The Improvements in the Prolactin, Thyroid Hormones and Lipid Profiles that are Observed During Three Pregnancy Periods

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Abstract

This research was to explore improvements in lipid profile, TSH, T3, T4 and prolactin during three stage of the progress. Pregnancy leads to development in both hormones and biochemistries of the maternal blood. The study involved a control group of 20 non-pregnant women and 30 pregnant and. Automatic hormone analyzer[1] and biochemistry automatic analyzer measured hormones, with the lipid profile[2]. Although T3, T4, TC and TG showed no significant increases in first ($p \geq 0.05$) compared with the control group, TC and TG increased significantly in the second phase and third stage of pregnancy trimesters and T3 and T4 significantly in the third phase of pregnancy quarter ($p \geq 0.05$) compared with the control team ($p \geq 0.05$). The results demonstrate a significant increase in prolactin in three stage of pregnancy ($p \geq 0.05$). No significant increase ($p \geq 0.05$) in comparison with control groups was shown in LDL-C and HDL -C over all phases of the prolactin. Increased prolactin, thyroid hormones and the rise in lipid fractions follow normal pregnancy.

Keywords*: LDL-C, HDL-C, TC, TG, T_3, T4, Prolactin, Stage, pregnancy*

Introduction

Pregnancy is the time when one or more offspring evolves inside the womb of a woman. Several gestations, including twins or triplets, may take place during pregnancy. Childbornness usually takes place after the birth about 38 weeks. For women with 4 weeks of a menstrual cycle, it is about 40 weeks after the last regular menstrual period[3]. During the pregnancy, there are great modifications in the physiology of a mother to provide the requisite fetal nutrients and the mother's additional work resources (before fetal needs occur). The first half (up to 13 weeks after conception) starts, when fetus is about 13 g and can be as long as 8 cm. These changes are not recognized. Rapid fetal growth occurs during the second trimester (13 to 26 weeks), and by the end of the second trimester, the fetus weighs around 70 g and is 30 cm long, within which the fetal organs would have begun to develop[4]. The fetal organs complete maturation during the third trimester (26–40 weeks)[5].Pregnancy is affected by a number of factors, including culture, the environment, social and economic circumstances and medical access[6]. Therefore, the prevention of adverse outcomes in pregnancy by identifying causal factors for these outcomes is of clinical and economic importance. Unfavorable outcomes of pregnancy have serious short-term consequences, such as increased perinatal morbidity and mortality of mother and child[7]. Women show an increase in lipid levels during normal pregnancy, including triglycerides (TG) and total cholesterol (TC) at gestational age [8-11].Pregnant complications including hypertension, preterm birth, low birth weight, placental abruption, and fetal death have been linked with clinical thyroid dysphunction. The relation between subclinical and pregnant hypothyroidism has not been well studied. In order to assess the outcome of pregnancy of women with high levels of thyroid thyrotropin (TSH) and normal free thiroxin levels, we carried out the prospective thyroid screening

test [12-15].Maybe prolactin's [PRL] most important traditional role is to encourage milk production in women following childbirth. Increases the concentrations of prolactin during pregnancy contributing to the enlargement and secretion of colostrums in mamma glands following delivery in preparation for breastfeeding [16-18].In this study we investigate the changes in lipids profile, T3, T4, TSH, and prolactin in a group of pregnant women to determine the changing these parameters during normal pregnancy[19-21].

Material & Methods

This study was conducted in AL-Batol Learning Hospital, Diyala Governorate, Iraq from May to October 2018. A total of 50 women aged between 20 and 41 volunteer for the study. This group consisted of 50 volunteers, 30 pregnant women and 20 non-pregnant women. In about the 32 weeks of pregnancy, 30 pregnant women were studied according to pregnancy period. Blood samples from all participating women were taken after having done quick 8-12 hours of blood overnight. Growing woman was collected with 5ml disposable syringes and transferred to the flat tube 5ml of fasting vein blood.

For half an hour, the blood was left to clotted, then centrifuged for 5 minutes at 4000 rpm andused to check the parameters tested. The study was performed by an automated to analyze total cholesterol (TC), triglycerides (TG) and cholesterol (HDL-C) in serum. Hormones were measured by hormones automated analyzer[1]. The Friede Wald method was used to estimate the low density of lipoprotein-cholesterol (LDL-C) = Total cholesterol - (TG/2.2 - HDL-Cholesterol) (mg / dl).The statistical software SPSS data analysis was submitted as a mean ± Standard Deviation (M±SD). The results are presented. The t-test student was used for data analyzes and $p \leq 0.05$ is statistically significant.

