cm from the pylorus and proceeding to the angle of His. The gastroepiploic vessels along the greater curvature of the stomach and the short gastric vessels are divided using the ligasure device (Covidien, Massachusetts, USA). Dissection of adhesions between the back of the stomach wall and the pancreas is performed. A 36-Fr calibrating bougie was introduced by anesthesiologist into the stomach and advanced along the lesser curvature into the pyloric channel and duodenal bulb. The stomach was divided using an ENDO GIA stapler with 4.8-mm staples (green cartridge) is introduced through the 15-mm port which is located at the right quadrant, to begin the division of the antrum 3 to 6 cm proximal to the pylorus. Gastric tubulization is completed by dividing the gastric corpus straight to the angle of His angle, applying four to five blue cartridges of the 3.5-mm ENDO GIA stapler. In all cases the resected stomach was removed via 12 mm port without the need to enlarge it further. Routine placement of drain at the operative bed was done in all cases.

### **Post-operative**

#### ***Diet regimen***

Patients were encouraged of early mobility few hours postoperative. Anticoagulation for DVT prophylaxis (enoxaparin 80 IU/day sc) was given till 14 days post-operatively. We started administration of IV proton pump inhibitors (PPI) from the first day post-operatively, and continued orally after patients started oral feeding for 6-8 weeks. Patients started oral fluid intake on the second post-operative day, after a gastrografin study showing no evidence of gastric leak. Gradually diet changed from fluid to solid during 6 to 8 weeks.

All patients were advised to have oral fluids for the first 15 days followed by soft diet until the first postoperative month, and then gradually proceed to regular food with exception of high sugar and fatty foods.

#### **Follow-up and data collection:**

The selected patients were followed up at 4 months  $\pm$  8 weeks from the day of surgery, BMI was calculated, Blood tests were requested in the form of complete blood picture, Serum Adiponectin, quantitative CRP, ESR and HbA1c. To be related to the preoperative results for the same patients.

#### **Technique of Adiponectin Measurement**

We used Human Adiponectin ELISA Kit for Adiponectin measurement<sup>(74)</sup>.

#### ***Assay principal***

This kit is an Enzyme-Linked Immunosorbent Assay (ELISA). ADP is added to the wells pre-coated with ADP monoclonal antibody. After incubation a biotin-conjugated anti-human ADP antibody is added

and binds to human ADP. After incubation unbound biotin-conjugated anti-human ADP antibody is washed away during a washing step. Streptavidin-HRP is added and binds to the biotin-conjugated anti-human ADP antibody. After incubation unbound Streptavidin-HRP is washed away during a washing step. Substrate solution is then added and color develops in proportion to the amount of human ADP. The reaction is terminated by addition of acidic stop solution and absorbance is measured at 450 nm.

#### ***Specimens collection***

**Serum** Allow serum to clot for 10-20 minutes at room temperature. Centrifuge at 2000-3000 RPM for 20 minutes<sup>(74)</sup>.

#### ***Reagent preparation***

All reagents should be brought to room temperature before use. **Standard** Reconstitute the 120  $\mu$ l of the standard (64mg/L) with 120  $\mu$ l of standard diluent to generate a 32mg/L standard stock solution. Allow the standard to sit for 15 mins with gentle agitation prior to making dilutions. Prepare duplicate standard points by serially diluting the standard stock solution (32mg/L) 1:2 with standard diluent to produce 16mg/L, 8mg/L, 4mg/L and 2mg/L solutions. Standard diluent serves as the zero standard (0 mg/L). Any remaining solution should be frozen at -20°C and used within one month. Dilution of standard solutions suggested are as follows: Wash Buffer Dilute 20ml of Wash Buffer Concentrate 30x into deionized or distilled water to yield 500 ml of 1x Wash Buffer. If crystals have formed in the concentrate, mix gently until the crystals have completely dissolved<sup>(1)</sup>.

