Biochemical Changes of Experimentally Thyrotoxicosis Induced in Rats

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Abstract

Thyrotoxicosis is hypermetabolic clinical syndrome resulting from elevations in thyroid hormonal level, especially free thyroxin (T_4), triodothyronine (T_3), or both. This study was applied to investigate the different metabolic effect of experimental thyrotoxicosis on one hundred white male albino rats. Rats were divided into two main groups. The first one was the control, comprised 30 rats, receive no drugs and experimental group included 70 rats received L-thyroxine (50 mg/kg B.W) daily for 4 weeks. Blood samples for serum separation were collected from all animal groups four times at 1, 2, 3, 4 weeks from onset of thyrotoxicosis induction. The obtained results revealed that an increased in thyroglobulin- antibody, thyroid peroxidase antibody, calcium and sodium, urea, uric acid and LDL-c concentration. Also ALT, AST, alkaline phosphates, LDH, CPK and Ck- MB activity were significantly increased. On the other hand, serum potassium, phosphorus, magnesium, serum cholesterol and high density lipoprotein HDL-c were significantly decrease in all experimental group when compared to control. From the obtained results it could be concluded that thyrotoxicosis causes changes in metabolism of lipids, carbohydrate, minerals and enzymatic activity of the body.

Introduction

Thyrotoxicosis is an increased levels of thyroid hormone led to biochemical and/or clinical signs of excess thyroid hormone at tissue level (5). Increased serum levels of total and free thyroid hormone is not sufficient for the diagnosis of thyrotoxixosis for instance, in resistance to thyroid hormone, increased thyroid hormone levels are found leading to thyrotoxicosis (6).

Thyrotoxicosis is accompanied by high serum concentration of thyroid peroxidase antibodies, thyroglobulin antibodies, or both (33). Overt hyperthyrodisim and hypothyrodism represent possible clinical condition characterized by respectively enhanced oxidative metabolism and reduced lipid lipoprotein plasma level (1).

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Thyroid hormones stimulate almost all aspects of carbohydrate metabolism, including enhancement of insulin- dependent, entry of glucose into cells and increased gluconeagensis and glycogenolysis to generate free glucose (**26**). However, it plays an important part in regulation of lipid metabolism by accelerating the endogenous production of cholesterol as well as it is penetration the cells (**13**).

Four mineral Na, K, Ca and Mg are essential to health, excess or deficiencies of any one of the four elements may severely disrupt thyroid function (**38**). Lowering of plasma cholesterol level is presumed to occur because cholesterol excertion or degradation due to effect of thyroid hormone is to enhance the turn over of LDL (**28**).

Moreover, (**7**) reported that, in hyperthyrodism the activity of thyroid hormone on certain molecular pathway in heart and vasculature was accompanied by increased in heart rate, myocardial contractility and oxygen demand.

This study was planed to investigate possible metabolic effect of thyrotoxcosis on different biochemical changes in experimentally induced thyrotoxicosis in rats.

Material and Methods

One hundred white male albino rats, 6-8 weeks old and average body weight (100-150) gm were divided into two groups. The first group composed of 30 rats served as control and the second one consists of 70 rats and injected daily with L. thyroxine I/P at a dose of 50µg/ kg B.W. for 30 days (**41**).

Blood samples were collected from all animal groups from retro orbital plexus at 1, 2, 3 and 4 weeks from onset of throtoxicosis induction. Serum was separated and used directly for determination of T3, T4 and TSH the methods described by (**43; 47 and 35**), respectively.

Also, serum antithyroglobulin and thyroid peroxidase antibodies, sodium, potassium, calcium, inorganic phosphorus, magnesium, uric acid, urea, creatinine, total cholesterol, trigly cerides, ALT & AST, alkaline phosphatase, HDL-C, LDL-C, LDH, creatinine kinase, MB-CK and CK the

methods described by (44), (25), (39), (27), (11) and (12). (10), (24), (14), (97), (46), (32), (19), (20), (2) and (16).

Statistical analysis was carried out using T-test according to (17).

Results and Discussion

The problem of thyrotoxicosis is much interest now days because an elevated concentration of thyroid hormone resulted in excess mortality from increased incidence of circulatory diseases renal nephropathy (15).

Table (1) showed a significant decrease in TSH value. While, T_3, T_4, A-TG and A-Tpo concentration showing a highly significant increase ($P<0.01$) all over the experimental period in experimental thyrotoxicosis induced group when compared to control one.

