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Gastric Banding Results in Weight Loss via Changes in Serum Levels of Obestatin and A-FABP in Obese Rats

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ABSTRACT : Background: *Obesity is characterized by an abnormal or excessive accumulation of fat in adipose tissues, which seriously damages health. Obesity occurs when energy intake exceeds energy expenditure, and may be caused by both genetic and environmental factors. Biochemical processes in the body determine satiety and hunger, and affect food selection, appetite, and eating frequency. The condition and energy storage activities of the adipose tissues are communicated to the central nervous system via leptin and other signals. The 1995 World Health Organization classification defines obesity as a body mass index of ≥ 30 kg/m^2 for men and ≥ 28.6 kg/m^2 for women. Obese individuals have low obestatin levels and high adipocyte fatty acid-binding protein (A-FABP) levels. This study used an in vivo rat model of obesity to investigate the effects of gastric banding (GB) on the serum levels of obestatin and A-FABP.*

METHODS: *Male Wistar rats (Rattus norvegicus) were divided into two groups: a control group who did not undergo GB, and a GB group who underwent esophagogastric banding. On days 0, 8, and 16, the rats were weighed and the serum levels of obestatin and A-FABP were measured using the ELISA method.* ***Results:*** *The serum obestatin level was not significantly different among the different time points in either the control or GB groups. The highest obestatin level was observed in the control group on day 8. The obestatin level was lower in the GB group than in the control group at each time point. There was an association between body weight and obestatin level, but the serum A-FABP level was not associated with either body weight or obestatin level. There was an association between a decrease in the A-FABP level and weight loss.*

CONCLUSIONS: *GB directly affected the serum obestatin level but not did not directly affect weight loss. In rats that underwent GB, a decreased serum A-FABP level occurred after weight loss.*

KEYWORDS: *Obesity, Gastric banding, Obestatin, A-FABP*

I. BACKGROUND

Obesity is defined as abnormal or excessive accumulation of fat in adipose tissues, which seriously damages health. Obesity occurs when energy intake exceeds energy expenditure, and may be caused by genetic or environmental factors. Biochemical processes in the body determine satiety and hunger, and influence food selection, appetite, and eating frequency. The condition and energy storage activities of the adipose tissues are communicated to the central nervous system via leptin and other signals. The 1995 World Health Organization classification defines obesity as a body mass index of ≥ 30 kg/m^2 for men and ≥ 28.6 kg/m^2 for women [1]. Obese individuals have low serum obestatin levels, low serum ghrelin/obestatin ratios, and high serum A-FABP levels. Obestatin induces satiety, resulting in decreased food intake and subsequent weight loss. However, the mechanisms underlying the metabolic regulation of obestatin are still unknown [2].A-FABP circulates in human plasma, and the level is increased in patients with metabolic syndrome [3]. Serum A-FABP levels are significantly higher in overweight/obese individuals than in normal-weight individuals. Serum levels of A-FABP are positively correlated with waist circumference, blood pressure, dyslipidemia, fasting insulin level, and the homoeostatic model assessment for insulin resistance index[3].As the changes described above are not sufficient to control body weight in obese individuals, other treatment such as bariatric surgery may be needed. Bariatric surgery carries a low risk of perioperative death, but can result in weight loss and significant reduction of the risk of obesity-related comorbidities.

Further research on bariatric surgery is therefore warranted [4,5]. The criteria for bariatric surgery include: BMI ≥ 40 kg/m^2 or BMI > 35 kg/m^2 with significant comorbidities, age between 16 and 65 years, being well informed and willing to accept the risks associated with surgery, failure of long-term non-surgical obesity treatment, psychological stability with realistic expectations, and having the motivation to modify lifestyle [4]. Bariatric surgical procedures have progressively improved over recent years, and such surgery is the only treatment option that results in significant long-term weight loss [6-8]. In obese individuals, bariatric surgery can result in reduction of the body weight by 20–40 kg and reduction of the body mass index by 10–15 kg/m^2. The mechanisms underlying the restoration of adipocyte function by alteration of the serum levels of obestatin and A-FABP after bariatric surgery remain unknown.