#### **Statistical methods:**

Data were coded and entered using the statistical package Statistical Package for the Social Sciences (SPSS) version 25. Data was summarized using mean, standard deviation, median, minimum and maximum in quantitative data and using frequency (count) and relative frequency (percentage) for categorical data. For comparison of serial measurements within each patient the non-parametric Wilcoxon signed rank test was used<sup>(2)</sup>.

Correlations between quantitative variables were done using Spearman correlation coefficient<sup>(3)</sup>. P-values less than 0.05 were considered as statistically significant.

### ***Results***

#### **Demographic data**

##### **1-Gender distribution**

**Table (1):** Gender distribution

		Count	%
Sex	M	9	22.5%
	F	31	77.5%

**2-Age distribution**

The age of the patients ranged between 16 years and 54years.

**Table (2):** Age distribution

	Mean	SD	Median	Minimum	Maximum
Age	34.20	8.85	32.00	16.00	54.00

**3-Weight, Height and BMI**

The patients preoperative BMI ranged from 42-68 with mean 48.68 Kg/ m<sup>2</sup>

**Table (3):** Weight, height and pre- operative BMI

	Mean	Standard Deviation	Median	Minimum	Maximum
Height	164.51	8.32	165.00	150.00	182.00
weight	132.14	17.50	130.00	100.00	170.00
BMI	48.68	5.00	48.00	42.00	68.00

**Medical and Surgical History**

All the 40 patients were diabetic, 20 with no surgical history and the others as table (4).

**Table (4):** Patients history

		Count	%
Diabetes	Yes	40	100.0%
	No	0	0.0%
HTN	Yes	3	7.5%
	NO	37	92.5%
previous surgery	Appendectomy	4	10.0%
	appendectomy, CS	1	2.5%
	CS	13	32.5%
	hemi thyroidectomy	1	2.5%
	Hydrocele	1	2.5%
	No	20	50.0%

**BMI change after Sleeve gastrectomy**

4 months postoperative BMI ranged from 32.5-62.

**Table (5):** BMI change post (SG).

	Mean	SD	Median	Minimum	Maximum	P value
BMI pre	48.50	4.89	48.00	42.00	68.00	<0.001
BMI post	41.46	5.08	40.45	32.50	62.00	

**Adiponectin Levels Before sleeve Gastrectomy and Changes after surgery**

**Table (6):** Results showing Significant Increase in Adiponectin Post (SG) (P value<0.001).

	Mean	SD	Median	Minimum	Maximum	P value
<b>Baseline Adiponectin</b>	1.16	.46	1.10	.60	3.20	<0.001
<b>Adiponectin Post 4 months</b>	1.55	1.26	1.30	.70	8.60	
<b>Adiponectin change</b>	.39	1.24	.30	-.70-	7.50	

Results also showed weak negative linear relationship Between BMI change and Adiponectin Level change post (SG), (P value 0.029). And this denotes that reduction in BMI Contributes to increase of adiponectin level but also that it is not the sole contributor to this increase and other factors share in this effect.

**Table (7):** Relationship between BMI change and Adiponectin Level change Post sleeve.

		BMI change
<b>Adiponectin change</b>	<b>Correlation Coefficient</b>	-.359-
	<b>P value</b>	.029

### CRP Levels Before sleeve Gastrectomy and Changes after surgery

**Table (8):** Results showing Significant Decrease in CRP Post sleeve Gastrectomy (P value<0.001).

	Mean	SD	Median	Minimum	Maximum	P value
<b>CRP pre</b>	13.43	14.27	8.40	.70	60.00	<0.001
<b>CRP post</b>	6.18	5.37	4.40	.20	23.00	
<b>CRP change</b>	-7.25-	10.89	-3.50-	-45.50-	4.10	

Results showing that there is no direct relationship between reduction in BMI and CRP reduction, denoting several metabolic mechanisms involved in this change.