Table (1): Mean values of serum T_3, T_4, TSH, A-TG and A Tpo (ng/dl) concentration of thyrotoxicosis induced in male rats and their control.

Duration	Parameters / Animal groups	T_3	T_4	TSH	A-TG	A-Tpo
1st week	Control	66.35±1.56	5.36±0.19	2.12±0.072	8.99±0.88	0.46±0.011
	Thyrotoxicosis	138.4±3.53*	8.59±0.28*	1.99±0.64	9.18±0.12	0.49±0.12
2nd week	Control	65.8±1.87	4.99±0.13	1.87±0.53	8.7±0.21	0.5±0.073
	Thyrotoxicosis	180.45±5.7**	12.9±0.58**	1.32±0.51**	9.23±0.3	0.56±0.17
3rd week	Control	66.2±1.86	5.21±0.17	1.99±0.64	8.76±0.25	0.49±0.064
	Thyrotoxicosis	275.2±9.72**	17.13±0.73**	1.02±0.4**	9.5±0.2*	0.67±0.21*
4th week	Control	66.7±1.12	5.43±0.21	2.21±0.093	9.1±0.29	0.43±0.081
	Thyrotoxicosis	314.55±9.8***	18.28±0.97**	0.875±0.02***	9.56±0.113*	0.73±0.09**

* Significant difference at P> 0.05 ** High significant difference at P > 0.01
*** Very high significant difference at P > 0.001

Table (2): Mean values of serum Na, K (nmol/L), Ca, P, Mg, Uric acid, Urea and Creatinine (mg/dl) concentrations of thyrotoxicosis induced rats and their control.

Duration	Parameters / Animal groups	Na	K	Ca	P	Mg	Uric acid	Urea	Creatinin
1st week	Control	91.0 ± 1.61	20.79 ± 0.89	7.69 ± 0.17	10.42± 0.73	1.035± 0.09	3.21± 0.14	35.89± 1.48	0.79± 0.03
	Thyrotoxicosis	105.01 ± 2.3	19.45 ± 0.63	7.99 ± 0.36	9.72±0.36	1.01± 0.063	3.315± 0.13	39.4± 0.05	0.785± 0.01
2nd week	Control	90.7 ± 1.83	20.1 ± 0.45	7.28 ± 0.19	10.13± 0.65	1.04± 0.076	3.16± 0.17	33.76± 1.67	0.73± 0.06
	Thyrotoxicosis	113.0 ± 2.8	18.1 ± 0.62*	8.25 ± 0.39	8.95± 0.54	0.93 ± 0.068	3.83± 0.17	43.3± 0.08	0.78± 0.02
3rd week	Control	92.1 ± 1.74	21.3 ± 0.67	7.47 ± 0.32	9.98± 0.32	1.1± 0.12	3.42± 0.18	36.1± 1.33	0.8± 0.07
	Thyrotoxicosis	121.0 ± 2.1**	17.6 ± 0.57**	8.5 ± 0.43*	8.21± 0.65*	0.74± 0.073**	4.32± 0.19	45.9± 0.06*	0.776± 0.04
4th week	Control	91.0 ± 1.67	20.46 ± 0.78	7.53± 0.24	10.53± 0.53	1.087± 0.096	3.27± 0.11	34.56± 1.24	0.77± 0.05
	Thyrotoxicosis	126.4 ± 1.74***	16.55 ± 0.36***	8.55± 0.12*	6.69± 0.73**	0.59± 0.071***	4.43± 0.16*	46.07± 0.03*	0.76± 0.025

Significant difference at P> 0.05
**** High significant difference at P > 0.01**
***** Very high significant difference at P > 0.001**

Table (3) Mean values of serum total cholesterol, HDL-c, LDL-c, Triacylglycerol, concentrations and ALT, AST, Alkaline phosphates, LDH (mg/dl), CK and CK-MB (u/L) activities of thyrotoxicosis induced rats and their control.

