ASSESSMENT OF THYROID PROFILE AND ITS CO-RELATION WITH DIFFERENT BLOOD GLUCOSE LEVELS

Zahid Irfan Marwat[1], Nadia Haleem[2], Chaman Gul[2], M. Arshad[2]

ABSTRACT

Background: This is an endocrine gland situated in the lower part of the front and side of the neck. It resembles an H in shape. (Follicles). These are spherical sacs consist of simple epithelial cells and whose lumen contains colloid. The wall of each follicle consists of two types of cells Follicular cells these cells reach the surface of the lumen of the follicle. They manufacture thyroxine T4 90 % and tri-iodothyronine 10 %. Para Follicular cells (C-cells). These are cells, which do not reach the lumen. They produce calcitonin. When the cells are inactive, they tend to be low cuboidal to squamous type, but when actively secreting hormones, they become more columnar.

Objectives: To determine TFTs and fasting and random blood glucose and to study co-relation of hypo and hyper thyroidism with Diabetes Mellitus.

Results: Mean age of the patients was 38.26±13.157 ranging from 13 to 75 years and mean T3 was 1.6120±0.84991 ranging from 0.50 to 3.80, mean TSH was 5.2055±13.47513 ranging from 0.10 to 88.00, mean T4 was 11.1294±5.84739, mean blood glucose (F) was 82.01±18.504 ranging from 70 to 208 and mean (R) was 112.59±35.373 ranging from 90 to 398. Among TFTs, 121(60.5%) were normal patients and 29(14.5%) had Hypo-thyroidism and the remaining 50 (25.0%) had Hyper-thyroidism out of the total 200 patients Out of total 6 diabetic patients, 3 had normal TSH and 3 had Hypo-thyroidism, while no patients had Hyper-thyroidism, while 194 non-diabetic patients, 118 had normal TSH, 50 had Hyper-thyroidism and 26 had Hypo-thyroidism out of total 200 patients. Out of 6 diabetic patients, 3 had normal T4 and 3 had Hypo-thyroidism, while no patients had Hyper-thyroidism, while 194 non-diabetic patients, 119 had normal T4, 52 had Hyper-thyroidism and 23 had Hypo-thyroidism out of total 200 patients. Out of total 6 diabetic patients, 3 had normal T3 and 3 had Hypo-thyroidism, while no patients had Hyper-thyroidism, while 194 non-diabetic patients, 131 had normal T3, 47 had Hyper-thyroidism and 16 had Hypo-thyroidism out of total 200 patients.

Conclusion: There was no correlation found in our study between diabetic and thyroid patients as well. Further studies may be conducted on the same topic increasing the sample size of the study to evaluate the correlation between the outcomes.

Key Words: thyroid profile, Co-relation, different blood glucose levels.

INTRODUCTION

The thyroid gland, or simply the thyroid in vertebrate anatomy, is one of the largest endocrine glands and consists of two connected lobes. The thyroid gland is found in the neck, below the thyroid cartilage (which forms the laryngeal prominence or "Adam's apple"). The thyroid gland controls how quickly the body uses energy, makes proteins, and controls how sensitive the body is to other hormones. It participates in these processes by producing thyroid hormones, the principal ones being tri-iodothyronine (T3) and thyroxine (sometimes referred to as tetra-iodothyronine (T4).[1] These hormones regulate the growth and rate of function of many other systems in the body. T3 and T4 are synthesized from iodine and tyrosine. The thyroid also produces calcitonin, which plays a role in calcium homeostasis.[2]

Beta blockers are used to decrease symptoms of hyperthyroidism such as increased heart rate, tremors, anxiety and heart palpitations, and anti-thyroid drugs are used to decrease the production of thyroid hormones, in particular, in the case of Graves' disease. These medications take several months to take full effect and have side-effects such as skin rash or a drop in white blood cell count, which decreases the ability of the body to fight off infections. These drugs involve frequent dosing (often one pill every 8 hours) and often require frequent doctor visits and blood tests to monitor the treatment, and may sometimes lose effectiveness over time.[3] Due to the side-effects and inconvenience of such drug regimens, some patients choose to undergo radioactive iodine-131 treatment. Radioactive iodine is administered in order to destroy a portion of or the entire thyroid gland, since the radioactive iodine is selectively taken up by the gland and gradually destroys the cells of the gland. Alternatively, the gland may be partially