Result

Table 1 displays the mean ± standard (M±SD) values for the control blood parameters and pregnant women that studied in three stages.

Table 1: The table illustrates (M ± SD) values of studied parameters in control and the three trimesters of pregnancy.

Parameters	control	1st trimester	2nd trimester	3rd trimester
TSH mIu/l	3.50±1.08	2.90 ± 1.23	3.40±0.69	3.50±1.15
T3 nmol/l	2.00±0.30	3.20 ± 0.44	2.90 ±0.96	2.60±0.47
T4 nmol/l	90.00±12.58	138 ± 22	140 ± 34	150.0±62.31
PRl ug/l	15.00±3.01	34.0 ± 32.7	100.0±33.45	131.1±31.44
TC mg/l	150.4±17.4	166.4±28.1	180.0±23.4	207.3±32.2
TG mg/l	80.7±6.6	108.7±13.1	126.5 ± 18.4	130.7 ± 20.8
HDL-C mg/l	43.1±5.4	44.7±4.2	48.0±6.3	52.0 ± 5.8
LDL-C mg/l	81.8 ± 14.6	90.3 ± 9.6	123.7 ± 10.9	159.5 ± 22.4

As shown in table 1 there has been no significant differences between the blood levels of these parameters, with the exception of prolactin, showing a significant increase in the first quarter when comparing the blood levels of these parameters with control groups ($p \leq 0.05$) [20-21].

Table 2: The relation with control of the studied parameters showed in the first trimester.

Parameters	Control	1st trimester	P-value
TSH	3.50±1.08	2.90±1.23	0.784
T3	2.00±0.30	3.20±0.44	0.234
T4	90.00±12.58	138.0±22.05	0.439
Prolactin	15.00±3.01	34.00±32.75	0.01*
TC	150.4±17.4	166.4±28.1	0.195
TG	80.7±6.6	108.7±13.1	0.125
HDL-C	43.1±5.4	44.7±4.2	0.254
LDL-C	81.8±14.6	90.3±9.6	0.321

The result showed an elevation in the levels of the parameters tested ($p \geq 0.05$), without noticeable difference, with the exception of prolactin, TC and TG, which showed a small elevation ($p \geq 0.05$), of the presence in the second quarter relative to control and TSH showed almost normalization.

Table 3: Comparison of studied parameters with control demonstrated in the second trimester.

Parameters	**Control**	**2nd trimester**	**P-value**
TSH	3.50±1.08	3.40±0.69	0.72
T3	2.00±0.30	2.90±0.96	0.089
T4	90.00±12.58	140.0±34.22	0.077
Prolactin	15.00±3.01	100.0±33.45	0.001*
TC	150.4±17.4	180.0±23.4	0.049*
TG	80.7±6.6	126.5±18.4	0.045*
HDL-C	43.1±5.4	48.0±6.3	0.231
LDL-C	81.8±14.6	123.7±10.9	0.063

The findings from Table3 show that there is a substantial increase of the number of parameters ($p \geq 0.05$) in most of the parameters, except for HDL and LDL rates showing a high presence of the three without significant differences($p \geq 0.05$) , and TSH showed a standardization as shown in Table 3.

Table 4: The Comparison of the parameters measured with control showed in the third trimester.

Parameters	**Control**	**3rd trimester**	**P-value**
TSH	3.50±1.08	3.50±1.15	0.733
T3	2.00±0.30	2.60±0.47	0.023*
T4	90.00±12.58	150.0±62.31	0.017*
Prolactin	15.00±3.01	131.1±31.44	0.001*
TC	150.4±17.4	207.3±32.2	0.042*
TG	80.7±6.6	130.7±20.8	0.044*
HDL-C	43.1±5.4	52.0±5.8	0.09
LDL-C	81.8±14.6	159.5±22.4	0.051

Table 4 explain the result that showed an increase of the number of parameters tested ($p \geq 0.05$), with noticeable difference, with all and only exception of HDL-C and LDL-C, which showed a small elevation ($p \geq 0.05$), of the presence in the third trimester relative to control and TSH showed almost normalization.

Discussion

The results showed an increase in T3 and T4 thyroid hormone levels, especially in the third stage of the trimester in pregnant women. This finding is consistent with the scientific evidence that the thyroid gland states during pregnancy increases and hyper-stimulate thyroid hormone levels, which will improve the basal metabolism rate appropriate for the mother and fetus. Therefore, a thyroid function assessment is important for thyroid hormone change during pregnancy. The predicted normal increase in thyroid hormone levels during pregnancy allows individualized supplementation when required [22-25]. Thyroid stimulation hormone (TSH) concentrations are small with standardization in the second and third trimesters in the first trimester [26-28]. Consequently, during pregnancy the average TSH level is lower or less than normal in contrast to non-pregnancy. TSH values vary with the mother going from the first to the third quarter [29].Prolactin concentration in this study revealed significant elevation in the first trimester when compared to control group and also increased progressively with high significant elevation in second and third stages of pregnancy. This elevation is to preparation and induces the mammalian gland for secretion of milk for feeding the neonate. This result agreement with some previous studies[30].

