Abstract

Background

Pregnancy is associated with normal physiological changes that assist fetal survival as well as preparation for labour. It is typically broken into three trimesters, each of about three months. These distinctions are useful in describing the changes that take place over time.

Biochemical parameters reflect these adaptive changes are clearly distinct from the non-pregnant state. The mother and the placenta produces many hormones, maternal thyroid function during pregnancy is important for both the mother and the developing fetus. Especially during the first trimester, when the fetus is completely dependent on the mother for thyroid hormone. It also decreases maternal tissue sensitivity to insulin, resulting in gestational diabetes. Dilution anaemia is caused by the rise in plasma volume, Blood volume increases by 40% in the first two trimesters this causes low level of albumin . This is just to an increase in plasma volume through increased aldosterone. Increase liver metabolism is also seen, with increased gluconeogenesis to increase maternal glucose levels .Renal plasma flow increases during pregnancy.

Objectives

1-This Study includes measurement of the thyroid hormones T3,T4,TSH and biochemical parameters(glucose, uric acid, total protein ,lipid profile , and hemoglobin) in serum of pregnant women during the three trimesters and serum of non-pregnant women.

2- Investigation the relationship between thyroid hormones level and bio-chemical tests profile in serum of the pregnants during three trimesters and also non-pregnant women.

Subject and Methods

In the present study the individual sera of hormones (triiodothyronin (T3) , thyroxin (T4) and thyroid –stimulating hormone (TSH) were assayed in 70 healthy women . Serum collected from fifty pregnant women at three trimesters and twenty non-pregnant women as a control group. The measurements occurs in both Al- ELWIA Hospital and Central Public Health Laboratories. The T3,T4 and TSH was measured by using Immunophlorences method by miniVidas. This measurement is recruited from 1/7/2007 to 8/4/2008.

This study also designed to investigate changes in the levels of biochemical parameters (Glucose, Uric acid ,Total protein, Lipid profile , and Hemoglobin)in sera of pregnant women at three trimesters and on sera of non-pregnant women. The glucose, uric acid, lipid profile were measured by spectrophotometer instrument .The Total protein was measured by using Clinical refractometers instrumental (ATAGO) and hemoglobin was measured by Micro-Haematocrit Centrifuge instrument ..

Results

Results of pregnant women at first trimester showed that there was a significant difference ($P<0.001$) in hormones T4 compared with control group . Results in the second trimester it was found that a highly significant difference ($P<0.001$)in hormones T3,T4 and TSH ($P<0.05$) compared with the control group .In the third trimester there was a significant differennce ($P<0.001$) in hormones T4 and T3 ($P<0.05$) compared with the control group.

The results of Biochemical parameters showed that at the first trimester there was a positive significant difference ($P<0.001$) in glucose, hemoglobin compared with control group ,In the second trimester there was a positive significant difference ($P<0.05$) in cholesterol , LDL and

hemoglobin , and in the third trimester it was found apositive significant differences (P<0.001) in Total protein, cholesterol , triglyceride , LDL and VLDL compared with the control group.

A strong positive correlation coefficient was found between T3 at the firsttrimister with a weak of gestation and HDL, and a negative correlation coefficient between T3 with glucose and uric acid . And a significant positive correlation coefficient was found at the second trimester between T3 with triglyceride, VLDL and CHO/HDL, while a negative correlation with uric acid ,total protein and hemoglobin . A positive correlation was found at third trimester between T3 with glucose and uric acid.

Results of non-pregnant group were found with a negative correlation coefficient between T3 with triglyceride, VLDL, LDL ,and LDL/HDL.

There was a negative correlation coefficient between T4 at the firsttrimister with glucose . Results At the second trimester there was a positive correlation coefficient between T4 with weak of gestation and T3,there was a negative correlation coefficient between T4 with total protein .Results At third trimester there was a negative correlation coefficient between T4 with triglyceride and VLDL.

Results in non-pregnant state there was a negative correlation coefficient between T4 with hemoglubin.

