The Relationship between Gastrin Hormone Level and Microalbumin Urea in Patients with Diabetic 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 micralbumin urea in diabetic patients, 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 microalbumin urea levels by the microalbuminuria strips is MICRAL-TEST marker 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. Analysis was carried out using SPSS version 18. Categorical variables were presented as frequencies and percentages . The mean differences between gastrin mean of microalbuminurea for control and gastrin mean level of microalbuminurea for all duration groups of type 2 DM, there is significant differences between gastrin mean and microalbumin urea levels (t-test=5.199.p<0.001*).

Introduction

Diabetes mellitus 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.

A large body of evidence exists that supports a range of interventions to improve diabetes outcomes ,and it is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both. (American Diabetes Association, 2013).

It is characterized by hyperglycemia and symptoms of marked hyperglycemia include polyuria, polydipisia, weight loss, sometimes with polyphagia, and blurred vision (American Diabetes Association, 2002, 2009).

Diabetes mellitus produces inadequate blood glucose control and leads to acute and chronic complications (Koda-Kimble and Carlisle, 1995). Acute complications include diabetic ketoacidosis and hyperosmolar nonketotic coma (Singh and Marshall, 1995).

Chronic complications can involve the kidneys, eyes, nervous system, and cardiovascular system and can be classified as either macrovascular or microvascular (Koda-Kimble and Carlisle, 1995).

The diagnosis of type 2 DM often is made 4 to 7 years after the disease process has begun, when most patients already have an increased risk of macrovascular processes (Peters and Schriger, 1998), despite this, 20% to 25%of patients with DM do not develop macrovascular complications (Koda-Kimble and Carlisle, 1995) and 20% of patients present with retinopathy; 8%, nephropathy; and 9%, neuropathy Peters and Schriger, (1998).

Glomerulonephritis,   is   a   progressive kidney   disease caused   by angiopathy of capillaries in the kidney glomeruli. It is characterized by nephrotic syndrome and diffuse glomerulosclerosis. It is due to longstanding diabetes mellitus, and is a prime indication for dialysis in many Western countries. However, of the long-term complications associated with DM, diabetic nephropathy is associated with the highest mortality (Nathan, 1993).

Diabetic nephropathy occurs when proteins deposit in the glomerulus Clark & Lee (1995). Thickening of the glomerular capillary basement membrane narrows the lumen of the capillaries, impeding blood flow and subsequently reducing the filtering surface of the glomerulus (Koda-Kimble &Carlisle, 1995).

The principal manifestation of diabetic nephropathy is microalbuminuria and proteinuria is generally regarded as a marker for the degree of glomerular damage: the levels of proteinuria correlates well with the prognosis for renal function, and interventions that retard the progression of renal disease also reduce proteinuria. Foggensteiner *et al*.,(2001).

MATERIALS AND METHOD :

1. Patients and conditions of study

We design the plan of this study to measure some parameters in subjects suffer from type 2 diabetes and compared it with healthy subjects in the same ages, this parameters include:

1- Measurment the level of gastrine hormone.

2-Measurment the level of microalbumin urea.

    This study was done in Merjan teaching hospital in Babylon province. The collection of samples were conducted during the period from January to February / 2013. The 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 estimate the levels of gastrine hormone by ELISA according to the manual (procedure) of DRG International Inc.,USA and 5ml of urine in order to estimate the levels of Microalbumine urea by the strip of Accu-Chek Micral test kit .

    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.Reagents:

Kit	Marker and Origin
Microalbumine urea	Accu-Chek Micral test / Mannheim,Germany
Gastine hormone	DRG International Inc.,USA

4.Methods :

A- Gastrin I (Human) ELISA : Porstmann,*et al* (1992); Avrameas,*et al* (1992).

B- **Measurement of Microalbuminuria:** (The Microalbuminuria Kit is MICRAL-TEST   marker).

  1- The test strip was placed in the urine such that the fluid level was just between the two black bars, making sure that it does not touch the side of the vessel in the process. The test strip was withdrawn after 5 seconds and placed it across the top of the urine vessel.