II. METHODS

Twelve white male Wistar rats *(Rattus norvegicus)* aged 2–2.5 months and weighing approximately 300 g were divided into two groups: a control group who did not undergo gastric banding (GB), and a GB group who underwent esophagogastric banding. In the GB group, the rats were fasted before surgery and were anesthetized by intraperitoneal injection of 10 mg/kg ketamine (1000 mg/10 mL, KTM-100; Indonesia **Address:** Kawasan Industri Manis Jl. Gatot Subroto Km 8, 5 Curug Tangerang Banten 15136, Indonesia **Phone:**+62 21 5918579 . Surgery was performed through a 2-cm midline abdominal incision. GB was performed by placing a Prolene mesh band (6 mm diameter, 2 mm wide) around the stomach below the fundus at the level of the esophagogastric junction to divide the stomach into upper and lower pouches. To prevent dislocation of the band, two sutures were placed in the anterior abdominal wall: one close to the curvature and the other close to the arch. The abdominal wall was closed with 3-0 Prolene sutures, and the wound was washed with 70% alcohol and covered with sterile gauze [9]. The food intake of the rats was measured morning at 8 am, and body weight was measured on days 0, 8, and 16. Blood samples were collected on days 0, 8, and 16 after a 2-h fast under ketamine anesthesia (10 mg/kg), and the serum levels of obestatin and A-FABP were measured by ELISA.

III. MEASUREMENT OF OBESTATIN LEVELS

Serum obestatin levels were measured using the rat obestatin ELISA kit (EK-031-90, Phoenix Lab; USA **Address:** 330 Beach Rd, Burlingame, CA 94010, United States **Phone:**(650) 558-8898. All the materials were prepared before ELISA was performed. The obestatin calibrator was dissolved in 1 mL of buffer solution, and the washing solution was prepared by mixing 500 mL of wash solution concentrate with de-ionized water 1 mL. The reagents were placed into wells at a temperature of 20–30°C. Each well was washed twice with 0.35 mL of washing solution that was left in the well for 30 s before being aspirated. Then, 50 μL of labelled antigen solution (0.01, 0.1, 1, 10, 100, or 1000 ng/mL) or a serum sample was added to each well, followed by 50 μL of mouse/rat obestatin antibody. The plates were sealed and incubated at 4°C for 18–20 h (overnight), after which each well was washed three times with 0.35 mL of washing solution. SA-HRP *(Streptavidin Horseradish peroxidase)* solution was added (100 μL/well) and the wells were incubated for 1 h at room temperature and then washed five times with washing buffer. TMB solution was added (100 μL/well) and the wells were incubated for 30 min at room temperature. Stop solution was added (100 μL/well) and the wells were incubated for 30 min at room temperature. Reactions were read using an ELISA reader at a wavelength of 450 nm.

Measurement of A-FABP levels

A-FABP levels were measured using the rat A-FABP ELISA kit (SK00030-03; Adipo Bioscience, USA Aviscera Bioscience, Inc 2348 Walsh Ave., Suite C Santa Clara, CA 95051 USA, Tel : (408)9820300. The materials and standards were stored at room temperature. The standards, blanks, and samples 50 mL of Buffer Concentrate were distilled water 450 mL to prepare 500 mL of Wash water and placed into the wells, and then incubated for 3 h at room temperature. Suspensions were washed four times with phosphate-buffered saline, and 200 μL of conjugates was added to each well. Serum samples were incubated for 2 h at room temperature. Suspensions were washed four times with washing buffer, and 200 μL of substrate solution was added to each well, followed by incubation for 30 min at room temperature. Stop solution was added to each well and samples were read after 30 min using an ELISA reader at a wavelength of 450 nm.

IV. RESULTS

Percentage body weight loss

Percentage changes in body weight are shown in Table 1. One-way analysis of variance showed a significant change in body weight in the GB group ($p < 0.05$). Tukey multiple comparisons showed no significant difference in weight loss between the control and GB groups on day 0, but showed a significant difference in weight between these two groups on day 16. In the GB group, body weight decreased by 27.87%

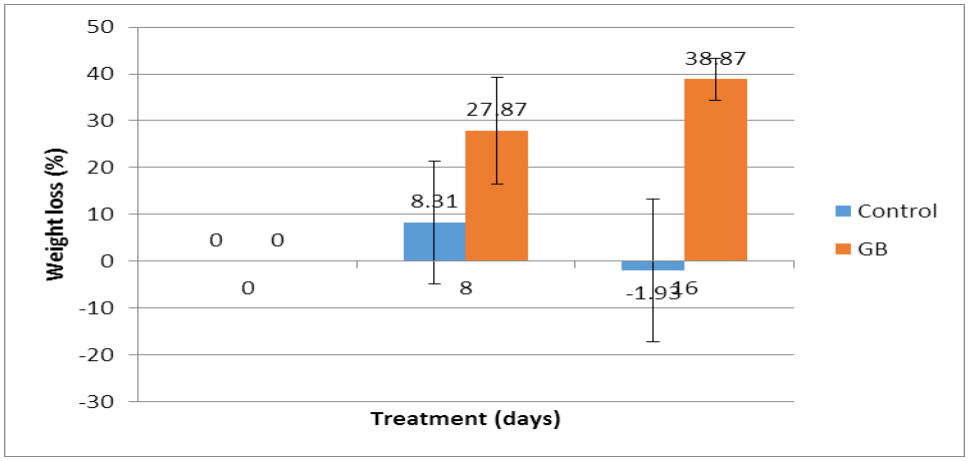
on day 8 and 38.87% on day 16, which were significant differences compared with the preoperative weight (both $p = 0.0001$; Figure 1).