There was a positive correlation coefficient between TSH at the firsttrimister with glucose, uric acid, cholesterol, VLDL, triglyceride, LDL, CHO/HDL and LDL/HDL .Results At the second trimester there was a positive correlation coefficient between TSH with glucose, and uric acid ,there was a negative correlation coefficient between TSH with triglyceride, VLDL and T3 . Results At third trimester there was a positive correlation coefficient between TSH with glucose.

Results in non-pregnant state there was a positive correlation coefficient between TSH with HDL, and T3 .

Conclusions:

1-In this study it was an increasing in higher T3 and T4 levels and decreasing in the TSH levels in serum of pregnant at the three trimesters which will produced hyperthyroidism if not controlled symptoms.

2- The study proves that changes in biochemical parameters in serum pregnant during three trimester , affects on the mother and fetal.

3- There were a relationships between thyroid hormone and biochemical parameters for pregnant women and non-pregnant women.

Republic Of Iraq
The Ministry Of Higher Education
And Scientific Research
Al-Nahrain University
College Of Science
Department Of Chemistry

Biochemical profile associated with thyroid hormones variation in pregnancy

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of Al-Nahrain University
In Partial Fulfillment of the Requirements
For the degree of Master of Science
In
Clinical Biochemistry

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SUHAD ABDUL AZIZ IBRAHEM

B.Sc. in Chemistry

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بسم الله الرحمن الرحيم

(وَإِذَا سَأَلَكَ عِبَادِي عَنِّي فَإِنِّي قَرِيبٌ أُجِيبُ دَعْوَةَ الدَّاعِ إِذَا دَعَانِ فَلْيَسْتَجِيبُواْ لِي وَلْيُؤْمِنُواْ بِي لَعَلَّهُمْ يَرْشُدُونَ)

صدق الله العظيم

(سوره البقره الايه ١٨٥)

Supervision Certificate

I certify that this thesis was prepare under my supervision at the College of Science in a partial fulfillment of the requirements for the degree of Master of Science in Clinical Biochemistry.

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Name: Dr .Salman A. Ahmed
Title: Assistant Professer
Date: / / 2008

In view of the available recommendation , it forwards this thesis for debate by examining committee.

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Committee certification

We, the examining committee, certify that we have read this thesis and have examined the student Suhad Abdul Azeez Ibrahim in its content, and that in our opinion it is adequate with ( ) standing as a thesis for the degree of Master of Science in Clinical Biochemistry.

Chairman
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Name:
Date: / /2008

Member
Signature:
Name:
Date: / /2008

Member
Signature:
Name:
Date: / /2008

Member (supervisor)

Signature:

Name: Assist. Prof. Dr. Salman A. Ahmed

Date: / /2008

Approved for the council of the College of Science.

Signature:
Name :Assist .Prof .Dr .LAITH ABDUL AZIZ AL-ANI
Dean of the College of Science
Date: / /2008

الإهداء

إلى من حملتني وهن على وهن.....

وسهرت الليالي على راحتي.....

إلى سيدة العاطفة ومنبع الحنان.....أمي الغالية

إلى القلب الطيب .....

إلى معلمي الأول وقدوتي في الحياة .....

إلى الذي وهب لي عمره وعطفه وكان لي أبا وصديقا.....أبي رحمه الله واسكنه أوسع جناته

إلى روافد الوفاء.....

إلى من اشد أزري في الحياة....

إلى من طال بهم شوط الانتظار.....إخوتي وأخواتي الأعزاء

إلى رفاق الدرب الطويل .....الأصدقاء الأوفياء

إليكم جميعا اهدي هذا الجهد المتواضع

سهاد

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SUHAD

Aim of the study:

1-To study the thyroid hormones (T3,T4,TSH) level in pregnant women

2-To study the biochemical profile(glucose, uric acid, total protein,

lipid profile and Hemoglobin)level in pregnant women

3-To study the relationship between the thyroid hormones and biochemical parameters (glucose ,uric acid ,total protein ,lipid profile and hemoglobin) in pregnant in three trimester : (first trimester, second trimester and third trimester)

4- To study the same relationship also in non- pregnant women group (control).

