

Study of Association between Obesity, Gastritis and Serum Adiponectin Level

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Abstract

Background: Increase prevalence of obesity is linked with increase the threat of various diseases such as cardiovascular disease, diabetes, cancer, and many gastrointestinal complications of obesity. **Objective:** Study the relationship between obesity, endoscopic gastritis, and serum Adiponectin level in obese persons.

Methods: This study was done on sixty-five patients diagnosed with chronic gastritis by upper endoscopy according to Sydney system, categorized into three groups according to their body mass index, group A: Body Mass Index less than 25, group B: Body Mass Index from 25 to 30 and group C: Body Mass Index more than 30. Careful clinical examination, Complete Blood Count, lipid profile, Helicobacter pylori antigen in the stool and serum Adiponectin by Enzyme Linked Immunosorbent Assay technique were done. **Results:** Body Mass Index showed statistically significant difference between all studied groups. 49.2% of our study patients had erosive gastritis, 29.9% had hemorrhagic gastritis and 21.5% had atrophic gastritis. Serum Adiponectin showed statistically significant difference between all studied groups. According to Receiver operating characteristic curve of serum Adiponectin, a cut off level lower than 7 microgram/ml is diagnostic of endoscopic gastritis in obese persons with Area under Curve about 0.841, sensitivity of 86%, specificity of 73%, negative predictive value of 81%, and positive predictive value of 79% and accuracy of 80%. Lower Adiponectin level was found in erosive

gastritis compared to hemorrhagic and atrophic gastritis. Erosive gastritis had the highest Body Mass Index followed by hemorrhagic and the least was atrophic gastritis group (p-value < 0.05).

Conclusion: Obese subjects have a lower serum Adiponectin level which may be a risk factor of incidence of gastritis and may affect its types.

Keywords: Endoscopic gastritis; Upper endoscopy; Obesity; Serum Adiponectin.

Introduction

Continuous increase incidence of obesity, markedly in developed countries, is a serious worry as it raises the threat of many diseases such as cardiovascular disease, diabetes, and cancer. ⁽¹⁾ The gastrointestinal tract (GIT) play a role in obesity through its contributions to satiation, production of gut hormones that influence appetite (such as ghrelin, cholecystokinin, and peptide YY), incretins (e.g., glucagon-like peptide-1) that influence postprandial glycemia, absorption of nutrients which determine the positive energy balance that results in obesity, changes in bile acids and the microbiome, and the metabolic products of microbial digestion of short-chain fatty acids which change some metabolic factors associated with obesity. ⁽²⁻⁴⁾

Obesity is linked with increased risks of several gastrointestinal diseases as gastro esophageal reflux disease (GERD), erosive esophagitis, Barrett's esophagus, esophageal adenocarcinoma, erosive gastritis, gastric cancer, diarrhea, colonic diverticular disease, polyps, nonalcoholic fatty liver disease (NAFLD), cirrhosis, hepatocellular carcinoma, gallstones, acute pancreatitis, and pancreatic cancer. ⁽⁵⁾ Endoscopic gastritis indicates inflammatory change in the gastric mucosa as color and/or structure that causes subsequent apoptosis and gland damage, leading to regeneration, fibrosis, or metaplasia. Endoscopic gastritis is classified into seven types: superficial erythematous, hemorrhagic, erosive, verrucous, atrophic, metaplastic and rugal hypertrophied gastritis. Erosive gastritis, being the most common type. There are different

classifications of gastritis; the most popular is Updated Sydney System ⁽⁶⁾.

Chronic gastritis may be caused by infectious or noninfectious conditions. Some bioactive molecules secreted from excess visceral fat dysregulation are proposed to promote the progression of metabolic disorders in obese persons ⁽⁷⁾. Adiponectin is one of these molecules. Circulating Adiponectin was found to have anti-atherogenic, anti-inflammatory, and anti-diabetic effects ⁽⁸⁾. Low serum Adiponectin levels in obese persons, was hypothesized that may cause metabolic disorders associated with obesity ⁽⁹⁾. The anti-inflammatory action of Adiponectin may have a protecting role in many gastrointestinal diseases, such as colitis, liver fibrosis, gallstone, and disease pancreatitis ⁽¹⁰⁻¹³⁾.