[1] Department of Biochemistry, Nowshera Medical College, Nowshera
[2] Department of Biochemistry, Ayub Medical College, Abbottabad

Address for correspondence:
Dr. Zahid Irfan Marwat
Assistant Professor
Department of Biochemistry, Nowshera Medical College, Nowshera
Cell: 0300-9112880
Email: drzahidirfan@gmail.com

or entirely removed surgically, though iodine treatment is usually preferred since the surgery is invasive and carries a risk of damage to the parathyroid glands or the nerves controlling the vocal cords. If the entire thyroid gland is remove, hypothyroidism results.[4]

Hypothyroidism is the underproduction of the thyroid hormones T3 and T4. Hypothyroid disorders may occur as a result of congenital thyroid abnormalities (Thyroid deficiency at birth, autoimmune disorders such as Hashimoto's thyroiditis, iodine deficiency (more likely in poorer countries) or the removal of the thyroid following surgery to treat severe hyperthyroidism and/or thyroid cancer.[5]

Both these disorders are the most vital endocrinological disorders in people coming for treatment. Both of them equally affect each other and a significant relationship has been reported between them.[6,7] It has been observed that T3 and T4 help in the control of breakdown of carbohydrate and pancreatic gland function, while DM has significant effect on TFTs. This relationship explains that how important is to point out the mutual link between these disorders which will help the practitioners in proper management. There are various mechanism through which Thyroid hormones affect glucose metabolism. Hyper function of thyroid gland has been pointed out to cause increase in blood glucose level.[8] When T3 & T4 level is increased, usually the effect of insulin is decreased.[9,10]

The higher danger of retinal problem and renal problem is seen in people of diabetes with hypothyroidism which is not seen clinically gives proof that patients with diabetes should be tested for dysfunctional thyroid gland and curing while found. Some research showed that even minor dysfunctions of thyroid are more common and may be more hazardous in patients with diabetes. Internationally 110 countries have been obvious with patients of the thyroid disease and about 1.6 billion population is in danger. In this country about 8 million people have deficiency of iodine. The aim of this research report is to find out the occurrence of hypo and hyperthyroidism in the community of Nowshera district, KPK, and also to investigate hidden link between thyroid diseases and DM.

MATERIAL AND METHODS

Blood samples were collected randomly form the population of District Nowshera for research purpose. Nearly two hundred samples were collected from DHQ Hospital Nowshera and Qazi Hussain Ahmed Medical Complex Nowshera for further investigation of TSH, T3, T4 and Blood glucose.

The ELISA test for TSH is on the basis of the standard of solid stage ELISA. "The test schemes utilize a distinctive monoclonal antibody bound against a different antigen on the unbroken TSH molecule. Mouse monoclonal anti-TSH antibodies have been used for solid stage restriction (on the microtiter wells). Goat anti-TSH antibody enzymes (horse reddish peroxidase) conjugate solution". The collected test has been permitted to act in response among the two antibodies at the same time. As a result the TSH molecules being squeezed in between the solid stage and enzyme linked antibodies. After hour incubation at room temperature, the wells have been rinsed with water to eliminate liberated antibodies. A blue color is formed when substrate solution has been added and incubated at room temperature in dark for 20 minutes. The color formation has been prevented with the adding up of stop solution, the color turn out to be yellow. The concentration of TSH is directly related to the intensity of color. The absorbance has been measured on spectrophotometer at 450nm wave length.

