Protein bound fucos, protein bound hexose and total calcium in sera of patient with thyroid dysfunction and control

W.F.Altai, N.F.AL-arrag ,T. M. A. Rajab and H. G. Hassan*

Department of Chemistry, College of Education, Ibn Al-Haithiam, University of Baghdad.

*** Department Chemistry, College of science, University of Sulaimani**

Abstract

Protein bound fucose (PBF), protein bound hexose (PBHex), and total calcium (T.Ca) were determined in sera of (40) hyperthyroidism , (40) hypothyroidism patients and (40) control. The results revealed a significant decrease in the level of PBF, PBHex and T.Ca in sera of patients with hyperthyroidism compared to control; In case of PBF, there no difference in its level between patients with hypothyroidism and control group. While there is a significant increment in PBHex level in both hyper and hypothyroidism with respect to that of control. Result indicates, that total calcium levels were in the normal range for all patients groups. Patient compared to control no difference in the level of (PBF) between hypothyroidism and control. A significant increase in the level of PBHex in sera of hyper and hypothyroidism compared to controlwas found.

Introduction

Glycoproteins are molecules that are combinations of sugar and protein.

Eminent scientists in the field of glycobiology have proposed that specific dietary sugars (glyconutrients) could represent a new class of nutrients with interesting benefits to health (1) Of the 200 monosaccharides that occur naturally in plants, fewer than ten are

predominantly used by the body for healthy function.
Glucose, found in table sugar, is only one of them. Others identified to data are:
Mannose, galactose, xylose, fucose, N-acetylglucosamine, N-acetylgalactosamine, and sialic acid (N-acetylneuraminic acid or NANA) (2).
In mammalian glycoproteins, only the L-enantiomer of fucose is identified (3).
In these glycoproteins, L-fucose and sialic acid typically occupy terminal positions at the nonreducing ends of oligosaccharide chains. Fucose also is found in a number of membran-associated glycolipids. There is evidence that L-fucose is accumulated in eukaryotic cells by a specific transport system (4)
The body's calcium economy is determined by the relationship between the intestinal absorption of calcium, the renal handling of calcium, and by the movements of calcium in and out of the skeleton. These processes are influenced by many factors, the most important of which are parathyroid hormone and the hormones derived from the renal metabolism of vitamin D3, notably 1,25-di hydroxy vitamin D3. The role of endogenous calcitonin in man is still controversial, but there are several other hormones which have some influence on calcium metabolism, including thyroid hormone, growth hormone, and the adrenal and gonadal steroids (5).
Calcitonin, a peptide hormone secreted by the thyroid gland, it is a 32-amino acid peptide with a molecular weight of about 3400 dalton, the primary stimulus for calcitonin secretion is increased plasma calcium ion concentration.
This contrasts with parathyroid hormone (PTH) secretion, which is stimulated by decreased calcium concentration (6)
The calcium-dependent carbohydrate- binding proteins E-selectin is expressed on the surfaces of activated venous endothelial cells, and P-selectin is expressed on platelets. (7)
Leukocyte adhesion deficiency type II (LADII) is a defect lack fucosylated glycoconjugates, the defect in LADII to the denovo pathway of GDP-fucose biosynthesis, by inducing cell-surface expression of fucosylated glycoconjugates after exposure of lymphoblastoid cell lines from the LADII patients to exogenous fucose (8).
Recently, it has been proposed that fucosylation of the L-, E-, and P-selectin ligands is essential for leukocyte adhesion and trafficking

(9)(10) The (1,3) fucosylatransferase, FucT-VII, has been shown to be the critical enzyme for synthesizing the sialyL Lewis x (slex) determinant and fucosylation of selectin ligands in mice (11)

Sampling

The samples were collected from (Specialized center of endocrinology and diabetes).

They have been classified into three groups as the following:

- Control group: include (40) healthy individual from both sexes, with no previous disease, which may interfere with the parameters analyzed in this study.
- Hyperthyroidism patients group: include (40) patient suffering from hyperthyroidism from both sexes.
- Hypothyroidism patients group: include (40) patient suffering from hypothyroidism from both sexes.

Collection of Blood

A (10) ml Vienous blood was taken from the above groups, placed in a plane tube (no anti coagulant) left for (15 min) at room temperature, the centrifuged (at 2500 rpm for 10min) to get the serum, which is stored at (-20oC) unless used immediately.

Methods

Determination of protein bound fucose (PBF)

Protein bound fucose (PBF) determination according to Dische and Shettles method (12)

Determination of protein bound hexoses (PBHex)

Protein bound hexoses (PBHex) determination according to Rimingto (13)

Determination total calcium (T.Ca)

Total calcium (T.Ca) determination according to Lorenz (14).