Duration	Parameter / Animal group	Total Cholesterol	HDL-c	LDL-c	Tri-acylglycerol	AST	ALT	ALP	LDH	CK	CK. MB
1st week	Control	134.04 ± 2.74	112.74 ± 1.22	7.53 ± 0.19	178.8 ± 4.89	22.04 ± 0.36	90.16 ± 4.34	56.82 ± 5.33	348.24 ± 33.05	47.99 ± 5.25	45.92 ± 2.11
	Thyrotoxicosis	131.9 ± 2.9	103.6 ± 2.1	19.6 ± 2.16	170.9 ± 5.4	23.7 ± 0.76	94.2 ± 3.9*	65.6 ± 2.5	551.0 ± 26.07*	60.3 ± 4.32	48.7 ± 3.32
2nd week	Control	125.6 ± 3.6	115.5 ± 1.62	8.3 ± 0.12	175.6 ± 4.63	21.06 ± 0.54	91.23 ± 2.35	55.9 ± 6.33	326.14 ± 21.9	45.32 ± 4.52	44.94 ± 1.21
	Thyrotoxicosis	126.7 ± 2.9	99.5 ± 2.6*	26.1 ± 3.32*	148.8 ± 5.2	25.6 ± 0.68	96.9 ± 4.7	69.2 ± 2.9	736.0 ± 25.13**	70.6 ± 4.52	51.7 ± 2.65
3rd week	Control	129.7 ± 2.92	110.9 ± 1.74	7.23 ± 0.18	179.3 ± 4.9	21.9 ± 0.61	90.8 ± 2.64	56.1 ± 5.94	338.35 ± 19.1	46.15 ± 4.32	46.8 ± 3.12
	Thyrotoxicosis	123.3 ± 3.1*	90.8 ± 2.3*	38.5 ± 4.19***	119.3 ± 3.6*	26.9 ± 0.72*	101.7 ± 5.6*	78.0 ± 0.91	867.0 ± 21.45**	81.0 ± 5.45**	53.0 ± 2.89*
4th week	Control	135.6 ± 2.53	116.0 ± 1.9	7.4 ± 0.11	174.2 ± 4.96	23.0 ± 0.75	88.98 ± 3.57	57.0 ± 6.45	352.0 ± 16.3	47.6 ± 5.1	45.31 ± 1.65
	Thyrotoxicosis	118.07 ± 3.74*	79.64 ± 2.9**	54.26 ± 5.27***	90.63 ± 4.03**	27.32 ± 1.74*	102.44 ± 3.8*	84.4 ± 0.63**	959.1 ± 8.7***	87.31 ± 3.23**	56.7 ± 0.65*

Significant difference at P> 0.05
**** High significant difference at P > 0.01**
***** Very high significant difference at P > 0.001**

This increase was similar to that obtained by **(48)** who explained that the patients with autoimmune thyroid diseases have immune reactivity, both antibodies and cell- mediated immunity, directed to the TSH receptor, thyroid peroxidase and thyroglobulin.

Table (2) revealed that there was significant increase in serum Na, Ca, urea and uric acid concentrations while K, Mg and Ph showed a significant decrease in thyrotoxicosis induced rats as compared to control group.

A significant increase of calcium agree with **(3)** who attributed that bone formation as well as destruction are proceeding at higher rates in thyrotoxicosis as well as . Expression of receptors to extra cellular calcium enables parafollicular cells of the thyroid gland to release calcitonin and serotonin in response to increased external Ca^{2+}.

Moreover, significant increase of Na that may be attributed to that hyperthyroidism has a tendency to sodium retension with decreased excretion which leads to high blood pressure **(34)**. Also, **(42)** explained these results due to decrease in the filtered load of Na and increase tubular reabsorpation.

On the other hand significant decrease of K agreed with **(4)** who recorded changes in electrolytes in tissue which alter the effect of thyroid hormone on NA/K-ATPase were explained by **(23)** who reported that hypokalomia result from intracellular shift of K induced by thyroid hormone sensitization of Na-K- ATPase rather than depletion of total body K.

Regarding renal function, significant increase of urea uric acid agreed with **(36)** which may be consequence of intensified metabolism and catabolism, increased purine synthesis and or increased degradation of purine nucleotides and increased response of organism to oxidative stress with mobilization of protective antioxidant mechanism.

Table (3) revealed significant decrease in serum total cholesterol, HDL-c and triacydeglycerol concentration despite LDL-c showed a significant increase in thyrotoxicosis induced rats when compared with control group.

Moreover, serum AST, ALT, alkaline phosphatase, LDH- Ck- MB and CK activities showed a significant increase in thyrotoxic group as compared to control.

The significant decrease in lipid profile are agreed with **(21)** who revealed that the hypocholestermic effect of thyroid hormone may be mediated by primary effect on cholesterol 7 alpha hydroxylase gene expression as well as cholesterol excretion and degradation is enhanced more than cholesterol synthesis **(37)**.