  2- After 1 minute, the color of the test pad above the inscription "Micral" was compared with the color scale on the test strip container label.

RESULTE

The clinical characteristic features of patients &control group:

    A total sample of this study consist of (n=67) of diabetic type 2 patients consist of 59.7% (n=40) males and 40.3% (n=27) females & 25 control , 39.9 % of the total samples have a family history for diabetes mellitus.

    In this study the percentage of patients who have duration of diabetes less than 10 years 34.4% (n=23), less than 20 years more than 10 years 32.8% (n=22), equal or more than 20 years 32.8% (n=22) and control (n=25),all diabetic patients divided into three groups dependend on duration of infected with suger**,** The patients who have microalbuminuria 55.2% (n=37).

    The mean differences between gastrin level of all duration groups of type 2 DM with control by microalbumin urea levels, there is significant differences between gastrin mean and microalbumin urea levels .

Table : Mean differences of gastrin level for each type 2 DM group with control by microalbumin urea levels

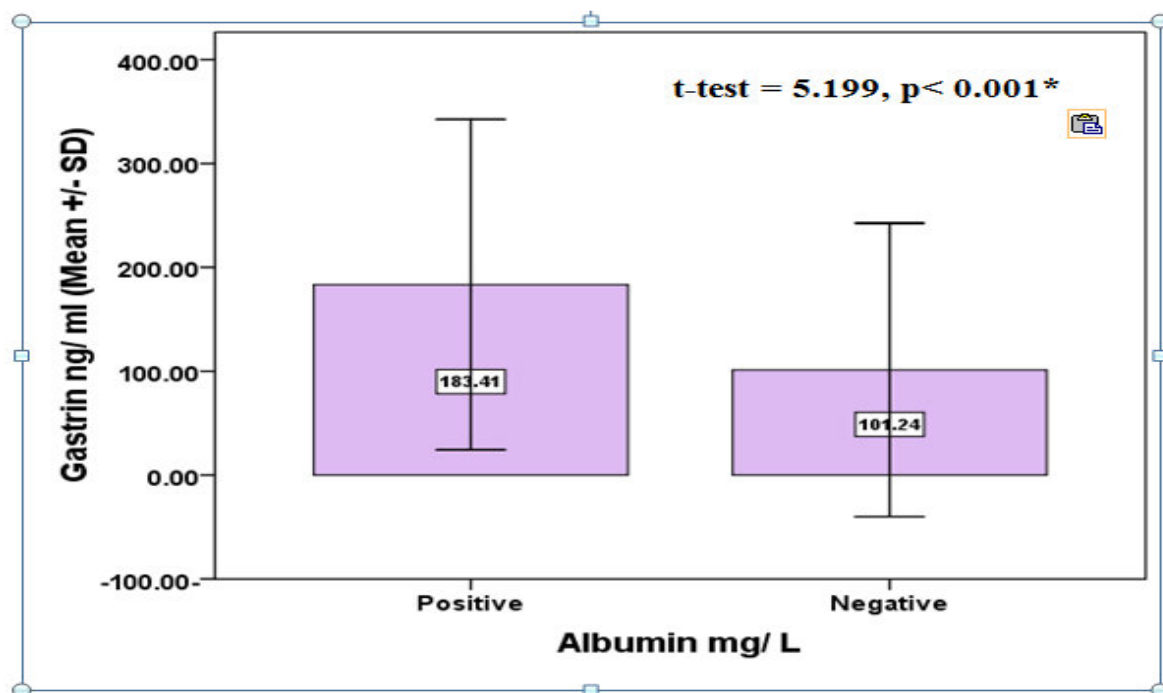| Gastrin level	microalbumin urea	N	Mean ±S.D	t-test	P value
1st group of type 2 DM	Positive	8	146.37 ±43.25	3.394	**0.001***
	Negative	40	75.60 ±55.53		
2nd group of type 2 DM	Positive	8	142.0 ±36.84	3.225	**0.002***
	Negative	39	78.74 ±52.67		
3rd group of type 2 DM	Positive	8	271.12 ±85.59	4.809	**<0.001***
	Negative	39	100.60 ±92.39		

Figure 4.2.12: Mean differences of gastrin for all duration groups of type 2 DM with control by microalbumin urea

DISCUTION :

The mean differences between gastrin level of all type 2 DM groups with control by microalbumin urea levels, The mean differences between gastrin mean of microalbuminurea for control and gastrin mean level of microalbuminurea for all duration groups of type 2 DM.