Appendix

The name of the patient: *age:* *Weight:* *Length:*
A week of pregnancy:
Diseases associated with pregnancy: pressure, Sugar ,other diseases
The type of drugs used:
The type of contraception: one or twin
The type of inflation gland: two 1-one 2-a multi-year, or
The nature of food: May contain iodine
What Repasts covered container on iodine
The symptoms of thyroid disease
1-temperature object i
2-temperature poetry ii
3-psychological condition iii
4-Increased sweat iiii
5-suffering lack sleep
6-case monthly session
Measuring the value of uric acid
S.tatal proten
Lipid profile
Glucose
Hemoglubin
Measuring T3,
Measuring T4
Measuring TSH

APPENDIX

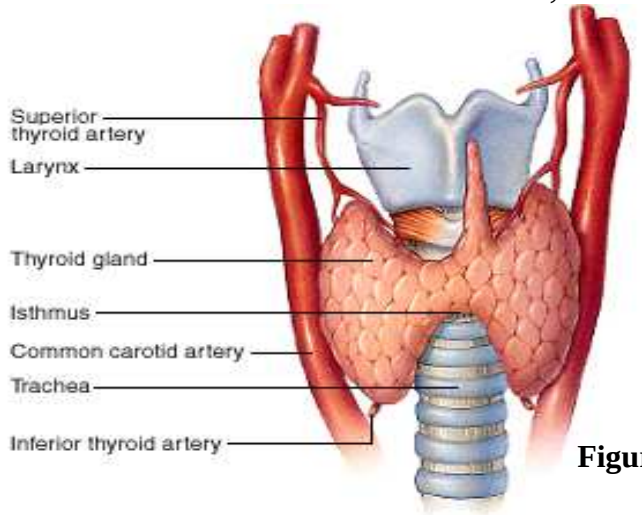
Chapter One Introduction

1.1- HORMONES

1.1.1 - Thyroid Hormones:

The thyroid gland is a small gland found in the neck below the larynx(Adams apple)(fig1.1). It makes and releases thyroid hormones to help regulate body growth and metabolism. Thyroxine (T4) and tri-iodothyronine(T3) are together known as the "thyroid hormones", they are synthesized in the thyroid gland by iodination and coupling of two tyrosine molecules while attached to a complex protein called thyroglobulin. T4 has four iodine atoms while T3 has three(fig1.2).(1)

The thyroid gland secretes mostly T4 whose concentration in plasma is around 100 nmol/l. The peripheral tissues, especially the liver and kidney,deiodinate T4 to produce approximately two-thirds of the circulating T3 which present at a lower concentration of around 2nmol/l. Most cells are capable of taking up T4 and deiodinating it to the more biologically active T3. It is T3 which binds to receptors and triggers the end-organ effects of the thyroid hormones. Alternatively, T4 can be metabolized to reverse T3, which is biologically inactive [2]



Figure(1-1) thyroid gland [1]

As shown in the following diagram, the thyroid hormones are basically two tyrosines linked together with the critical addition of iodine at three or four positions on the aromatic rings. The number and position of the iodines is important. Several other iodinated molecules are generated that have little or no biological activity; so called "reverse T3" (3,3',5'-T3) is such an example.