Computed the mean absorbance with known value of six reference standards and compared with

Table 1: Frequency distribution of TFTs among the collected samples

S. No.	TFTs	Frequency	Percent
1.	Normal	121	60.5
2.	Hypo-Thyroidism	29	14.5
3.	Hyper-Thyroidism	50	25.0
4.	Total	200	100.0

Table 2: Frequency distribution of TSH among the collected samples

S. No.	TSH among TFTs	Frequency	Percent
1.	Normal	121	60.5
2.	Hyper Thyroidism	50	25.0
3.	Hypo Thyroidism	29	14.5
4.	Total	200	100.0

Table 3: Frequency distribution of T3 among TFTs in collected samples

S. No.	T3 among TFTs	Frequency	Percent
1.	Normal	134	67.0
2.	Hypo Thyroidism	19	9.5
3.	Hyper Thyroidism	47	23.5
4.	Total	200	100.0

Table 4: Frequency distribution of T4among TFTs in collected samples

S. No.	T4 among TFTs	Frequency	Percent
1.	Normal	122	61.0
2.	Hypo Thyroidism	26	13.0
3.	Hyper Thyroidism	52	26.0
4.	Total	200	100.0

Table 5: Frequency distribution of Diabetes to TSH among the collected samples

Diabetic	Total No. of Patients	TSH Group			Total
		Normal	Hypo Thyroidism	Hyper Thyroidism	
Yes	200	3	0	3	6
No	200	118	50	26	194
Total	200	121	50	29	200
P-value		0.030			
Chi-Square		7.035			

Table 6: Frequency distribution of Diabetes to T4 among the collected samples

Diabetic	Total No. of Patients	T4 Group			Total
		Normal	Hypo Thyroidism	Hyper Thyroidism	
Yes	200	3	3	0	6
No	200	119	23	52	194
Total	200	122	26	52	200
P-value		0.030			
Chi-Square		7.035			

Table 7: Frequency distribution of Diabetes to T3 among the collected samples

Diabetic	Total No. of Patients	T3 Group			Total
		Normal	Hypo Thyroidism	Hyper Thyroidism	
Yes	200	3	3	0	6
No	200	131	16	47	194
Total	200	134	19	47	200
P-value		0.002			
Chi-Square		12.400			

patient samples. Used the mean absorbance value for each specimen to decide the known values of TSH in μIU/mL from the standard curve and mean absorbance value for each specimen to decide the known concentration of T3 in ng/mL from the standard curve and mean absorbance value for each specimen to decide the known concentration of T4 in ng/mL from the standard curve.

RESULTS

A sample of 200 patients was collected from DHQ Hospital Nowshera and Qazi Hussain Ahmed Medical Complex, Nowshera. Blood sample of the selected patients was taken to determine the TFTs and fasting and random blood glucose level and to know the relationship of hypo and hyper thyroidism with Diabetic and Non-diabetic patients.

Mean age of the patients was 38.26±13.157 ranging from 13 to 75 years and mean T3 was 1.6120±0.84991 ranging from 0.50 to 3.80, mean TSH was 5.2055±13.47513 ranging from 0.10 to 88.00, mean T4 was 11.1294±5.84739, mean blood glucose (F) was 82.01±18.504 ranging from 70 to 208 and mean (R) was 112.59±35.373 ranging from 90 to 398. According to the frequency of gender of the patients, Male patients was 33 (16.5%) while the female patients was 167 (83.5%). According to the frequency of diabetic patients, 6 (3.0%) patients had diabetes while the other 194 (97.0%) were non-diabetic. According the History of TFTs, 121(60.5%) was normal patients and 29(14.5%) had Hypo-thyroidism and the remaining 50(25.0%) had Hyper-thyroidism (Table 1). According the frequency of TSH among TFTs, 121(60.5%) was normal patients and 29(14.5%) had Hypo-thyroidism and the remaining 50(25.0%) had Hyper-thyroidism (Table 2). According the frequency of T3 among TFTs, 134(67.0%) was normal patients and 19(9.5%) had Hypo-thyroidism and the remaining 47(23.5%) had Hyper-thyroidism (Table 3). According the frequency of T4 among TFTs, 122(61.0%) was normal patients and 26(13.0%) had Hypo-thyroidism and the remaining 52(26.0%) had Hyper-thyroidism (Table 4).