Although diabetic nephropathy is a very rare cause of kidney failure during childhood, the underlying events leading to progressive kidney injury begin during childhood in many patients with type 1 diabetes mellitus (Type1DM) and in increasing numbers of children with type 2 diabetes mellitus (Type2DM). The Pima Indians of Arizona represent an exceptionally thoroughly studied population suffering from very high rates of Type2DM and diabetic nephropathy (Type2DN). This population well illustrates the often inexorable progression from glomerular hyperfiltration to microalbuminuria to overt proteinuria and loss of glomerular filtration rate (GFR), paralleled by the accumulation of mesangial matrix and basement membrane, glomerular hypertrophy, loss of podocytes and eventual glomerular sclerosis and interstitial fibrosis. Structural changes quantitatively account for the loss of GFR in T2DN. The mechanism of albuminuria (and its relationship to GFR loss) is much less clear. There is strong functional and structural evidence for defects in glomerular size-selectivity (shunts) due to podocyte pathology, but only beginning at relatively high levels of proteinuria (albumin/creatinine ratios > 3000 mg/g). Podocyte loss accompanies, and may underlie, the loss of glomeruli to sclerosis. At this point, most evidence in humans suggests detachment of intact podocytes from the glomerular basement membrane, rather than apoptosis, as the predominant mechanism of podocyte loss. Lemley.(2008)

In this study the result   might be show that the level of gastrin hormone increased in all type 2 DM this agree with Emas and Grossman (1969) found that truncal vagotomy caused an increase in the response of Heidenhain pouches to a feeding meal and the level of microalbuminurea in urine was increased Lemley.(2008).

References:

- American Diabetes Association (2013) Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care, 32* (1): S62-S67.
- American Diabetes Association (2009) Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care, 38* (1): S72-S77.
- American Diabetes Association. (2002) Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care, 23* (1):S5-S20.
- Avrameas, S. Journal of Immunological Methods, Amplification Systems in Immunoenzymatic Techniques 150 (1992) 23-32.
- Clark, C.M. and Lee, D.A. (1995) Drug therapy: prevention and treatment of the complications of diabetes mellitus. *New Engl J Med, 332*:1210-1217.

- 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.
- Lemley KY.(2008). Diabetes and chronic kidney disease: lessons from the Pima Indians. Division of Nephrology, Childrens Hospital Los Angeles, Los Angeles. 23(11):1933-40.
- Koda-Kimble, M.A. and Carlisle, B.A. (1995) Diabetes mellitus. By: Young, L.Y.; Koda-Kimble; M.A.; Kradjan, W.A. and Guglielmo, B.J. (eds) In: Applied therapeutics: the clinical use of drugs. 6th ed. Vancouver (WA): Applied Therapeutics 48. p. 48-1-48-58.
- Nathan, D.M. (1993) Medical progress: long-term complications of diabetes mellitus. *New Engl J Med, 328*:1676-85.
- Peters, A.L. and Schriger, D.L.(1998) The new diagnostic criteria for diabetes: The impact on management of diabetes and macrovascular risk factors. *Am J Med, 105*: 15S-19.
- Singh, I. and Marshall, M.C. (1995) Diabetes mellitus in the elderly. *Endocrinol Metab Clin North Am, 24*:255-72.
- Porstmann, T., and Kiessig, S.T. Journal of Immunological Methods ,Enzyme Immunoassay Techniques, 150 (1992) 5-21.

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