The Relationship between Gastrin Hormone and Fasting Blood Sugar Levels in Patients with Diabetes Mellitus Type 2

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Abstract

This study dealing with diabetic type 2 patients to estimate the relationship of gastrin hormone level and the fasting blood sugar in diabetic patients from plasma of blood, A total samples of this study consist of (n=67) of diabetic type 2 patients & 25 control.

In this study the patients was divided in to three groups according the duration of diabetic,the percentage of patients who have duration of diabetes less than 10 years 34.4% (n=23) as 1st group, less than 20 years more than 10 years 32.8% (n=22) as 2nd group and equal or more than 20 years 32.8% (n=22) as 3rd group ,all samples test and control analysis the fasting blood sugar by chemical reagent (glusenate) and the reagent readied by spectrophotometer while the gastrin hormone level was analyses by enzyme immunosorbent assay (ELISA) kit, This Enzyme immunosorbent assay kit is designed to detect a specific peptide and its related peptides based on the principle of "competitive" enzyme immunoassay. The results of gastrin hormone were readied by ELISA. Analysis was carried out using SPSS version 18. Categorical variables were presented as frequencies and percentages. There is significant differences(p<o.oo1) between gastrin hormone levels and fasting blood sugar levels in all patients duration groups with type 2 DM.

Key word : Diabetes type 2 , Gastrin

الخلاصة

هذه الدراسة تهتم بالمرضى المصابين بداء السكري النوع الثاني لتقدير علاقة مستوى هرمون الكاسترين مع قيمة سكر الدم قبل الافطار من بلازمة مرضى السكري ، العدد الكلي للعينات في هذه الدراسة يتكون من 67 مصاب بالسكري النوع الثاني و25 سيطرة اصحاء.

في هذه الدراسة قسم المرضى الى ثلاث مجاميع على اساس فترة اكتشاف مرض السكري لديهم ،حيث ان نسبة المرضى الذين مدة اصابتهم بالمرض اقل من عشرة سنوات هي 34.4% وعددهم 23 مريض وهم المجموعة الاولى ،ونسبة المجموعة الثانية التي تكون مدة اصابتهم تكون اكثر من عشرة سنوات واقل من عشرين سنة هي 32.8% وعددهم 22 مريض واخيرا المجموعة الثالثة وتكون فترة الاصابة مساوية او اثر من عشرين سنة ونسبتهم 32.8% حيث كان عددهم 22 مريض ،كل العينات من مصابين بالسكري واصحاء السيطرة عمل لهم تحاليل السكري قبل الافطار بواسطة الكواشف الكيميائية وتقرأ بواسطة جهاز مقياس الطيف الضوئي بينما مستوى هرمون الكاسترين يقاس بواسطة جهاز ELISA ،ج القراءات عوملت إحصائيا باستعمال برنامج SPSS النسخة 18 وكانت هناك اختلافات معنوية بين مستوى هرمون الكاسترين ومستوى السكر في المجاميع كلهم المقسمة على اساس فترة الاصابة بداء السكري النوع الثاني .

كلمة مفتاحية: داء سكري النوع الثاني ، هرمون الكاسترين

Introduction

Diabetes mellitus is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both and it is a chronic illness that requires continuing medical care and ongoing patient self-management education and support to prevent acute complications and to reduce the risk of long-term complications. Diabetes care is complex and requires multifactorial risk reduction strategies beyond glycemic control (American Diabetes Association, 2013).

In humans, gastrin is a peptide hormone that stimulates secretion of gastric acid (HCl) by the parietal cells of the stomach and aids in gastric motility. It is released by G cells in the antrum of the stomach (the portion of the stomach adjacent the pyloric valve), duodenum, and the pancreas. It binds to cholecystokinin B receptors to stimulate the release of histamines in enterochromaffin-like cells, and it induces the insertion of K^+/H^+ATPase pumps into the apical membrane of parietal cells (which in turn increases H^+ release into the stomach cavity). Its release is stimulated by peptides in the lumen of the stomach Fritsch, Hausamen, Scholten (1977).

Its existence was first suggested in 1905 by the British physiologist John Sydney Edkins (Rozengurt, Walsh 2001; Dockray 2005)., and gastrins were isolated in 1964 by Roderic Alfred Gregory at the University of Liverpool (Anlauf *et al,.* 2006) . In 1964 the structure of Gastrin was determined
(Polosatov *et al* 1979).

Gastrin levels are measured with a fasting blood test. Gastrin is released when food enters the stomach, so non fasting blood tests have false result . Several drugs, such as protein pump inhibitors, opiates, and aspirin, can interfere with the testing of gastrin and cause incorrect readings. Prior to testing, the physician will direct the patient how far in advance of the test to discontinue use of these drugs A gastrin test may be done to:
Find out why a peptic ulcer keeps coming back , Check for certain diseases, such as tumors of the pancreas or small intestine (Zollinger-Ellison syndrome) or abnormal growth of cells that line the stomach (G-cell hyperplasia) and Help identify pernicious anemia. (Anlauf *et al.* 2006).