This work aimed to study the association between obesity, endoscopic gastritis, and serum Adiponectin levels in obese persons.

Patients and methods

This is a prospective cohort study was done from September 2019 to September 2020. The study had been approved by the ethical committee, Tanta Faculty of Medicine with approval code {M.S. 19.2.32946}. Sixty-five chronic gastritis patients diagnosed by upper endoscopy, recruited prospectively among individuals undergoing upper endoscopy selected from Out-patient's Clinic and In-patient's Wards of Gastroenterology and Hepatology Unit, Internal Medicine Department, Tanta University Hospitals.

Inclusion criteria:

The study included adult patients with definitive diagnosis of gastritis confirmed by clinical, endoscopic, and histological examination in doubtful cases. They were classed into three groups according to their Body Mass Index (BMI).

- Group (A): BMI less than 25
- Group (B): BMI from 25 to 30
- Group (C): BMI more than 30

Exclusion criteria:

- Patients with active upper GIT bleeding.
- Patients with NSAID induced gastritis.
- Patients with H. Pylori proven gastritis.
- Patients underwent gastric surgery.

All patients subjected to:

1. Through history taking (with special emphasis on abdominal pain, persistent vomiting, anemia, history of recent GIT bleeding, detailed drug history specially NSAID, history of H. pylori treatment and history of gastric surgery).
2. Full clinical examination with special stress on heart rate (HR), systolic blood pressure (SBP) and diastolic blood pressure (DBP).
3. BMI calculation via equation $\text{weight}/(\text{height in meter})^2$
4. Upper endoscopy with gastritis classification according to Sydney classification flat, erosive, elevated, atrophic, verrucous, metaplastic, hypertrophied, hemorrhagic and biliary reflux gastritis ⁽⁶⁾. Upper endoscopy was done using high definition videoscope EPK i. scan 5000 (Pentax medical. Japan). (All

patients were instructed to fast for 8 hours, procedural sedation using midazolam with monitoring of vital signs throughout the procedure)

5. Biopsy and histopathological examination of gastric samples in doubtful cases.
6. Complete blood count (CBC)
7. Serum triglyceride and cholesterol levels
8. H. pylori antigen in the stool
9. Serum Adiponectin using ELISA technique

Statistical methodology:

The data was coded and entered using the statistical package SPSS version 26 (IBM®, USA). All quantitative data were expressed as mean, and standard deviation values. Chi Square (Fisher's exact test) was used to test statistical differences between studied groups. Comparison of two groups for all continuous normally distributed data was done by Independent t-test. A p-value ≤ 0.05 was considered statistically significant. The Spearman correlation coefficient test (r) was used to test a positive or negative relationship between two variables and correlations were done to test linear relations between variables. Receiver Operating Characteristic (ROC) curve analysis was used to determine sensitivity, specificity, positive and negative predictive values together with relative risk.

Results

The patient age ranges from 20-40 year with mean 31.13 ± 6.02 , 27-62 year with mean 43.27 ± 11.24 and 26-29 year with mean 27.87 ± 1.06 in group A, B and C respectively with statistically significant difference

between group A and B ($p=0.001$), significant difference between group A and C ($p=0.001$) and no significant difference between group B and C ($p=0.511$). BMI ranges from 19-24.5 (kg/m^2) with mean 22.41 ± 1.49 (kg/m^2), 26-29 (kg/m^2) with mean 27.87 ± 1.06 (kg/m^2) and 30-44 (kg/m^2) with mean 34.06 ± 3.73 (kg/m^2) in group A, B and C respectively with statistically significant difference between all studied groups ($p=0.001$) as shown in table (1).

All patients undergo upper endoscopy due to persistent upper abdominal pain, persistent vomiting, and anemia. Group A (5 anemia, 6 abdominal pain and 4 vomiting), group B (7 anemia, 6 abdominal pain and 2 vomiting) and group C (10 anemia, 18 abdominal pain and 7 vomiting) with no statistically significant difference between all groups as regards indication of upper endoscopy ($P=0.710$), (Table 2).

