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STUDY TO DETERMINE THE RELATION OF ADIPONECTIN LEVELS WITH GASTROESOPHAGEAL REFLUX DISEASE

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Abstract:
Purpose: *Gastroesophageal reflux disease (GERD) has become the most common gastrointestinal disorder in clinics. Gastric acidity due to impaired gastroesophageal junction will affect the esophageal epithelium.*
Aim of the study: *To investigate the relationship between circulating adiponectin and GERD levels in patients undergoing upper endoscopy.*
Place and Duration: *In the Gastroenterology Department of Services Hospital Lahore for one year duration from March 2019 to March 2020.*
Patients and methods: *Data from 40 patients with GERD and 30 healthy controls and endoscopy were collected. Calculated weight, height, waist circumference, body mass index. A lipid profile and adiponectin were done.*
Results: *The results showed a strong negative correlation between adiponectin and age, height, weight, BMI, waist circumference, waist to hip ratio, cholesterol, triglycerides and LDL. There is only a positive correlation between adiponectin and HDL and LDL / HDL (+0,214 and +0,014 respectively).*
Conclusions: *This study emphasizes that impaired circulation of intestinal adiponectin hormones plays a role in the pathogenesis of GERD and may be susceptible to Barrett's esophagus.*
Key words: *adiponectin, GERD, lipids.*

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INTRODUCTION:

Gastroesophageal reflux disease (GERD) has increased significantly in recent years and is known to be a risk factor for Barrett's esophagus and esophageal adenocarcinoma. Increase the risk 30-40 times. Many mechanisms contribute to the pathogenesis of GERD and affect the function of the gastroesophageal junction, such as obesity and increased intra-abdominal pressure. Adiponectin is another circulating metabolic factor associated with obesity. Adiponectin is a specific protein or peptide produced by adipose tissue and visceral adipocyte, and its level is inversely proportional to obesity and lower in men than in women. It had many functions, such as regulating inflammation and inhibiting carcinogenesis. Adiponectin causes high alpha TNF and weight loss in animals through the NP-1 accelerator activator. It also reduces mitochondrial oxidative damage and oxygen-glucose deprivation caused by apoptosis via the JAK2 / STAT3 pathway. It binds to and mediates the specific AdipoR1 and AdipoR2 receptors in the esophagus mucosa. The relationship between adiponectin and GERD or Barrett's esophagus is conflicting. Low plasma adiponectin levels in patients with dyspepsia and heartburn are associated with an increased risk of Barrett's esophagus in patients undergoing upper endoscopy to confirm the diagnosis. On the other hand, no such relationship was found in another pilot study. Therefore, it is assumed that reducing adiponectin levels is a risk factor for GERD. Therefore, it is a case-control study to investigate the relationship between circulating adiponectin levels and GERD levels in patients undergoing upper endoscopy.

PATIENTS AND METHODS:

The study is a planned case study held in the Gastroenterology Department of Services Hospital Lahore for one year duration from March 2019 to March 2020. Patient's informed consent was obtained. The Scientific and Ethical Committee approved and registered the study. Written informed consent was obtained from patients and normal blood donors.

Admission Criteria

Patients suffering from indigestion, discomfort in the upper abdomen, acid deficiency at least once a month for the last 6 months, more than 3 days of heartburn per week as defined by Montreal and gastroesophageal reflux disease classification were included. In a research.

Exclusion criteria

Patients with previous gastric surgery, peptic ulcer, and gastric cancer, previously damaged H. pylori, esophageal varices and antacids, H2 blockers, proton pump inhibitors and nonsteroidal anti-inflammatory drugs were excluded from the study. Data from 40 GERD patients and 30 healthy controls, including demographic information such as age, gender and residence status, were collected using a questionnaire.

Endoscopy

All patients underwent endoscopic upper gastrointestinal examination with gastroscopy: GIF-H260; Olympus, Tokyo, Japan and screen; Olympus OEV-261H liquid crystal monitor; Olympus, Tokyo, Japan. Endoscopic examination was performed by well-trained gastroenterologists with at least 5 years of endoscopic experience. The gastroesophageal junction was defined as the flat-column junction and the proximal gastric fold margin. The presence or absence of GERD was determined by the endoscopist according to criteria.

Anthropometric measurements

Weight, height, waist circumference, body mass index is calculated by dividing the weight in kilograms by the square of the height in meters. Waist circumference was measured in centimeters (cm) at the end of normal exhalation between the lowest rib and the iliac peak, with the tape measure being one side to ensure it is horizontal at the top. back and front of the participant.

Blood collection and analysis

Blood was drawn from two groups, and 5 ml of venous blood was aspirated from the appropriate vein. Blood samples were divided into 2 parts, one for lipid profile analysis and the other for the analysis of other parameters. Each serum sample was analyzed for lipid profile (cholesterol, triglycerides, HDLP, LDLP (human-Germany), adiponectin (Shanghai Biological Technology-China), C-reactive protein (human-Germany) and H pylori (Eco China test).

Statistics analysis

Statistical analysis was performed using MiniTab version 3.0. Data analysis was performed using a chi-square test for frequencies, t test for means and standard error. The correlation coefficient was used to find the correlation between Pearson's correlation and various parameters. A p value below 0.05 was considered statistically significant.