In autoimmune gastritis, the immune system attacks the parietal cells leading to hypochlorhydria (low stomach acidity). This results in an elevated gastrin level in an attempt to compensate for increased pH in the stomach. Eventually, all the parietal cells are lost and achlorhydria results leading to a loss of negative feedback on gastrin secretion. Plasma gastrin concentration is elevated in virtually all individuals with mucolipidosis type IV (mean 1507 pg/mL; range 400-4100 pg/mL) (normal 0-200 pg/mL) secondary to a constitutive achlorhydria. This finding facilitates the diagnosis of patients with this neurogenetic disorder Schiffmann *etal*., (1998).
the aim of the Study: Because of the distribution of diabetes mellitus and the little studies that concerns by subjects suffer from diabetes mellitus especially type 2 in Iraq, we went to study gastrine hormone levels and there relationship with fasting blood sugur in patients with type 2 diabetes mellitus.

Materials and Methods

1. Patients and Conditions of Study:

This study was done in Merjan teaching hospital in Babylon province. The blood samples taken from 67 patients from the diabetic clinic in the mentioned hospital and 25 healthy subjects were taken as control (total persons 92). All patients were suffered from type 2 diabetes from which males and females. The ages of patients and controls were ranges between 20-60 years old.

2. Collection of blood samples

Blood samples were collected often fasting (8-12hr.) from healthy control and diabetic patients by vein puncture using 5 ml disposable syringes in order to the levels of plasma blood glucose were estimated according to manual (procedure) of Biomerx marker and the levels of gastrine hormone by ELISA according to the manual (procedure) of DRG International Inc.,USA.

Before the plasma samples were taken to estimate the levels of gastrin hormone the patients and control must be do not drink alcohol for 24 hours before the test ,do not eat for 12 hours before the test, do not eat or drink anything with caffeine, such as coffee, for 12 hours before the test and do not chew gum or smoke cigarettes for 4 hours before the test but they can drink as much water as you want up to 1 hour before the test

3.Calculation:

Determine the absorbance change as:
Δ A Sample = (A sample – A blank)
Δ A Standard = (A standard – A blank)
and used this for the calculation of serum glucose.
The concentration of the supplied standard / calibrator R4: (100 mg/dl) or (5.55mmol/L). (Clinical Guide to laboratory test 2006).

4. Statistical Analysis

Statistical analysis was carried out using SPSS version 18. Categorical variables were presented as frequencies and percentages. A p-value of ≤ 0.05 was considered as significant.

Results

The clinical characteristic features of patients &control group:

A total sample of this study consist of (n=67) of diabetic type 2 patients. In this study the patients were divided according to duration of diabetes,1st group the duration of diabetes less than 10 years 34.4% (n=23),2nd group duration of diabetes less than 20 years more than 10 years 32.8% (n=22),3rd group equal or more than 20 years 32.8% (n=22) and control (n=25).

The results of the present study showed that there were significant differences between gastrin mean of fasting blood sugar levels for control and gastrin means of FBS levels for all patients groups of type 2 DM, as show in figure (1) there are relationship between gastrin mean of fasting blood sugar levels for control and gastrin means of FBS levels for all patients group of type 2 DM and figure(2) show that the level of mean gastrin few less 100 ng/ml when the fasting blood sugar was less than 100mg/dl (Normal) but have high distribution , the level of mean gastrin was increased few more than 100 nm/ml when fasting blood sugar was between 100-125mg/dl Impaired fasting glucose (IFG) but have little distribution and when the fasting blood sugar was increased more than 125mg/dl mean there was diabetic mellitus (DM) the mean of gastrin hormone was increased near 200ng/ml and have high distribution .