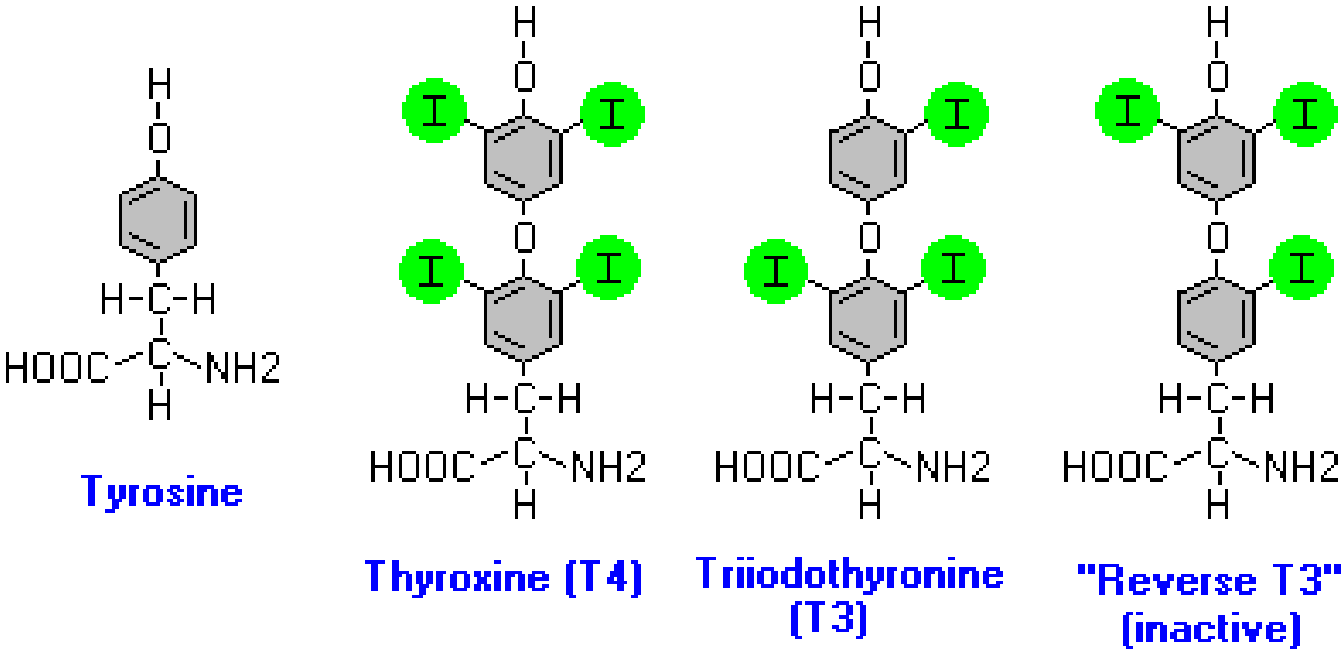


Figure (1-2) The chemical structure of the thyroid hormones,T4andT3,and the inactive metabolite of T4,rT3 [1]

1.1.2- Thyroid hormones Synthesis :

Thyroid hormone synthesis includes the following steps: (1) iodide (I^-) trapping by the thyroid follicular cells; (2) diffusion of iodide to the apex of the cells; (3) transport of iodide into the colloid; (4) oxidation of inorganic iodide to iodine and incorporation of iodine into tyrosine residues within thyroglobulin molecules in the colloid; (5) combination of two diiodotyrosine (DIT) molecules to form tetraiodothyronine (thyroxine, T4) or of monoiodotyrosine (MIT) with DIT to form triiodothyronine (T3); (6) uptake of thyroglobulin from the colloid into the follicular cell by endocytosis, fusion of the thymoglobulin with a lysosome, and proteolysis and release of T4, T3, DIT, and MIT; (7) release of T4 and T3 into the circulation; and (8) deiodination of DIT and MIT to yield tyrosine. T3 is also formed from monodeiodination of T4 in the thyroid and in peripheral tissues.(figure 1.3),(fig1.4) [3]