Out of total 6 diabetic patients, 3 had normal TSH and 3 had Hypo-thyroidism, while no patients had Hyper-thyroidism, while 194 non-diabetic patients, 118 had normal TSH, 50 had Hyper-thyroidism and 26 had Hy-

po-thyroidism (Table 5). Out of total 6 diabetic patients, 3 had normal T4 and 3 had Hypo-thyroidism, while no patients had Hyper-thyroidism, while 194 non-diabetic patients, 119 had normal T4, 52 had Hyper-thyroidism and 23 had Hypo-thyroidism (Table 6). Out of total 6 diabetic patients, 3 had normal T3 and 3 had Hypo-thyroidism, while no patients had Hyper-thyroidism, while 194 non-diabetic patients, 131 had normal T3, 47 had Hyper-thyroidism and 16 had Hypo-thyroidism (Table 7).

DISCUSSION

In this study a sample of 200 patients was collected from DHQ Hospital Nowshera and Qazi Hussain Ahmed Medical Complex Nowshera. Blood sample of the selected patients was taken to determine the TFTs and fasting and random blood glucose level and to know the relationship of hypo and hyper thyroidism with Diabetic and Non-diabetic patients.

In our study total 6 diabetic patients, 3 had normal TSH and 3 had Hypo-thyroidism, while no patients had Hyper-thyroidism, while 194 non-diabetic patients, 118 had normal TSH, 50 had Hyper-thyroidism and 26 had Hypo-thyroidism out of total 200.

In this study total 6 diabetic patients, 3 had normal T4 and 3 had Hypo-thyroidism, while no patients had Hyper-thyroidism, while 194 non-diabetic patients, 119 had normal T4, 52 had Hyper-thyroidism and 23 had Hypo-thyroidism out of total 200. Total 6 diabetic patients, 3 had normal T3 and 3 had Hypo-thyroidism, while no patients had Hyper-thyroidism, while 194 non-diabetic patients, 131 had normal T3, 47 had Hyper-thyroidism and 16 had Hypo-thyroidism out of total 200. No correlation was found in diabetic patients with thyroid in our results.[11-14]

The association of thyroid diseases with DM is considered by a complicated symbiotic interaction. The resistance to insulin may cause thyroid gland nodularity and parallel DM may boost threat of loss of vision in patients with Grave's disease.[15-17] Hyperthyroidism harms glycemic control in diabetic patients, while hypothyroidism may increase vulner ability to hypoglycemias thus confusing diabetes control.[18] Moreover, thyroid hormones can additionally effect metabolism of carbohydrate through its interaction among, adiponectin, leptin and gut hormones especially ghrelin. Though, the association and the consequential changes in metabolic effects need additional study.[19] It has been experimental that thyroid malfunctions are more prominant in diabetic patients particularly type 1 DM. Also, it looks like that undiagnosed thyroid dysfunction has negative impact on diabetes and its complications.[20-22]

A greater frequency of visual problem and renal problem was seen in diabetic patients with subclinical hypothyroidism. Therefore, treatment of subclinical nature hypothyroidism in diabetic patients will be more advantageous. We determine that an efficient approach to thyroid testing in diabetic patients is acceptable; however, no complete strategies exist about screening for thyroid disorder in diabetic patients. Finally, whether patients with subclinical nature thyroid disease should be investigated for DM and its complications or whether all patients with diabetes should be screened for thyroid function test.[23]

CONCLUSION

There was no correlation found in our study between diabetic and thyroid patients as well. Further studies may be conducted on the same topic increasing the sample size of the study to evaluate the correlation between the outcomes.