**Table (9):** Relationship between BMI change and CRP Level change Post sleeve.

		BMI change
<b>CRP change</b>	<b>Correlation Coefficient</b>	.206
	<b>P value</b>	.221

### ESR Levels Before sleeve Gastrectomy and Changes after surgery

**Table (10):** Results showing Significant Decrease in ESR in both first and second hours Post sleeve Gastrectomy (P value<0.001).

	Mean	SD	Median	Minimum	Maximum	P value
<b>ESR pre (1hr)</b>	33.97	16.22	33.00	11.00	100.00	<0.001
<b>ESR post (1hr)</b>	23.27	12.22	21.00	7.00	74.00	
<b>ESR pre (2hrs)</b>	59.83	19.53	65.00	25.00	98.00	<0.001
<b>ESR post (2hrs)</b>	45.08	19.54	44.00	12.00	99.00	
<b>ESR 1st hour change</b>	-10.70-	7.02	-8.00-	-28.00-	-2.00-	
<b>ESR 2nd hour change</b>	-16.25-	11.82	-14.00-	-42.00-	12.00	

Results showing that there is no direct relationship between reduction in BMI and ESR reduction, denoting several metabolic mechanisms involved in this change.

**Table (11):** Relationship between BMI change and ESR change Post sleeve.

		BMI change
<b>ESR 1st hour change</b>	<b>Correlation Coefficient</b>	-.083-
	<b>P value</b>	.625
<b>ESR 2nd hour change</b>	<b>Correlation Coefficient</b>	.015
	<b>P value</b>	.930

### TLC Levels Before sleeve Gastrectomy and Changes after surgery

**Table (12):** Results showing Significant Decrease in TLC Post sleeve Gastrectomy (P value<0.001).

	Mean	SD	Median	Minimum	Maximum	P value
TLC pre	7.86	1.84	8.00	4.10	11.00	<0.001
TLC post	6.27	1.39	6.20	4.00	9.10	
TLC change	-1.59-	1.39	-1.20-	-4.50-	.80	

Results showing that there is no direct relationship between reduction in BMI and TLC reduction, denoting several metabolic mechanisms involved in this change.

**Table (13):** Relationship between BMI change and TLC change Post sleeve.

		BMI change
TLC change	Correlation Coefficient	-.164-
	P value	.333

### HbA1c Levels Before sleeve Gastrectomy and Changes After surgery

**Table (14):** Results showing Significant Decrease in HbA1c Post sleeve Gastrectomy (P value<0.001).

	Mean	SD	Median	Minimum	Maximum	P value
Hb A1c pre	7.2	.43	7.10	6.60	8.20	<0.001
Hb A1c post	6.6	.45	6.60	5.30	7.40	

Results showing that there is no direct relationship between reduction in BMI and HbA1c reduction, denoting several metabolic mechanisms involved in this change.

**Table (15):** Relationship between BMI change and HbA1c change Post sleeve.

		BMI change
Hb A1 c change	Correlation Coefficient	.129
	P value	.427

### Discussion

In obesity, chronic low-grade inflammation and dysfunctional adipose tissue with its altered adipocytokine patterns seem to play a crucial role in development and progression of obesity-related diseases. Bariatric surgery has been shown to be a very effective option weight loss in morbidly obese patients, leading not only to a reduction in obesity-associated morbidity, but also overall mortality probably because of ameliorations of plasma lipids and glucose homeostasis, changes in adipocytokine pattern and reduced subclinical inflammation, respectively <sup>(4)</sup>.

Sleeve Gastrectomy has been increasingly performed as a stand-alone bariatric procedure with good weight loss and resolution of obesity related comorbidities <sup>(5)</sup>.

In this Study we chose to evaluate and monitor the obesity related low-inflammatory state -by assessing several inflammatory markers-, and to evaluate the impact of sleeve gastrectomy on these markers and the relation between weight loss and improvement of our patient's inflammatory markers values, and for this study we chose Adiponectin, CRP, ESR and TLC as inflammatory markers to be measured and also we measured Hb A1c as a marker for glycemic control.