Forty-nine point two percentages of our patients had erosive gastritis, 29.9 % had hemorrhagic gastritis and 21.5 % had atrophic gastritis with a statistically significant difference between all studied groups as regards endoscopic gross pictures (table 3). Serum Adiponectin level ranges from 3-17 with mean 9.27 ± 4.13 , 4.48-17 with mean 10.16 ± 4.42 and 2-11.28 with mean 5.11 ± 2.10 in group A, B and C respectively with statistically significant difference between all studied groups ($p=0.001^*$) as shown in table (4). There was a statistically non-significant difference

between group A and B ($p=0.455$) with statistically significant difference between group A and C ($p=0.001$), and between group B and C ($p=0.001$).

According to Roc curve of serum Adiponectin level demonstrated in figure (1) and table (5), we recommend cutoff level lower than 7 microgram/ml for diagnosis endoscopic gastritis in obese persons with AUC about 0.841, sensitivity of 86%, specificity of 73%, negative predictive value of 81%, positive predictive value of 79% and accuracy of 80% with a statistically significant difference of serum Adiponectin between all studied patients. As regards endoscopic gross pictures, Adiponectin level was lower in erosive gastritis than hemorrhagic and atrophic ($p=0.004$).

The most type of gastritis had the highest mean and range of BMI was erosive gastritis followed by hemorrhagic and the least was atrophic gastritis, with a statistically significant difference between studied groups ($p=0.004$). There was statistically non-significant difference between patients with erosive and hemorrhagic gastritis as regard MBI with a statistically significant difference between erosive and atrophic gastritis patient ($p=0.001$). There was a statistically significant difference between hemorrhagic and atrophic gastritis ($p=0.073$) as demonstrated in table (6).

There was significant inverse correlation between BMI and S. Adiponectin as shown in figure (2).

Table (1): Comparison between all studied groups as regard age and BMI.

	Group A (N= 15)	Group B (N= 15)	Group C (N= 35)
Age (yrs)			
Mean ± SD	31.13 ± 6.02	43.27 ± 11.24	45.11 ± 9.07
P		0.001*	
P1		P2	P3
0.001*		0.001*	0.511
BMI (kg/m²)			
Mean ± SD	22.41 ± 1.49	27.87 ± 1.06	34.06 ± 3.73
P		0.001*	
P1		P2	P3
0.001*		0.001*	0.001*

* Significant < 0.05 P1: Group A & Group B, P2: Group A & Group C P3: Group B & Group C

BMI: Body Mass Index.

SD: standard deviation.

p-value: level of significance.

Table (2): Comparison between all studied groups as regard as indication of upper endoscopy.

Indication		Group A (N= 15)	Group B (N= 15)	Group C (N= 35)	Total
Anemia	N	5	7	10	22
	%	33.3%	46.7%	28.6%	33.8%
Abdominal pain	N	6	6	18	30
	%	40.0%	40.0%	51.4%	46.2%
Vomiting	N	4	2	7	13
	%	26.7%	13.3%	20.0%	20.0%
Total	N	15	15	35	65
	%	100.0%	100.0%	100.0%	100.0%
Chi-square	X2			2.141	
	P-value			0.710	

* Significant < 0.05 p-value: level of significance.

Table (3): Comparison between all studied groups as regard as endoscopic gross pictures.

Upper Endoscopy		Group A (N= 15)	Group B (N= 15)	Group C (N= 35)	Total
Erosive	N	5	3	24	32
	%	33.3%	20.0%	68.6%	49.2%
Hemorrhagic	N	5	4	10	19
	%	33.3%	26.7%	28.6%	29.2%
Atrophic	N	5	8	1	14
	%	33.3%	53.3%	2.9%	21.5%
Total	N	15	15	35	65
	%	100.0%	100.0%	100.0%	100.0%
Chi-square	X2			19.838	
	P-value			0.001*	

* Significant < 0.05 p-value: level of significance.

Table (4): Comparison between all studied groups as regard as serum Adiponectin.