REFERENCES

1. Bhattacharyya A, Wiles PG. Diabetic ketoacidosis precipitated by thyrotoxicosis. Postgraduate Medical Journal, 1999;75:883, 291–2.
2. Cettour RP, Theander-Carrillo C, Asensio C. Hypothyroidism in rats decreases peripheral glucose utilisation, a defect partially corrected by central leptin infusion, Diabetologia 2005;48:(4):624–33.
3. Chen HS, Wu TEJ, Jap TS. Subclinical hypothyroidism is a risk factor for nephropathy and cardiovascular diseases in Type 2 diabetic patients, Diabetic Medicine, 2007;24, (12):1336–44.
4. Gray RS, Irvine WJ, Clarke BF, Screening for thyroid dysfunction in diabetics. British medical journal 1979; 2 (6202):1439-79.
5. Hollander JGD, Wulkan RW, Mantel MJ, Berghout A. Correlation between severity of thyroid dysfunction and renal function, Clinical Endocrinology, 2005;62(4):423–7.
6. Maxon HR, Kreines KW, Goldsmith RE, Knowles HC. Long-term observations of glucose tolerance in thyrotoxic patients," Archives of Internal Medicine, 1975;135 (11):1477–80.
7. Meara NM, Blackman JD, Sturis J, Polonsky KS. Alterations in the kinetics of C-peptide and insulin secretion in hyperthyroidism, Journal of Clinical Endocrinology and Metabolism. 1993;76(1):79–84.
8. Dimitriadis G, Baker B, Marsh H. Effect of thyroid hormone excess on action, secretion, and metabolism of insulin in humans. The American journal of physiology,1985;248(5):593–601.
9. Kim DW, Jung SL, Baek JH. The prevalence and features of thyroid pyramidal lobe, accessory thyroid, and ectopic thyroid as assessed by computed tomography: a multicenter study. 2013; 1: 84-91
10. Beer SF, Parr JH, Temple RC, Hales CN. The effect of thyroid disease on proinsulin and C-peptide levels," Clinical Endocrinology. 1989; 30(4):379–83.
11. Kitabchi AE, Umpierrez GE, Miles JM, Fisher JN. Hyperglycemic crises in adult patients with diabetes. Diabetes Care 2009;32 (7): 1335–43.

12. Bech KP, Eldrup E. β-Cell function and glucose and lipid oxidation in Graves' disease. Damsbo Clinical Endocrinology,1996; 44(1), 59–66.

13. Lai Y, Wang J, Jiang F. The relationship between serum thyrotropin and components of metabolic syndrome," Endocrine Journal 2011;58(1):23–30.

14. Levin RJ, Smyth DH. The effect of the thyroid gland on intestinal absorption of hexoses. The Journal of physiology. 1963; 169:755–69.

15. Maratou E, Hadjidakis DJ, Kollias A. Studies of insulin resistance in patients with clinical and subclinical hypothyroidism. European Journal of Endocrinology. 2009; 160(5):785–90.

16. Maxon HR, Kreines KW, Goldsmith RE, Knowles HC. Long-term observations of glucose tolerance in thyrotoxic patients. Archives of Internal Medicine. 1975;135(11):1477–80.

17. Miki N, Ono M, Hizuka N, Aoki T, Demura H, Thyroid hormone modulation of the hypothalamic growth hormone (GH)- releasing factor-pituitary GH axis in the rat. Journal of Clinical Investigation. 1992;90(1):113–120.

18. Mokuno T, Uchimura K, Hayashi R. Glucose transporter 2 concentrations in hyper- and hypothyroid rat livers. Journal of Endocrinology. 1999; 160(2) 285–9.

19. Okajima F, Ui M. Metabolism of glucose in hyper- and hypo-thyroid rats in vivo. Glucose-turnover values and futile-cycle activities obtained with 14C- and 3H-labelled glucose. Biochemical Journal. 1979; 182(2):565–75.

20. Rochon C, Tauveron I, Dejax C. Response of glucose disposal to hyperinsulinaemia in human hypothyroidism and hyperthyroidism. Clinical Science. 2003;104(1):7–15.

21. Sestoft L, Christensen NJ, Saltin B, Responses of glucose and glucoregulatory hormones to exercise in thyrotoxic and myxoedematous patients before and after 3 months of treatment. Clinical Science. 1991;81(1):91–99.

22. Singer MA, Of mice and men and elephants: metabolic rate sets glomerular filtration rate, American Journal of Kidney Diseases 2001;37;1:164–78.

23. Mirella H, Mira S, Zantout, Sami T. Thyroid disorders and diabetes mellitus. 2011; 4 :3