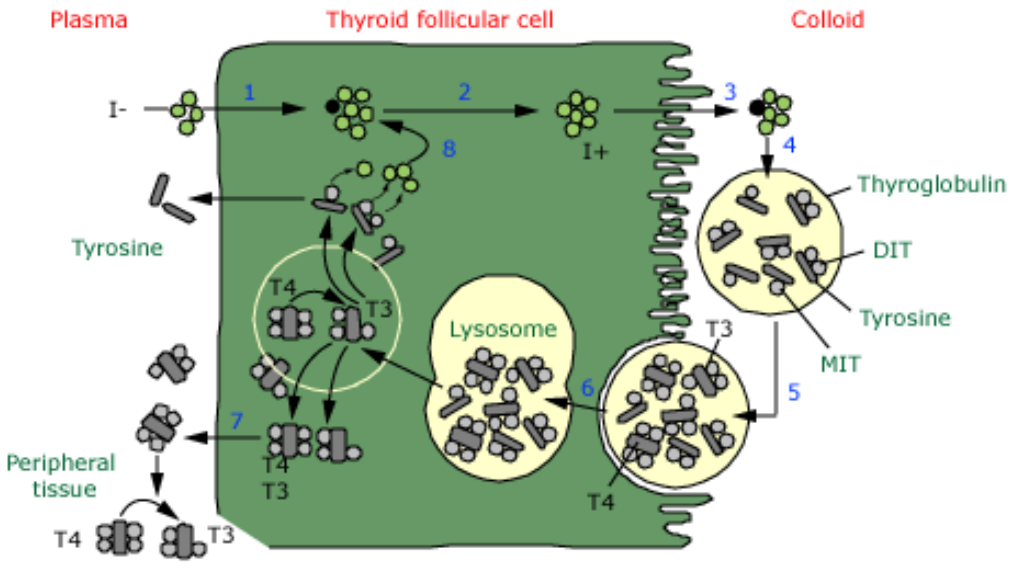
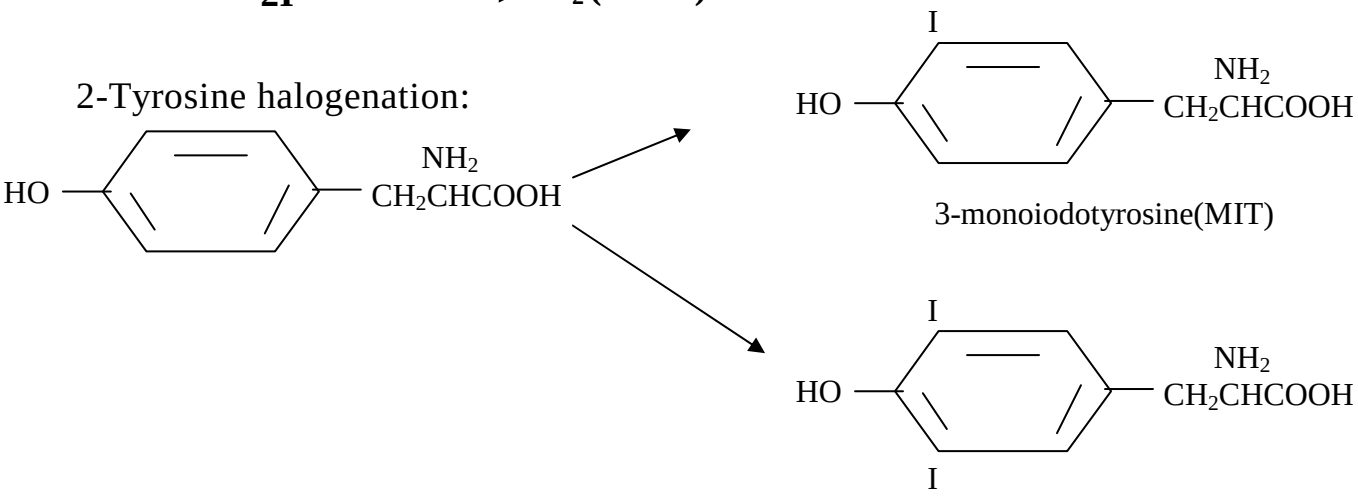


Figure (1-3) Thyroid Hormone Synthesis [3]