Table 1 Percentage weight change in the control and GB groups

Group	*n*	Weight loss (%)				*P*
		Mean	SD	Min	Max	
Control, day 0	7	0.00	0.00	0.00	00:00	
Control, day 8	7	8.31	13.07	–8.18	34.59	0.240
Control, day 16	7	–1.93	15.22	–16.67	27.36	
GB, day 0	7	0.00[a]	0.00	0.00	00:00	
GB, day 8	7	27.87[b]	11.50	11.47	39.50	0.000*
GB, day 16	7	38.87[c]	4.54	33.01	45.11	

*$p < 0.05$.
[a,b,c]Different superscripts indicate the mean differences between groups.

Figure 1



Obestatin levels

The serum obestatin levels were normally distributed in 2 groups (all $p > 0.05$; Appendix). Levene tests of homogeneity of variance showed that the groups were homogeneous ($p > 0.05$). Because of the normally distributed data and homogeneity of groups, one-way analysis of variance was performed, which showed significant differences between groups ($p < 0.05$; Table 2). Tukey multiple comparisons showed that the serum obestatin levels were significantly different between the control and GB groups.

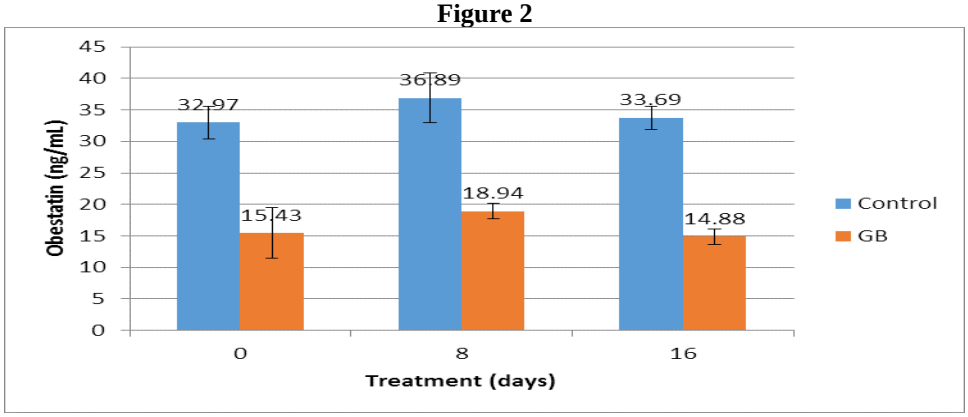
Figure 2 shows that the serum obestatin levels were not significantly different among days 0, 8, and 16 in either the control or GB groups. The highest serum obestatin level was observed in the control group on day 8. Serum obestatin levels were lower in the GB group than in the control group at each time point.

Table 2 Serum obestatin levels in the control and GB groups

Group	*n*	Obestatin (ng/mL)				One-way ANOVA
		Mean	SD	Min	Max	
Control, day 0	7	32.97[a]	2:56	29.43	37.50	
Control, day 8	7	36.89[a]	3.93	30.39	41.63	
Control, day 16	7	33.69[a]	1.82	31.54	35.93	F = 95.74
GB, day 0	7	15:43[b]	4.04	12.44	24.13	$p = 0.000$*
GB, day 8	7	18.94[b]	1.21	17.29	20.87	
GB, day 16	7	14.88[b]	1.27	13.10	16.42	

*$p < 0.05$
[a,b]Different superscripts indicate inter-group differences based on the Tukey multiple comparisons test.