Moreover, the decreases of triacylglycrol agreed with **(29)** who concluded that accelerated removal of plasma triglycerides in thyrotoxicosis could be accounted by an increased activity of lipoprotein lipase or by an increased rate of blood flow at active removal sites or both.

However, decreased serum HDL-C concentration. May be due to increased catabolic rate for the cholesterol in general **(8)** on the other hand increased level of LDL attributed to decreased cellular receptor number and consequent reduced removal of LDL from plasma **(45).**

The significant increases in serum AST, ALT and Alkaline phosphatase were agreed with **(18)** who found significant increase in AST, ALT of thyrotoxic patient although the majority of these patients showed no other clinical or biochemical feature of liver impairment .

The increase of this liver enzyme may be attributed to hyper-plasia of kupffer cell with extensive liver necrosis **(22).**

Thyrotoxicosis have clinical features that reflect the effects of excess thyroid hormone on the cardiovascular system **(31)** these data are agreed with our result as thyrotoxicosis caused significant increase of heart function tests as LDH, CK, and CK-MB which similar to result of myocardial infarction as small mass of damaged tissue break down-hemolysis which is sufficiently sever and produce increase of Ck, Ck-MB & LDH **(40)**.

So we concluded by periodical examination analysis to thyroid function at appearance of signs on the body activity due to its large effecting on body parameters and organs.

References

1. Asayama, K and Kato, K. (1990): Oxidative musclar injury and its relevance to hyperthyrodism. Free Radic. Biol. Med.; 8: 293-303.

2. Bais, R. and Crit, R. E. V. (1982): Determination of Ck isoenzymes activity of cardiat tissue .Clin. Lab. Sci., 18: 291.

3. Begic- Karup, S. Wagner, B. and Raber, W. (2001): Serum calcium in thyroid disease. Wein Klin Wochen Schr. 15; 113 (1-2): 65-8.

4. Change- Husum Hseieh, Shi- wen Kuo, and Ji-hen Hung (2004): Thyrotoxic periodic paralysis. Ann Saudi Med. 24(6).

5. Chopra, I. J. and Baber, K. (2001): Use of oral cholecystographic agents in the treatment of amiodarone- induced hyperthyrodism. J. Clin. Endocrinol Metal 86:4707-4710.

6. Ciccarelli, A.; Valdes- Socin, H.; Parma, J.; Khoo, S.K.; Schoumaus, J.; Colao, A.; Hamoir, E. and Beckers A. (2004): Thyrotoxic adenoma followed byatypical hyperthyrodism due to struma ovarii: clinical and genetic studies. Eur J. Endocrinal. Apr.; 150 (4): 431-7.

7. Davis, P. J. and Davis, F. B. (1996): Nongenomic action of thyroid hormone. Thyroid; 6: 497-504.

8. De Lacruz, J. P. Quinter, L. Villalobos, M. A.; and Sanchez de la cueta, F. (2000): Lipid peroxidation and glutathione system in hyperlipidemic rabbits: influence of olive oil administration: Biochem. Biophys. Acta, 1485, 36-44.

9. Finely, P. R.; Schifmou, R. B.; William, R. J. and Lichti, D. A. (1978): Cholesterol in high- densitylipo protein use of mg^{2+}/ destran sulfate in its enzymatic measurement, clinichem 24:931-933.

10. Gochman, N. and Schmitz, J. M. (1971): Determination of serum uric acid concentration. Clin. Chem. 17: 1154.

11. Guider, W.; Itoffman, G. and Oppitz, K. (1982): A normal base izhkinischer befunde in den staditischer krankenhausen. Miinchenscited in Biochemicaljit.

12. Henery, J. B. (1984): Clinical diagnosis and management, 17th ed., W.B. Saunder Co., Philadephia, P. 157.

13. Hyalmar Gullberg, Mats Rudling, Carmen Salto, Douglas Forrest, Boangllin and Bjorn Vennström (2002): Requirements for thyroid hormone receptor B in T_3 Regulation of cholesterol metabolism in mice. Molecular Endocrinology, 16 (8): 1767-1777.

14. Jaffée, M. (1986): Ueber den Niederschlag. Welchen pikrinsaure in normalem Harn erzeugt und über eine neue Reaktion des kreatinins. Z physiol. Chem., 10: 391-400.

15. Jayaprasad, N.; Johnson, MD.; Francis, M. D. (2005): Atrial fibrillation and hyperthyrodism. Indian pacing and Electrophysiology Journal (ISSN 0972-6292), 5 (4): 305-311.