The current study revealed increases of lipid fractions (TC, TG, HDL-C and LDL-C) [31]. The growth and development of the fetus and support its structures during pregnancy period demand increases the metabolic fuels and lipids are high energy food [32-33].

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Ethical Clearance: The project was approved by the local ethical committee (College of Education for pure science. University of Diyala).

Conflict of Interest - (nil).

References

1. Zec, I.; Kučak, I.; Begčević, I.; Šimundić, A.-M.; Tišlarić-Medenjak, D.; Megla, Ž. B.; Vrkić, N., Reference intervals for reproductive hormones in prepubertal children on the automated Roche cobas e 411 analyzer. Clinical Biochemistry 2012, 45 (15), 1206-1212.
2. Klima, H.; Domke, I., Th-P17: 422 Development and multicenter evaluation of a new HDL-cholesterol reagent on Roche/Hitachi and COBAS Integra® systems. Atherosclerosis Supplements 2006, 7 (3), 586.
3. Mohamed, A. O.; Hamza, K. M.; Babker, A., Physiological changes in some hematological and coagulation profile among Sudanese healthy pregnant women. Int J Med Sci Public Health 2016, 5 (3), 525-8.
4. Oke, O. T.; Awofadeju, S. O.; Oyedeji, S. O., Haemorheological profiles in different trimesters among pregnant women in South West Nigeria. Pakistan Journal of Physiology 2011, 7 (2), 17-20.
5. Lawson, S. N., Morphological and biochemical cell types of sensory neurons. Sensory neurons: diversity, development, and plasticity 1992, 27-59.
6. Purohit, G.; Shah, T.; Harsoda, J., Hematological profile of normal pregnant women in Western India. Sch J Appl Med Sci 2015, 3, 2195-9.
7. Catov, J. M.; Ness, R. B.; Wellons, M. F.; Jacobs, D. R.; Roberts, J. M.; Gunderson, E. P., Prepregnancy lipids related to preterm birth risk: the coronary artery risk development in young adults study. The Journal of Clinical Endocrinology & Metabolism 2010, 95 (8), 3711-3718.
8. Brizzi, P.; Tonolo, G.; Esposito, F.; Puddu, L.; Dessole, S.; Maioli, M.; Milia, S., Lipoprotein metabolism during normal pregnancy. American journal of obstetrics and gynecology 1999, 181 (2), 430-434.
9. Mazurkiewicz, J.; Watts, G.; Warburton, F.; Slavin, B.; Lowy, C.; Koukkou, E., Serum lipids, lipoproteins and apolipoproteins in pregnant non-diabetic patients. Journal of clinical pathology 1994, 47 (8), 728-731.
10. Sattar, N.; Greer, I. A.; Louden, J.; Lindsay, G.; McConnell, M.; Shepherd, J.; Packard, C. J., Lipoprotein subfraction changes in normal

pregnancy: threshold effect of plasma triglyceride on appearance of small, dense low density lipoprotein. The Journal of Clinical Endocrinology & Metabolism 1997, 82 (8), 2483-2491.