Figure 2



A-FABP levels

The serum A-FABP levels were normally distributed in each group (all $p > 0.05$; Appendix). Levene's test of homogeneity of variance showed that the groups were inhomogeneous ($p < 0.05$). Because of the normally distributed data and inhomogeneity of groups, data were analyzed using the Brown-Forsythe test, which showed significant differences between groups ($p < 0.05$; Table 3). Further analysis using the independent *t*-test found no significant differences in serum A-FABP levels in the control group among days 0, 8, and 16. In the GB group, there was no significant difference in the serum A-FABP level between days 0 and 8, but there was a significant difference between days 0 and 16.

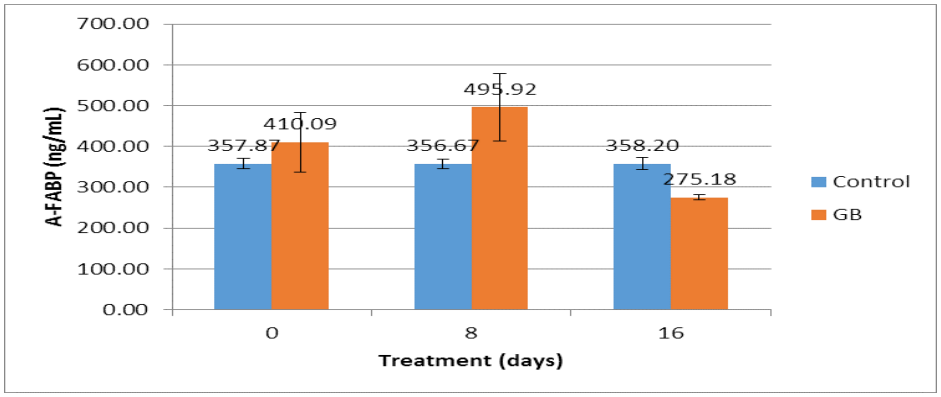
Figure 3 shows that the serum A-FABP level was higher in the GB group than in the control group on days 0 and 8, after which the level decreased in the GB group, and was lower in the GB group than in the control group on day 16.

Table 3 Serum obestatin levels in the control and GB groups

Group	n	A-FABP (ng/mL)				Brown-Forsythe Statistic
		Mean	SD	Min	Max	
Control, day 0	7	357.87[a]	12:03	345.15	380.87	Statistic = 14:41 $p = 0.000$*
Control, day 8	7	356.67[a]	11.81	344.64	379.34	
Control, day 16	7	358.20[a]	14:49	344.39	381.63	
GB, day 0	7	410.09[ab]	73.87	370.66	574.49	
GB, day 8	7	495.92[b]	83.08	360.46	596.43	
GB, day 16	7	275.18[c]	6167	265.31	282.40	

*$p < 0.05$.
[a,b,c]Different superscripts indicate inter-group differences based on the independent t-test.

Figure 3



V. DISCUSSION

The objective of the present study was to evaluate the effects of GB on body weight, serum obestatin level, and serum A-FABP level. The effects of GB were evaluated on days 0, 8, and 16 after surgery. Evaluation of success was based on the age of the rat. A rat age of 3 months and 8 days can be considered to be equivalent to a human age of 13.5 years, and a rat age of 3 months and 16 days can be considered to be equivalent to a human age of 14 years [10]. Our selection of postoperative interval for evaluation was based on the fact that humans have significant weight loss during the first 2 years after GB surgery. An 8-day postoperative period in rats was considered to be equivalent to a 2-year postoperative period in humans, and a 16-day postoperative period in rats was considered to be equivalent to an 8-year postoperative period in humans. Rats were evaluated from 8 days after surgery as earlier postoperative weight loss could be caused by loss of nitrogen [9,11,12]. Previous studies reported associations between body weight and serum levels of ghrelin, obestatin, adiponectin, and A-FABP; and the ghrelin/obestatin ratio. Bariatric surgical procedures have progressively improved over recent years, and such surgery is the only treatment option that results in significant long-term weight loss [6-8]. A randomized trial of laparoscopic gastric banding involving 100 patients showed a 58.9% weight loss over 3 years [13].