16. Kaehamr, J. F. and Moss, D. W. (1976): In "Fundamentals of clinical chemistry" Tietz N. W. ed. Saunders, W. B. Co., Philadlephia, 686.
17. Kempthorn, O. (1969): The design and analysis of experiments; john wiley and sonsine (editor), New York, 47: 96.
18. Kim, H. J.; Kim, B. H.; Han, Y. S.; Yang, I; Dong, S. H.l Chang, Y. W.; Lee, J. I. And Change, R. (2001): The incidence and clinical characteristics of symptomatic propylthiouracil- induced hepatic injury in patients with hyperthyroidism: a single- center retrospective study. Am J. Gastroenterol : 96: 165-9.
19. Kind, P. R. N. and King, E. J. (1954): J. Clin. Path, 7:322.
20. Kreutzer, H. H. (1964): Clin. Chem. Acta. 9:64.
21. Lein, T. F. and Horng Y. M. (2003): The effect of supplementary dietary L-carnitine on the growth performance, serum component, concase traits and enzyme activities in relation to fatty acid B-oxidation of broiler chicken. British. Poultry Science 42(1): 92-95.
22. Levy, M. (1993): Propylthiouracil hapatotoxicity: a review and case presentation. Clin pediatr cphilal; 32: 25-9.
23. Lien Lam, D. O.; Rajasree, J. Nair, M. D. and Leslie Tringle, M. D. (2006): Thyrotoxic periodic paralysis. Proc. (Bayl Univ. Med. Cent.); 19:126-129.
24. Mackay, E. M. and Mackay, L. L. (1927): The concentration of urea in the blood of normal induitials. J. CLin. Invest. 4:295.
25. Maruna, R. F. (1958): Clin. Chem. Acta, 2 : 581.
26. Michael, Rosenbaum; Jules, Hirsch; Ellen, Murphy and Rudolph, Leibel (2000): Effects of changes in body weight on canbohydrate metabolism, catecholamine excretion, and thyroid function American Journal of clinical Nutrition, Vol. 71, No. 6, 1421-1432.

27. Moorehead, W. R. and Briggs, H. C. (1974): Clin. Chem., 20: 1458.
28. Morton, R. E.; Nunes, V; Izem, L. and Quintao, EC. R. (2001): Markedly elvatated lipid transfer inhibitor protein in hypercholesterolenic subjects in mitigated by plasma Trighlycerde level, Arterioschler. Thromb. Vasc. Biol. 21:1642-1649.
29. Neyyir, T. E. (2003): Usage of cornitine in blood cardioplegia in coronary artery by pass grafting. J. of Ankara- Medical School. 124, 3.
30. Pantazi, H. and Papapetrou, P. D. (2000): J. Clin. Endcrinol. Meta. 6. Mar. 85 (3): 1099-106.
31. Peters, A.; Eblers, M. Blank, B. Exler, D.; Falk, C. Kohlmann, T.; Fruehwald-Schultes, B.; Wrilhoner, P. Kerner, W. and Fehm, H. (2000): Excess Triiodothyronine as a risk factor of coronary events. Arch. Intern. Med. 160: 1993-1999.
32. Reitman, S. and Frankel, S. (1957): Amer. J. Clin. Path. 28-56.
33. Roti, E. and Emerson, C.H. (1992): J. Clin. Endocrinol. Metals. 74:3-5.
34. Sarlis, N. J. and Gourgiotis, L. (2003): Thyroid emergencies. Reviews in Endocrine and Metabolic disorders. 4 : 129-136.
35. Soos, M. and Siddle, K. (1982): J. Immunol. Methods; 51: 57-68.

36. Suher, M.; Koc. E. and Ata, N. (2005): Relation of thyroid days dysfunction, thyroid auto antibodies, and renal function. Ren fail, 27 (6): 739-42.

37. Tan, K. C. B.; Shiu, S. W. M and Kung, A. W. C. (1998): Plasma cholesterol ester transfer protein activity in hyper- and hypothyroidism. J. Clin. Endocrinol. Metab. 83: 140-143.

38. Tanwani, L. K.; Lohano, V.; Broadstone, V. L. and Mokshagundam. S. P. (2002): Minimal change nephropathy and Grav's disease: report of a case and review of the literature. Endocrine practice 840-43.