11. Ogura, K.; Miyatake, T.; Fukui, O.; Nakamura, T.; Kameda, T.; Yoshino, G., Low-density lipoprotein particle diameter in normal pregnancy and preeclampsia. Journal of atherosclerosis and thrombosis 2002, 9 (1), 42-47.
12. Utiger, R. D., Maternal hypothyroidism and fetal development. Mass Medical Soc: 1999.
13. Xin-Min, J.; Xue-Yi, C.; Ji-Yong, J.; Tai, M.; James, D. W.; Rakeman, M. A.; Zhi-Hong, D.; Mamette, M.; Amette, K.; Ming-Li, Z., Dynamics of environmental supplementation of iodine: four years' experience of iodination of irrigation water in Hotien, Xinjiang, China. Archives of Environmental Health: An International Journal 1997, 52 (6), 399-408.
14. DeLong, G. R.; Stanbury, J. B.; Fierro-Benitez, R., Neurological signs in congenital iodine-deficiency disorder (endemic cretinism). Developmental Medicine & Child Neurology 1985, 27 (3), 317-324.
15. Davis, L. E.; Leveno, K. J.; Cunningham, F. G., Hypothyroidism complicating pregnancy. Obstetrics and Gynecology 1988, 72 (1), 108-112.
16. Gellersen, B.; DiMattia, G. E.; Friesen, H. G.; Bohnet, H. G., Prolactin (PRL) mRNA from human decidua differs from pituitary PRL mRNA but resembles the IM-9-P3 lymphoblast PRL transcript. Molecular and cellular endocrinology 1989, 64 (1), 127-130.
17. Andersen, J. R., Decidual prolactin. Studies of decidual and amniotic prolactin in normal and pathological pregnancy. Danish medical bulletin 1990, 37 (2), 154.
18. Ben-Jonathan, N.; Mershon, J. L.; Allen, D. L.; Steinmetz, R. W., Extrapituitary prolactin: distribution, regulation, functions, and clinical aspects. Endocrine reviews 1996, 17 (6), 639-669.
19. Pałubska, S.; Adamiak-Godlewska, A.; Winkler, I.; Romanek-Piva, K.; Rechberger, T.; Gogacz, M., Hyperprolactinaemia - a problem in patients from the reproductive period to the menopause. Prz Menopauzalny 2017, 16 (1), 1-7.
20. Skałba, P. H., Diagnostyka i leczenie zaburzeń endokrynologicznych w ginekologii. Medycyna Praktyczna: 2014.
21. Karasek,M.;Pawlikowski,M.,Hyperprolactinemia–the Essentials. European Endocrinology 2006, 1, 53-57.
22. Glinoer, D., What happens to the normal thyroid during pregnancy? Thyroid 1999, 9 (7), 631-635.
23. Mircheff, A.; Wang, Y.; Nakamura, T.; Ding, C.; Schechter, J., Enhanced lacrimal gland mucosal immune function associated with pregnancy. Investigative Ophthalmology & Visual Science 2006, 47 (13), 1944-1944.
24. Taylor, P. N.; Minassian, C.; Rehman, A.; Iqbal, A.; Draman, M. S.; Hamilton, W.; Dunlop, D.; Robinson, A.; Vaidya, B.; Lazarus, J. H., TSH levels and risk of miscarriage in women on long-term levothyroxine: a community-based study. The Journal of Clinical Endocrinology & Metabolism 2014, 99 (10), 3895-3902.
25. Alijotas-Reig, J.; Garrido-Gimenez, C., Current concepts and new trends in the diagnosis and management of recurrent miscarriage. Obstetrical & gynecological survey 2013, 68 (6), 445-466.
26. Frederiksen, M. C., Physiologic changes in pregnancy and their effect on drug disposition. Seminars in Perinatology 2001, 25 (3), 120-123.
27. Zarghami, N.; Rohbani-Noubar, M.; Khosrowbeygi, A., Thyroid hormones status during pregnancy in normal Iranian women. Indian Journal of Clinical Biochemistry 2005, 20 (2), 182-185.
28. Sheehan, P. M.; Nankervis, A.; Araujo Júnior, E.; Da Silva Costa, F., Maternal thyroid disease and preterm birth: systematic review and meta-analysis. The Journal of Clinical Endocrinology & Metabolism 2015, 100 (11), 4325-4331.
29. Klein, R.; Haddow, J.; Falx, J.; Brown, R.; Hermos, R.; Pulkkinen, A.; Mitchell, M., Prevalence of thyroid deficiency in pregnant women. Clinical endocrinology 1991, 35 (1), 41-46.
30. Pałubska, S.; Adamiak-Godlewska, A.; Winkler, I.; Romanek-Piva, K.; Rechberger, T.; Gogacz, M., Hyperprolactinaemia–a problem in patients from the reproductive period to the menopause. Przeglad menopauzalny= Menopause review 2017, 16 (1), 1.
31. Sep, S.; Rijvers, C.; Smits, L.; van Bilsen, M.; Bekers, O.; Peeters, L., Early-pregnancy changes in maternal lipid profile in women with recurrent preeclampsia and previously preeclamptic women with normal next pregnancy. Reproductive Sci*ences*

2011, 18 (10), 998-1004.

32. Tripathy, C.; Malik, S.; Shah, P.; Lakshmy, R.; Tripathy, D., Serum Insulin and Lipid Profile in Normal Pregnant and Pregnancy-Induced Hypertensive Women from North India. *Australian and New Zealand journal of obstetrics and gynaecology* 1999**,** *39* (3), 321-323.
33. Saihood, S. T., Early-Pregnancy Changes in Maternal Lipid Profile in Women with Recurrent Preeclampsia and Women with Normal Pregnancy. *Journal of the Faculty of Medicine Bag*hdad 2017, 59 (2), 128-131.