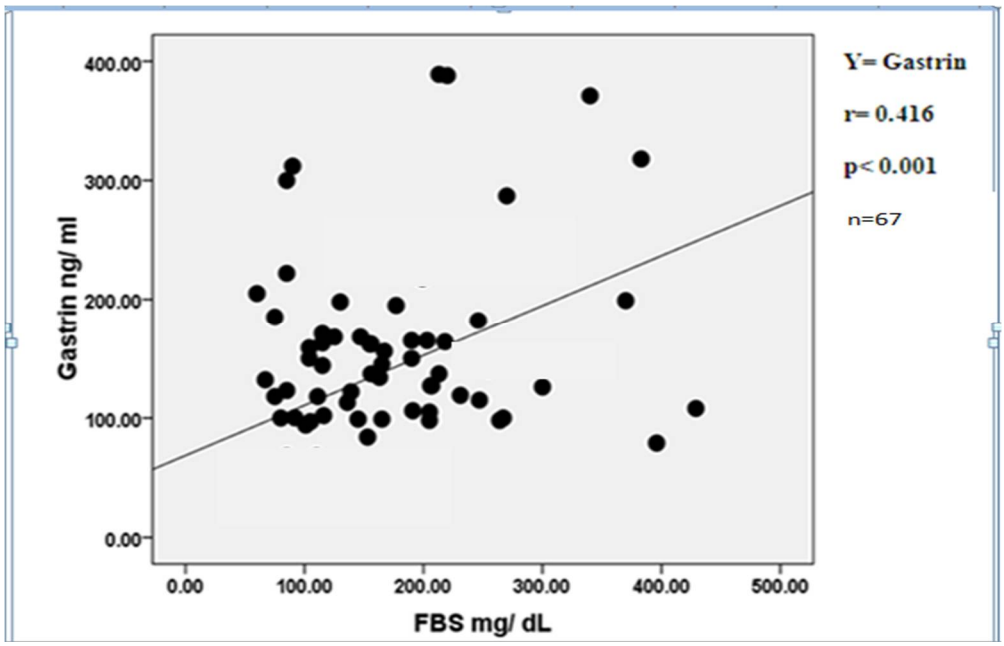
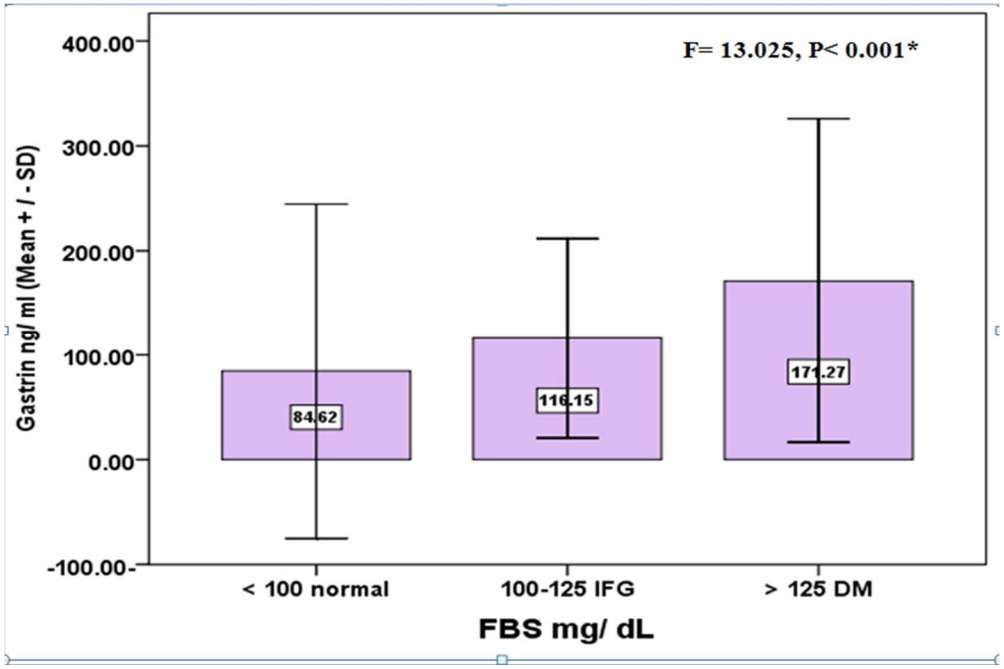


Figure (1): The relationship between gastrin levels (ng/ml) for all duration groups of type 2 DM patients and FBS(mg/dl)



Figure(2).The mean differences between gastrin levels for each duration groups of type 2 DM by FBS levels(ng/ml), separately.

As show in table (1) there is three groups of diabetic patients according the duration of disease and each group which divided in to three groups according to level of fasting blood sugar <mg/dl,100-125mg/dl and >125mgldl then compeer between the means of gastrin hormone levels by F test that show significant differences between them in 1st group p.value 0.023,2nd group p.<0.001 and 3rd group p.value <0.001 .

Table (1) .shows the mean differences between gastrin levels (ng/ml) for each duration groups of type 2 DM by FBS levels, separately.