First of All, we noticed a baseline tendency towards inflammation in our patients represented with elevated CRP with preoperative mean of 13.43 mg/l with normal reference range of 0-6 mg /l, also we found prolonged ESR1 and ESR2 to the mean of 33.97 mm/hr and 58.83mm/hr respectively with normal values of less than 20mm/hr for both, furthermore the mean baseline TLC was normal but with tendency towards high normal with baseline mean of (7.86) 10<sup>3</sup> /cm with normal range from (4-11) 10<sup>3</sup> /cm, Finally we found decreased baseline adiponectin with baseline mean of 1.16 mg/L with normal range 2-20 mg/l.

These results coincide with results from various studies, including a study done by Illán-Gómez, et al. <sup>(6)</sup> in 2012 that denoted statistically significant difference in Adiponectin, CRP and other inflammatory markers between obese and lean people relating obesity to chronic low grade inflammatory state.

Adiponectin, which is an anti-inflammatory and insulinomimetic chemokine recreated from Adipocytes, with higher levels in lean people than in obese patients was our main target for study, bariatric surgery appears to improve systemic adiponectin levels, especially in the long term. Studies that have examined this adipokine in the short term (7 and 15 days, 1 and 3 months) are less conclusive.

Our results showed that (SG) resulted in statistically significant increase in adiponectin level 4 months post sleeve gastrectomy compared to baseline adiponectin level with P value less than 0.001, with mean Adiponectin of 1.16 mg/L preoperatively increased to 1.55 mg/L postoperatively.

These results coincides with a recent study done by Jesús M. Gomez-Martin et al. in 2017, they studied 20 obese women submitted to laparoscopic Roux en Y gastric bypass (RYGB) and 20 to sleeve gastrectomy (SG). Twenty control women also included. Both patients and controls were followed up for one year after surgery or conventional treatment with diet and exercise, respectively. Serum adiponectin was measured at baseline, 6 months and 1 year after, and regarding adiponectin concentrations, there was an increase after surgery of a higher magnitude after RYGB than after SG whereas no changes were observed in the control women submitted to conventional diet therapy <sup>(7)</sup>.

Another study done by Sdralis, El, et al. <sup>(8)</sup> at 2013 in which Thirty-one obese patients were randomized into two groups: SG alone or with omentectomy. They measured various inflammatory markers including Adiponectin and high-sensitivity C-reactive protein (hs-CRP) before surgery and at 7 days, and 1, 3 and 12 months after surgery, they noted that adiponectin levels increased from the seventh postoperative day in the omentectomy group prior to any significant weight loss while in the sleeve gastrectomy without omentectomy group, the increase was not statistically significant in the early phases but became significant 12 months postoperatively.

A positive correlation between the reduction of BMI and increase in Adiponectin level was revealed, and this denotes a direct relationship between Adiponectin increase and decrease in BMI caused by Bariatric surgery, however, the increase in Adiponectin seems to be mediated by several mechanisms not solely by change in BMI.

CRP, which is a well-known acute phase reactant, was evaluated in our study for several reasons, including that it is a very important marker for the risk of complications related to metabolic syndrome as coronary heart disease, stroke risks and others, and as other inflammatory markers it was elevated preoperatively with a mean of 13.43 mg / L with normal reference range from 0-6 mg/L and the level of CRP showed statistically significant drop 4 months after sleeve gastrectomy with a mean of 6.18 mg/L with P value of less than 0.001. These results coincide with most of the studies in literature for example Mallipedhi, et al in a

study published in 2014 noticed a drop of CRP level with 44.8% at 6 months post sleeve gastrectomy compared to baseline <sup>(9)</sup>.

We also studied the change in ESR and TLC between baseline and 4 months after sleeve gastrectomy, and for instance these markers were less commonly studied and quite few studies addressed their change after bariatric surgeries.