Serum Adiponectin ($\mu\text{g/ml}$)	Group A	Group B	Group C
Mean \pm SD	9.27 \pm 4.13	10.16 \pm 4.42	5.11 \pm 2.10
P	0.001*		
Group A & Group B	Group A & Group C	Group B & Group C	
0.455	0.001*	0.001*	

* Significant < 0.05

p-value: level of significance.

SD: standard deviation.

Table (5): Serum Adiponectin Sensitivity and Specificity

	Cut off	AUC	Sensitivity	Specificity	PPV	NPV	Accuracy
S. Adiponectin	7	0.841	86	73	79	81	80

* Significant < 0.05

AUC: area under curve.

Ppv : positive predictive value.

Npv: negative predictive value.

Table (6): Correlation between BMI, S. Adiponectin and types of endoscopic gastritis.

	Erosive	Hemorrhagic	Atrophic
	Serum Adiponectin ($\mu\text{g/ml}$)		
Mean \pm SD	4.88 \pm 1.69	6.84 \pm 2.62	13.18 \pm 3.06
P	0.001*	0.005*	0.001*
	BMI (kg/m^2)		
Mean \pm SD	31.92 \pm 5.58	29.44 \pm 5.62	26.09 \pm 3.48
P	0.106	0.073	0.001*

* Significant < 0.05

BMI: Body Mass Index.

p-value: level of significance.

SD: standard deviation

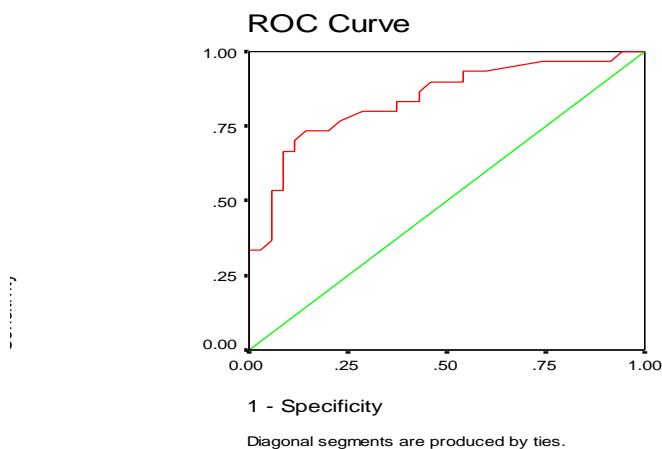


Figure (1): ROC Curve of serum Adiponectin ($\mu\text{g/ml}$)

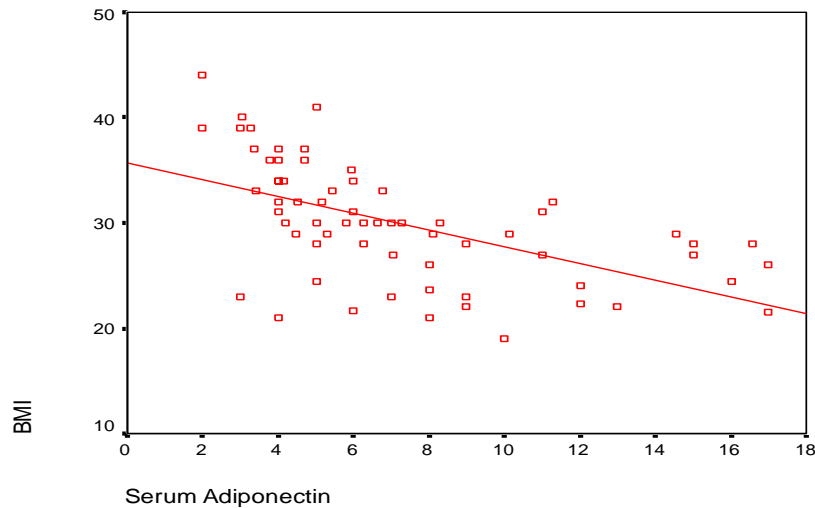


Figure (2): Correlation between serum Adiponectin and BMI, there was significant inverse correlation between BMI (kg/m²) and S. Adiponectin (mg/ml)

Discussion

Gastritis is described as an inflammatory process in the gastric mucosa and submucosa in response to injury, which may be acute or chronic ⁽¹⁴⁾. Adiponectin represents approximately 0.05% of total plasma proteins present in the circulation, ranging from 3 to 30 $\mu\text{g/mL}$ ⁽¹⁵⁾. Several searches have studied the relationship between Adiponectin levels and pro-inflammatory markers in various populations ⁽¹⁶⁾. Previous studies showed that Adiponectin may have a protecting role in various gastrointestinal diseases via its anti-inflammatory properties ^(17, 18).