Groups	FBS	N	Gastrin(ng/ml) Mean±S.D	F	P value
1st group of type 2 DM	<100 mg/dl	12	69.04±65.01	4.117	**0.023***
	100-125 mg/dl	4	97.74±48.83		
	>125 mg/dl	7	123.33±30.36		
2nd group of type 2 DM	<100 mg/dl	13	59.08±41.48	20.17	**<0.001***
	100-125 mg/dl	3	104.74±51.96		
	>125 mg/dl	6	147.83±30.40		
3rd group of type 2 DM	<100 mg/dl	12	86.51±16.64	12.15	**<0.001***
	100-125 mg/dl	4	118.90±42.03		
	>125 mg/dl	6	81.34±23.48		

Discussion

There are significant differences between gastrin mean level of control and gastrin means level of groups for all type 2 DM groups **table(1).**

These studies indicate that in normal man the ingestion of various foods elicits a rise in serum gastrin. As judged by the time of occurrence of the peak response this is probably due to local stimulation of gastrin release. That there is a vagal component as well is suggested by the commencement of the response within 10 minutes of ingestion.

Although comparisons of stimuli were not always made in the same subjects, a number had their response studied following four or five different stimuli, the responses of these individuals were similar to the mean of each group studied, and hence it was felt that valid conclusions could be drawn in the comparison of different foods Gail and Gary (2013).

This agree with Emas and Grossman (1969) found that truncal vagotomy caused an increase in the response of Heidenhain pouches to a feeding meal.

They suggested that although the vagal component of gastrin release was eliminated, these procedures produced stasis of food in the stomach and increased the contact time with antral mucosa.

These studies do indicate that there may be some physiological basis for cream, white meat, bread and butter diets in the therapy of duodenal ulceration Foggensteiner (2001).

Indeed if gastrin has any acid stimulatory capacity then pure protein feedings would be least desirable and pure fat or carbohydrate feedings most desirable.

However, any therapeutic applications of the present results would have to be tempered by the assessment of the acid neutralizing properties of substances studied as well as of their ability to release gastrin, the secretion of gastrin was increased in diabetic patients even there was no cardiovascular diseases in patients Gulliford, Bicknell, Scarpelo (1988).

References

American Diabetes Association (2013) Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care, 32* (1): S62-S67.

Anlauf M, Garbrecht N and Henopp T. *et al.* (2006). "Sporadic versus hereditary gastrinomas of the duodenum and pancreas: distinct clinico-pathological and epidemiological features". *World J. Gastroenterol.* **12** (34): 5440–6.

Avrameas, S. Journal of Immunological Methods, Amplification Systems in Immunoenzymatic Techniques 150 (1992) 23-32.

Dockray GJ (2005). "Clinical endocrinology and metabolism. Gastrin". *Best Pract. Res. Clin. Endocrinol. Metab.* 18 (4): 555–68.

Emas, S., and Grossman, M. I. (1969). Response of Heidenhain pouch to histamine, gastrin and feeding before and after truncal vagotomy in dogs. Scand. J. Gastroent., 4, 497-503.

Foggensteiner, L.; Mulroy, S. and Firth,J. (2001) Management of diabetic nephropathy.J R Soc Med, 94:210-217.

Fritsch WP, Hausamen TU and Scholten T (1977). "[Gastrointestinal hormones. I. Hormones of the gastrin group]". *Zeitschrift für Gastroenterologie* **15** (4): 264–76.

Gail L Matters1 and Gary A Clawson(2013) A Speculative Role for Stromal Gastrin Signaling in Development and Dissemination of Pancreatic Ductal Adenocarcinoma, Department of Biochemistry and Molecular Biology, Hershey Medical Center, Pennsylvania State University, Hershey, PA, USA.

Gulliford MC, Bicknell EJ and Scarpelo JH.(1988) Evaluation of gastrin secretion in diabetic subjects with normal or abnormal cardiovascular autonomic function tests. Department of Endocrinology and Diabetes Mellitus, North Staffordshire Royal Infirmary, Stoke-on-Trent, U.K. 25(4):275-82.

Polosatov MV, Klimov PK, Masevich CG and *et al.* (1979). "Interaction of synthetic human big gastrin with blood proteins of man and animals". *Acta hepato-gastroenterologica* 26 (2): 154–9.

Porstmann, T., and Kiessig, S.T (1992). Journal of Immunological Methods ,Enzyme Immunoassay Techniques, 150 5-21.

Rozengurt E and Walsh JH (2001). "Gastrin, CCK, signaling, and cancer". *Annu. Rev. Physiol.* **63**: 49–76.

Schiffmann R, Dwyer NK, Lubensky IA, Tsokos M, Sutliff VE, Latimer JS, Frei KP, Brady RO, Barton NW, Blanchette-Mackie EJ, Goldin E (1998). "Constitutive achlorhydria in mucolipidosis type IV". Proc Natl Acad Sci U S A. 95 (3): 1207–12.

Tietz .N.W. (2006).Clinical Guide to laboratory test ,4th Ed., p.1074-1077 .

Watson S and Arkinstall. S. (1994). Cholecystokinin (CCK) and gastrin. New York: Academic Press pp. 89–95.