At this study significant decrease in ESR in both first and second hours Post sleeve Gastrectomy (P value < 0.001) was revealed, with the mean ESR1 and ESR2 dropped from 33.97 mm / hr and 59.83 mm / hr respectively preoperatively into 23.27 mm / hr and 45.08 mm / hr respectively 4 months postoperatively, moreover the TLC significantly decreased TLC post sleeve gastrectomy (P value < 0.001) and the mean TLC was  $7.86 \times 10^3$  / cm preoperatively and  $6.27 \times 10^3$  / cm 4 months after surgery, and again these results denoting the significant effect of bariatric surgery on the inflammatory status of obese patients.

In 2011, Johansson et al. <sup>(10)</sup> did a study on the effect of roux en y gastric bypass on several inflammatory markers including ESR and TLC, the study included Twenty-one consecutive patients And his study stated that a significant ESR decrease by 35% was observed after RYGBP and also significant TLC decrease by 20% after RYGBP both 1 year after surgery.

Our study showed that there was statistically significant decrease in fasting HbA1c blood level 4 months post sleeve gastrectomy (p value less than 0.001), where HbA1c is a good indicator for the glycemic control of the patients with type 2 diabetes, In 2011, NoCCA, et al performed a multicenter prospective study at 1 year, on the impact of (SG) and laparoscopic gastric bypass on HbA1c blood level and pharmacological treatment of type 2 diabetes mellitus in severe or morbidly obese patients in which there was significant decrease in fasting HbA1c level after 1 year postoperatively in both surgeries and the percentage of patients who stopped pharmacological treatment (remitters) was 75.8% of 33 patients who underwent sleeve gastrectomy and 60% of 35 patients underwent roux-en-y gastric bypass <sup>(11)</sup>.

In another study done by Keidar, et al. <sup>(12)</sup> in June 2013 which was a randomized trial about roux-en-Y gastric bypass vs. sleeve gastrectomy for obese patients with type 2 diabetes in which there was significant decline in fasting HbA1c blood level at 3 and 12 months postoperatively in both surgeries with no procedure superior over the other regarding to fasting HbA1c level and glycemic control.

In November 2013 Nannipieri, et al. <sup>(13)</sup> performed a study which entailed the mechanisms of diabetes remission and role of Gut Hormones after roux-en-y gastric bypass and sleeve gastrectomy in which they detected significant decrease in fasting HbA1c blood level after 1 year postoperatively in both surgeries.

At the end we emphasize on the facts that adipose tissue that was recognized simply as an inert storage

organ is now considered as an endocrine organ and part of an innate immune system. Factors secreted from adipose tissue are very important contributors in regulation of metabolism and inflammatory responses.

And also we emphasize that it would be very important to follow up obese patients going for sleeve gastrectomies for a longer periods in order to verify the behavior of the inflammatory profile and its relation with change in BMI and also to study the long term effect of bariatric surgeries on these markers.

Limitations: low number of case, short period follow up.

### Conclusion

- Obesity is associated with chronic low grade inflammatory state with elevated inflammatory markers in obese patients
- Sleeve gastrectomy is effective solo bariatric operation with significant medium term effect on BMI and on Inflammatory profile
- Adiponectin level Rises Significantly after sleeve gastrectomy signifying better glycemic control and lesser tissue inflammation
- The rise in Adiponectinis correlated with the drop of BMI
- CRP, ESR and TLC are decreased significantly 4 months after sleeve gastrectomy denoting improvement in inflammatory status
- Statistically significant relation between drop in CRP, TLC and ESR1 and Drop in BMI could not be established denoting the involvement of other mechanisms in improvement of inflammatory profile rather than BMI drop only.

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### Declarations

- Financial support and sponsorship: Nil.
- Conflicts of interest: There are no conflicts of interest.
- Ethical approval : The study was approved by the Institutional Ethics Committee

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