Hypoadiponectinemia has been linked to a variety of gut diseases involving GERD, gastritis, inflammatory bowel disease (IBD), pancreatitis non-alcoholic fatty liver disease (NAFLD), and malignancy. ⁽¹⁹⁾ Adiponectin is an adipocyte-secreted hormone consisting of 244 amino

acid residues and the product of the apM1 gene, which is specifically expressed in human adipocytes. ⁽²⁰⁾ Our results showed that Adiponectin level was below the reference value in all patients. Moreover, lower Adiponectin levels were found in patients with erosive gastritis compared to patients with hemorrhagic or atrophic gastritis. Interestingly, we found that the most type of gastritis with the highest BMI is erosive gastritis followed by hemorrhagic and the least is atrophic gastritis. Previous reports revealed an increased incidence of erosive gastritis with increased BMI. ⁽²¹⁾

Different mechanisms have been considered to explain obesity-related gastritis. First, obesity is associated with physical changes such as increased intra-abdominal pressure and impaired gastric emptying leading to increased rates of GERD and could also play causal roles in

erosive gastritis in obese subjects. ⁽²²⁾ Second, food habits such as excess food intake, and food restriction might be associated with increased gastrointestinal symptoms and gastric diseases in obese persons. ⁽²³⁾ A third mechanism is hormonal factors ghrelin and leptin dysregulation. ⁽²⁴⁾ ⁽²⁵⁾ Adiponectin might have gastric protective effect be through decrease neutrophil infiltration, reduction in gastric motility, relaxation of circular muscles, flattening of the folds and diminish the volume of the gastric irritants on the rugal crest. ⁽²⁶⁾

A study done on 2,400 participants showed that, BMI was significantly higher in gastritis-positive participants compared to gastritis-negative participants. Also, serum Adiponectin levels were significantly lower in gastritis-positive participants compared to gastritis-negative participants ⁽²⁷⁾. Multivariate logistic regression analysis revealed that lower serum Adiponectin level (OR 0.96; 95% CI 0.93-0.99), smoking (OR 0.50; 95% CI 0.30-0.80), higher blood pressure (OR 1.02; 95% CI 1.01-1.03), and duodenitis (OR 1.8; 95% CI 1.00-3.09) were significantly associated with endoscopic erosive gastritis. Another study found that, Adiponectin level was lesser in erosive gastritis than hemorrhagic and atrophic gastritis. ⁽²⁸⁾ We had excluded patients with H. pylori proven gastritis in our study. However, a study conducted in Iraq reported that Adiponectin was significantly lower ($p=0.001$) in gastritis patients whether infected (8.783 ± 0.968) with H. pylori or not (8.278 ± 0.838) compared to control group (9.119 ± 0.1593). They concluded that Adiponectin had an important role in gastritis especially with H. pylori infection.

⁽²⁹⁾ A similar finding concluded that hypoadiponectinemia might increase risk H. pylori infection. ⁽³⁰⁾

Our study revealed an inverse association between serum Adiponectin level and BMI. This agrees with this study that demonstrates a negative relation between plasma Adiponectin level and BMI, body weight, and waist circumference. ⁽³¹⁾ A probable explanation is that a feedback inhibition of Adiponectin production may occur due to increase production of other adipocytokines as TNF which decrease adipocyte expression and secretion of Adiponectin.

Conclusion

Based on our results, lower serum Adiponectin level in obese subjects may be considered as a risk factor of gastritis and may have an important role in the type of gastritis. Further studies should be conducted including a larger number of patients.

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