Statistical analysis

Data presented as the means ± standard deviations, student-t-test was used to compare the significance of the difference in the mean values of any two groups, ($P<0.05$) was considered statistically significant

The overall predictive values for the results in all studied groups were performed according to program of office XP 2002

Results and discussion

Table (1). Fig. (1a) and Fig. (1b) showed the result of PBF, PBF/TP, PBHex, and PBHex/TP.

A significant reduction of protein bound fucose for hyperthyroidism compared to control with P value (0.0001) while no alteration in PBF between hypothyroidism and control groups was found. Also significant differences were found between hyper and hypothyroidism with P value (0.0001).

An elevated levels of PBHex for hyper and hypothyroidism compared to control also a significant relation was found between hyper and hypothyroidism themselves

Table (2) and Fig (2) showed the concentration of total calcium in three studied groups, a significant elevation for both hyper and hypo thyroidism was found compared to control while no significant relation between the two patients groups with P value (0.478).

The disturbance in the level of PBF in patients group should be due to the fucoligands in the cells of interest.

Enzymes required for synthesis of fucoligands could be present in the cells. Fucoligand expression on tumor cells should be abundant at the cells that are involved in a cell contact or cell-adhesion process (i.e., metastasis or invasion) if distribution of fucoligands on the cell surface is not uniform (15).

Cell-surface fucoligands have been demonstrated in most common human malignant neoplasms. including carcinomas of thyroid, (16,17,18)

The importance of exchangeable calcium is that it provides a rapid buffering mechanism to keep the calcium ion concentration in the extracellular fluids from rising to excessive levels or falling to very low levels under transient conditions of excess or hypo availability of calcium (6).

Calcitonin has a weak effect on plasma calcium concentration in the adult human the reason for the weak effect of calcitonin on plasma calcium are:

- First, any initial reduction of the calcium ion concentration caused by calcitonin leads within hours to a powerful stimulation of PTH secretion, which almost overrides the calcitonin effect when the thyroid gland is removed and calcitonin is no longer secreted, the long-term blood calcium ion concentration is not measurably altered, which again demonstrates the overriding effect of the PTH system of control.

- Second, in the adult, the daily rates of absorption and deposition of calcium are small, and even after the rate of absorption is slowed by calcitonin, this still has only a small effect on plasma calcium ion concentration (19,20,21)

Referance

1. Berger, V.; Perier, S. and Pachiaudi, C. (1998). Metabolism., 47 (12): 1499-1503.
2. Murray, R. K. (1996). "Glycoproteins. In": Murray, R.K.; Granner, D. K.; Mayes, P.A. and Rodwell, V.W. editor (5). "Harper's Biochemistry Appleton and longe" PP.648-666.
3. Flowers, H. M. (1981). Adv. Carbohydr chem. Biochem; 39: 279-345,
4. Wiese, T.J.; Dunlap, J.A. and Yorek, M. A. (1994). J.Biol Chem.; Sep 269: 22705-22711.
5. Russell, R. G. G. (1976). Ann. Clin. Biochem., 13:518-539.
6. Guyton, A. C. and Hall, J. E. (2000). "Text Book of medical physiology" 10th ed. PP. (862-865), P.899, 909,908.
7. Kojima, N.; Handa, K. and Newman, W. (1992). Cells. Biochem Biophys Res commun., 182: 1288-1295.
8. Karsan, A.; Cornejo, C. J.; Winn, R. K.; Schwartz, B.R.; Way, W.; Lannir, N.; Gershoni, B. R.; Etzioni, A.; Ochs, D. H. and Harlan, J.M. (1998). J. Clin. Invest., 101: 2438-2445.
9. Wagers, A. J.; Lowe, J. B. and Kansas, G. S. (1996). Blood., 88: 2125-2132.
10. Maly, P. A. D.; Thall, B.; Petryniak, S. E.; Rogers, P. L.; Smith, R. M.; Marks, R.J.; Kelly, K. M.; Gersten, G.; Cheng, T. L. and Saunders, L. (1996). Cell., 86: 643-653.
11. Natsuka, S. K. M.; Gersten, K.; Zenita, Kannagi, R. and Lowe, J. B. (1994). J. Biol. Chem., 269: 16789-16794.
12. Dische, Z. and Shettles, L. B. (1948). J. Biol. Chem., 175: 595-603.
13. Rimingto, C. J. (1940) Biochem., 34: 931.
14. Lorenz, K. (1982). Clin. Chem. Acta., 126: 327-334.
15. Vas, W. L. C. and Almeida, P. F. F. (1993). Curropin struct Biol., 3: 482-488.
16. Schroder, S. and Dralle, H. (1989). Horm Metab Res Suppl., 21: 26-28.
17. Vierbuchen, M.; Larena, A. and Schroder, (1992). Virchows Arch

cell Pathol Incl Molpathol., 62: 79-88.
18. Miettinen, M. and Karkkainen, P. (1996). Virchows Arch., 429: 213-219.
19. Livolsi, V. A. and Delellis, R. A. (1993). "Pathology of the parathyroid and thyroid glands".Baltimore: Williams and Wilkins.
20. Petersen, O. H.; Peterson, C. C. and Kasai, H. (1994). Annu Rev Physiol., 56: 297.
21. Kettyle, W. M. and Arky, R. A. (1998) "Endocrine pathophysiology" Philadelphia: Lippincott - Raven.