39. Terri, A. E. and Sesin, P. G. (1958): Am. J. Clin. Path., 29: 86.

40. Uday, Kumar; Anand, Sharan. And Schaheena, Kamal (2003): Raised serum lactate dehydrogenase associated with gangrenous small bowel volvulus. Indian Journal of clinical Biochemistry, 18(2): 6-7.

41. Valeria safa- Tisseront, Pascal Ponchon, Dominique Laude and Jean- Lue Elghozi (1998): Autonomic contribution to the blood pressure and heart rate variability changes in early experimental hyperthyroidism. Journal of hypertension, 16: 1989-1992.

42. Vargas, F.; Atucha, N.; Sabio, J. M.; Quesada, T. and Garcia- Estan, J. (1994): Pressure - diuresis natriuresis response in hyper- and hyper thyroid rats. Clinical sciences 87: 323-328.

43. Walker, W. H. (1977): Introduction: An Approach to Immunoassay. Clin. Chem. 23:384.

44. Wall, J. R. and T. Kuroki (1985): Immunologial factors in thyroid disease. Medical nics of North America 69:913.

45. Walzem, R. L.; Watkinss; Frankel, E. N.; Hansen, R. J. and German, J. B. (1995): Older plasma lipoproteins are more susceptible to oxidation: a linking mechanism for the lipid and oxidation theories of atherosclerosis cardiovascular diseases. Proc. Natl. Acad. Sci. USA.; 92: 7460-7464.

46. Warnick, G. and Tussell, D. (1995): National cholesterol Education program Recommendations for measurement of High density lipoprotein cholesterol, Executive summary, clinical chemistry, 41, 10.

47. Wistom, G. B. (1976): Enzyme- Immunoassay. Clin. Chem. 22: 1242.

48. Yaron Tomer and Terry F. Davis. (2003): Searching for the autoimmune thyroid disease susceptibility genes: From gene mapping to gene function.

التغيرات الكيميائية الحيوية في حالات التسمم الدموي لزيادة هرمون الثيروكسين المحدث تجريبا في الجرازن

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الملخص العربي

أجريت هذه الدراسة للوقوف علي مدى تأثير هرمون الثيروكسين الزائد علي العمليات الحيوية المختلفة في الجسم . وقد أجريت علي عدد ١٠٠ من الذكور الفئران البيضاء والذى تراوح وزنها من ١٠٠-١٥٠جرام قسمت إلي مجموعتين . المجموعة الأولى المجموعة الضابطة والمجموعة الثانية تم حقنها بهرمون الثيروكسين يوميا طوال فترة الدراسة . وتم تجميع العينات أربع مرات بعد أسبوع ثم أسبوعين ثم ثلاثة أسابيع ثم أربعة أسابيع من بداية التجربة. وقد أوضحت الدراسة ما يلي . زيادة معنوية في تركيز أنتى ثيرجلبيولين انتى بودي ، انتى بدواوكسيديز انتى بودي مقارنة بالمجموعة الضابطة . كما لوحظت أيضا زيادة معنوية في تركيز صوديوم والكالسيوم في حين حدث نقص معنوي في تركيز الفوسفور والماغنسيوم . وبالنسبة لتأثير زيادة الهرمون علي الكلى أوضحت زيادة معنوية عالية في حامض البوليك وتركيز اليوريا علي حين حدث نقص معنوي في الكرياتنين أيضا ولوحظ نقص معنوي عالي في تركيز الكلوليسترول الكلى والدهون عالية الكافة والجليسريدات الثلاثية علي حين حدث زيادة معنوية في الدهون منخفضة الكثافة . وكان للكبد تأثير من التسمم الدموي بهذا الهرمون حيث حدث زيادة معنوية في تركيز إنزيم الآلنين لنيوترانسفنيز وإنزيم أسبرتيت امينو تراتسفنيز وإنزيم الفوسفاتيز القاعدي علي عكس الألبيومين أوضح نقص معنوي واضح مقارنة بالمجموعة الضابطة .

أكدت الدراسة زيادة معنوية عالية في تركيز لاكتيت ديهيدروجينيز إنزيم وإنزيم الكرياتنين كيناز وإنزيم الكرياتنين فوسفو كينيز . طبقا لنتائج هذه الدراسة يتضح لنا أن التسمم الدموي بهرمون الثيروكسين يؤدي إلي تغير شامل في كل العمليات الحيوية في الجسم ووظائف الأعضاء المختلفة كالكبد والقلب ولذلك ننصح بالفحص الدورى لهذ الهرمون لتجنب الأثار السيئة التى تنتج عن زيادته.