RESULTS:

The total number of working groups was 60, of which 40 were GERD and the rest were healthy controlled. The age of the patient group was 42.25 ± 2.41, and the control group was not significant with 38.27 ± 3.78 ($p = 0.387$). In the group of patients, 22 men (55%) constituted more than 18 women (45%), which was a significant difference from the control group ($p = 0.028$). BMI is significantly higher in patients with GERD ($p = 0.011$) than in the control group. In this study, adiponectin and a non-TG lipid

profile were significantly higher in the patient group (Table 1). Most patients with GERD were positive for C-reactive protein and H pylori (p = 0.000).

Table 1 Demographic difference of various parameters between patients with gastroesophageal reflux disease (GERD) and control group

Parameters	GERD patients N=40	Control Group N=20	p-value
	X ± SEM	X ± SEM	
Age (year)	42.25 ± 2.41	38.27 ± 3.78	
Range	(25-62)	(25-68)	0.387
Male %	22 (55%)	14 (70%)	
Female %	18 (45%)	06 (30%)	0.028
Height (cm)	165.55 ± 0.0170	172.30 ± 0.0265	0.048
Weight (Kg)	76.05 ± 2.92	82.23 ± 5.51	0.338
BMI Kg/m^2	31.78 ± 1.04	27.41 ± 1.13	0.011
Waist circumference (cm)	120.10 ± 3.02	103.90 ± 5.24	0.005
Waist to Hip Ratio	1.036 ± 2.30	1.114 ± 3.43	0.984
Adiponectin Mg/ml	15.240 ± 1.462	9.119 ± 2.315	0.023
Cholesterol Mg/ml	287.0 ± 26.3	104.7 ± 52.1	0
Triglyceride Mg/ml	140.0 ± 25.3	137.0 ± 75.8	0.962
HDL Mg/ml	35.80 ± 3.40	50.42 ± 4.25	0.012
LDL Mg/ml	212.1 ± 25.9	155.1 ± 39.3	0.219
LDL/H|DL	4.5 ± 0.0614	2.688 ± 0.283	0
C-reactive protein +	20 (50%)	1 (2.5%)	0
H. pylori +	26 (65%)	1 (2.5%)	0

The results showed a strong negative correlation between adiponectin and age, height, weight, BMI, waist circumference, waist to hip ratio, cholesterol, triglycerides and LDL. As shown in Table 2, there is only a positive correlation between adiponectin and HDL and LDL / HDL (+0,214 and +0,014, respectively).

Table 2 Pearson correlation analysis of adiponectin with different parameters in GERD patients

Parameters	Patients with GERD N=40 r	p-value
Age (year)	-0.284	0.225
Height (cm)	-0.311	0.182
Weight (Kg)	-0.325	0.162
BMI Kg/m^2	-0.145	0.541
Waist (cm)	-0.396	0.084
Hip Waist Ratio	-0.079	0.742
Cholesterol Mg/dl	-0.215	0.362
Triglyceride Mg/dl	-0.118	0.621
HDL Mg/dl	0.214	0.365
LDL Mg/dl	-0.009	0.969
LDL/HDL	0.014	0.953

DISCUSSION:

GERD pathogenesis is complex and multifactorial. Obesity and increased body mass index are one of the predisposing factors. This study found a significant increase in BMI, waist circumference and waist / hip ratio in patients with GERD compared with a control group that agrees with other studies. There are many explanations for the role of obesity in the development of GERD, one of the adiponectin peptides is secreted from adipose tissue and plays an important role in gastrointestinal motility, and this peptide may have circulatory disorders in patients with mucositis and symptoms. WITH GERD. The relationship between low

circulating adiponectin and central obesity is well known. It had a central effect on monocytes as an anti-inflammatory and protective-sensory protective effect, so its deficiency was associated with various inflammatory gastrointestinal diseases. This study showed a significant increase in adiponectin compared to a negative correlated control group (p = 0.023). This is consistent with other studies. These high levels of adiponectin may be responsible for abnormal healing of the esophagus, leading to metaplasia and Barrett's esophagus, or is a marker of other factors. Another study has shown that visceral fat may increase the risk of GERD by increasing levels of inflammatory cytokines and adiponectin. Therefore, high levels of adiponectin may provide protection against Barrett's esophagus in patients with GERD. Regarding the lipid profile, the study showed a significant increase in the lipid profile compared to the control, except for triglycerides, and there was a negative correlation with them consistent with another study. Triglycerides regulate the oxidation of free fatty acids in the muscles and liver of mice, which regulate the production of proteins associated with acyl-CoA oxidase, active protein kinase and metabolism of the active β receptor (PPARγ) of the peroxisome proliferator.). Despite methodological limitations and the small sample size, this study showed that abdominal obesity plays a role in the risk of GERD mediated by adiponectin, not the mechanical effect of GERD that supports obesity. Thus, the reduction of anti-inflammatory cytokines such as adiponectin may be associated with the development of GERD-related reflux symptoms, erosive esophagitis and Barrett's esophagus.

CONCLUSION:

This study underlines that circulating adiponectin hormonal intestinal disorder plays a role in the pathogenesis of GERD and may be susceptible to Barrett's esophagus.

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