Table (1) PBF, PBF/TP, PBHex and PBHex/TP levels in sera of three studied groups

Group description	No.	PBF (mg/dl) mean ±SD	P	PBF/ TP (mg/g) mean ±SD	P	PBHex (mg/dL) mean ±SD	P	PBHex /TP(mg/g) mean ±SD	P
Control	40	9.5 ± 1.47		1.3 ± 0.23		124.2 ± 0.27		17.5 ± 1.27	
Hyperthyroidism	40	5.6 ± 1.54	0.0001	0.8 ± 0.21	0.0001	133.8 ± 1.59	0.0001	18.9 ± 1.46	0.103*
Hypothyroidism	40	9.6 ± 1.49	0.656	1.34 ± 0.31	0.768	141.9 ± 1.99	0.0001	19.9 ± 3.51	0.0001
			0.0001*		0.0001*		0.0001*		0.0001

*Represent P value between hyperthyroidism and hypothyroidism

Table (2) T.Ca in sera of three studied groups

Group description	No.	T.Ca (mg/dl) mean ±SD	p
Control	40	9.4 ± 0.06	
Hyperthyroidism	40	10.9 ± 0.48	0.0001
Hypothyroidism	40	10.9 ± 0.53	0.0001
			0.478 *

*Represent P value between hyperthyroidism and hypothyroidism.

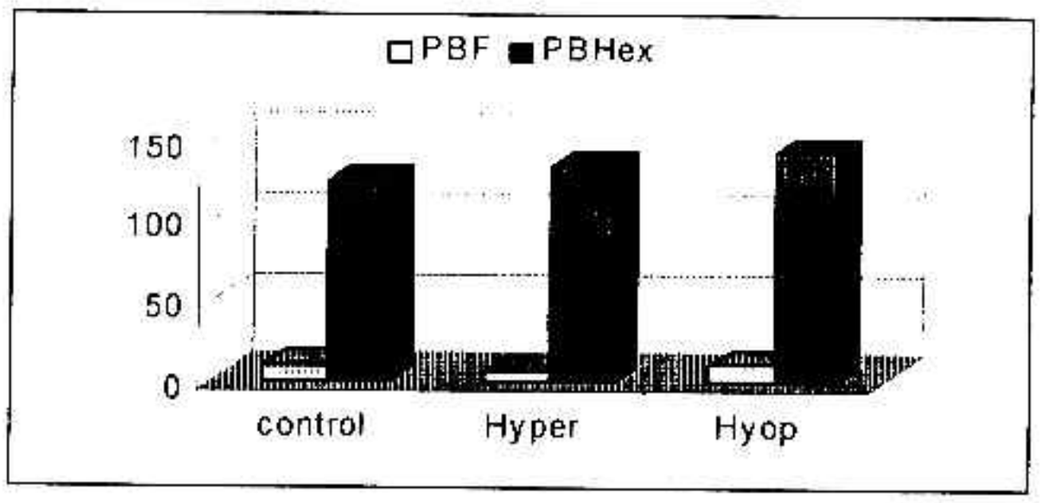

Fig. (1a) PBHex and PBF levels in sera of hyperthyroidism, hypothyroidism and control