In this study, there was a significantly greater percentage of weight loss in the GB group than in the control group, with a loss of 27.87% and 38.87% on days 8 and 16, respectively. A similar study by Monteiro et al. reported that GB resulted in weight loss in obese Zucker rats [14]. Weight loss after bariatric surgery depends on the surgical technique used [15-18], with the average weight loss after GB being about 28% [15,16]. The mechanisms by which GB contributes to weight loss remain unclear. Recently reported data suggest that adipocyte function and neural and hormonal changes may contribute to weight loss after GB [19,20]. Bariatric surgery may result in reduced lipogenesis because the reduced food intake results in reduced total energy expenditure [21]. Shak et al. reported that GB resulted in a significant weight loss at 6–12 months in 24 patients [22], and Colles et al. reported that GB resulted in a significant weight loss at 4–12 months in 85 patients [23]. Madeleine et al. reported that laparoscopic GB significantly reduced body weight, waist circumference, plasma free fatty acid level, subcutaneous abdominal fat, and visceral fat in obese non-diabetic women after 12 weeks, associated with a significant improvement in insulin sensitivity [24].Obestatin is a peptide composed of 23 amino acids, which is derived from the same precursor protein as ghrelin. In this study, there were no significant differences in serum obestatin levels among the different time points in either the control or GB groups. The highest serum obestatin level was observed in the control group on day 8. Serum obestatin levels were lower in the GB group than in the control group. The difference in serum obestatin level between the control and GB groups on day 0 is thought to be due to the administration of anesthesia to the GB group. Ataka et al. reported that intravenous anesthesia decreases the serum obestatin level after 30–90 min [25]. Our findings that there were no significant differences in the serum obestatin level among days 0, 8, and 16 in either the control or GB groups indicate that GB surgery did not affect the serum obestatin level.

Zizzari et al. reported that obestatin inhibited the effects of ghrelin on appetite [2]. Under underweight fasting conditions, the levels of ghrelin increased whereas those of obestatin decreased.The circulating obestatin level is related to the circulating ghrelin level only in obese individuals. High serum levels of obestatin induce satiety, resulting in reduced food intake and weight loss. However, the metabolic regulation of obestatin remains unclear [2]. The role of obestatin in the regulation of hormone secretion has been investigated in *in vivo* and *in vitro* rat models (*Rattus norvegicus*). *In vivo*, obestatin did not stimulate secretion of growth hormone or corticosterone. These results suggest that obestatin protects against improper secretion of these hormones [2].
Obestatin has been reported to inhibit jejunal activity and affect gastric emptying [26], and stimulate pancreatic beta glands [27]. The effects of obestatin and ghrelin on the stomach and hypothalamus play important roles in the regulation of energy homeostasis. Both peptides are derived from preproghrelin, and they have opposite effects on food intake. In underweight individuals, serum ghrelin levels are high, serum obestatin levels are low, and the ghrelin/obestatin ratio is high [28].

The serum A-FABP level was higher in the GB group than in the control group on day 8, but was lower in the GB group than in the control group on day 16. These results indicate that the serum A-FABP level was not associated with GB surgery. There serum A-FABP level was not significantly associated with the serum obestatin level or weight loss, but our analysis showed that weight loss had a direct effect on A-FABP secretion. There was no significant association between GB and the serum A-FABP level, indicating that the A-FABP level alters metabolism to induce weight loss. The increase in the serum A-FABP level after day 8 could be explained by the mobilization of fat secondary to hormone-sensitive lipase levels, leading to increased lipolysis. The reduced body fat on day 16 secondary to decreased food intake resulted in decreased lipolysis. The serum A-FABP level is a major indicator of lipolysis, and cytoplasmic A-FABP increases the hydrolytic activity of

hormone-sensitive lipase [29]. Mobilization of stored fat is mediated by lipolytic enzymes. The results of recent studies suggest that adipose triglyceride influences triglyceride hydrolysis, leading to lipolytic catabolism of fats in adipose tissues. Hormone-sensitive lipase hydrolyzes triglycerides when diglycerides are predominant [30]. The serum A-FABP level was reported to be positively correlated with waist circumference, blood pressure, dyslipidemia, fasting insulin, and the homoeostatic model assessment for insulin resistance index [3]. A-FABP circulates in human plasma and is increased in individuals with metabolic syndrome [3]. Serum A-FABP levels are significantly higher in overweight/obese individuals than in normal-weight individuals.

VI. CONCLUSIONS

GB decreases the serum obestatin level but does not affect weight loss. However, the serum A-FABP level affects weight loss after GB.

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List of Abbreviations

A-FABPadipocyte fatty acid-binding protein
GB Gastric banding
SA-HRPStreptavidin Horseradish Peroxidase

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

KSO contributed to study conception and design, acquisition of data, and analysis and interpretation of data. HS contributed to drafting and revisions of the manuscript. MRI contributed to the writing of the manuscript. KUN contributed to study design and statistical analysis. ATJ contributed to study design and coordination of research. DS contributed to drafting and revisions of the manuscript. AS contributed to development of the surgical procedure. All authors read and approved the final manuscript.

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