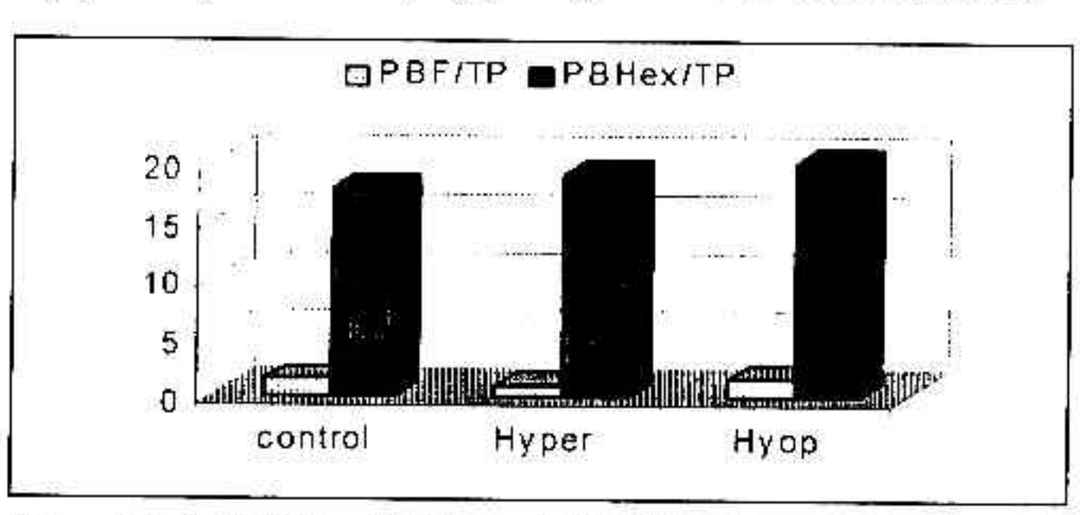

Fig. (1b) PBHex/TP and PBF/TP ratio in sera of hyperthyroidism, hypothyroidism and control

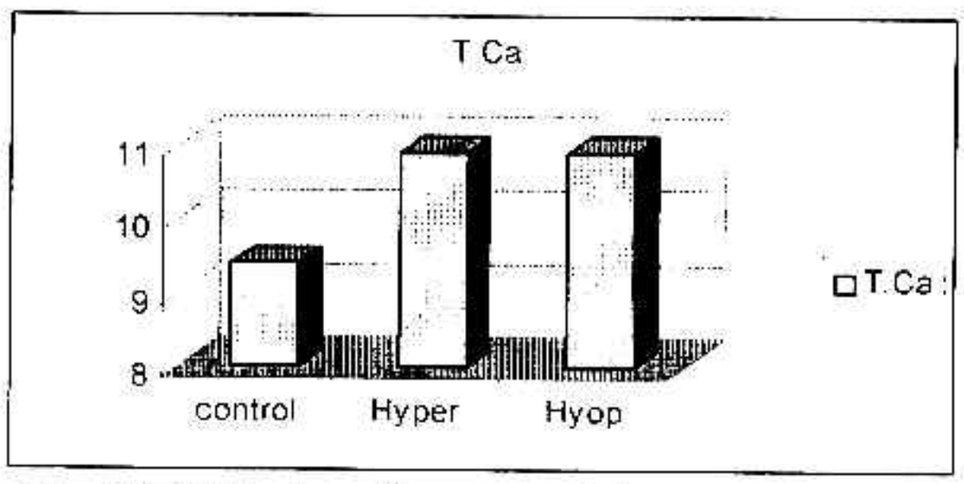

Fig. (2) T.Ca level in sera of hyperthyroidism, hypothyroidism and control

البروتين المرتبط بالفوكوز،البروتين المرتبط بالهكسوز والكالسيوم الكلي في مصل دم مرضى الخلل الوظيفي في الدرقية والسيطرة عليه

وفاء فاضل الطائي ،نجود فيصل السراج ،طارق محمد علي رجب و حامد غفوري حسن*

قسم الكيمياء ، كلية التربية – ابن الهيثم ، جامعة بغداد

* قسم الكيمياء ،كلية العلوم ،جامعة السليمانية

الخلاصة

تم قياس الفوكوز المرتبط بالبروتين والسكريات السداسية المرتبطة بالبروتين والكالسيوم الكلي في مصول دم (40) من المرضى المشخصين بالفرط في إفراز الدرقية و(40) من المشخصين بالقصور في الدرقية وكذلك (40) عينة لمجموعة السيطرة من الأصحاء ظهر من النتائج أن هناك انخفاض معنوي في مستوى الفوكوز المرتبط بالبروتين والسكريات السداسية المرتبطة مع البروتين وكذلك الكالسيوم الكلي في مصول دم مرضى الفرط في إفراز الدرقية مقارنة مع مجموعة السيطرة بينما لم تظهر فروق معنوية في مستوى الفوكوز المرتبط بالبروتين في مرضى قصور الدرقية مقارنة مع مجموعة السيطرة كذلك هنالك زيادة معنوية في السكريات السداسية المرتبطة مع البروتين في مرضى الفرط في إفراز الدرقية ومرضى القصور في إفرازها مقارنة مع الأصحاء اما الكالسيوم الكلي فقد كان في المستويات الطبيعية بالنسبة لمجاميع المرضى مقارنة مع مجموعة السيطرة . كذلك لا توجد فر وقات في مستوى الفوكوز المرتبط مع البروتين بين مرضى القصور في إفراز الدرقية ومجموعة السيطرة، هناك زيادة معنوية في السكريات السداسية المرتبطة مع البروتين بالنسبة لمجموعة الفرط في إفراز الدرقية والقصور في إفرازها مقارنة مع الأصحاء .