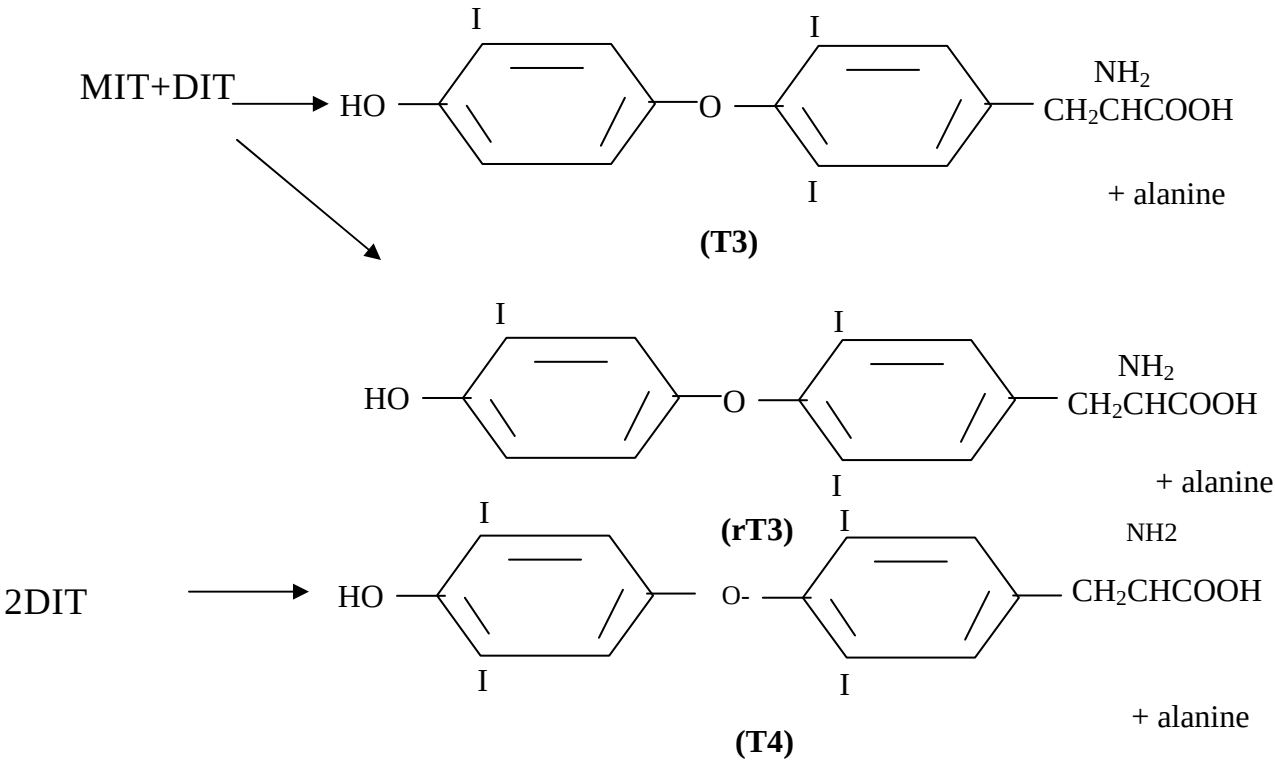
1- oxidation of I^{-1} to active form:

$$2I^{-1} \xrightarrow{\text{peroxedase}} I_2 \text{ (active)}$$

2-Tyrosine halogenation:



3-copling of MIT and DIT:



Figure(1-4) biosynthesis of thyroid hormones(T3),(T4) [3]

1.1.3- Function of Thyroid Hormone:

Thyroid hormones are essential for normal growth , development and have many effects on metabolic processes . They act by entering cells and binding to specific receptors in the nuclei, where they stimulate the synthesis of a variety of species of mRNA, thus stimulating the synthesis of polypeptides, including hormones and enzymes. Their most obvious overall effect on metabolism is to stimulate the basal metabolic rate. [4]

1.1.4- Effects of thyroid hormones :

1- Fetal brain development and skeletal maturation are dependent of fetal thyroid hormone production. In the absence of fetal thyroid hormone secretion ,cretinism results . [5]

2- T3 increases oxygen consumption and heat production which contributes to increased basal metabolic rate and the increased sensitivity to heat in hyperthyroidism and increased sensitivity to cold in hypothyroidism . [6]

3. Thyroid hormone stimulates hepatic gluconeogenesis and glycogenolysis as well as intestinal absorption of glucose .This results in increased serum glucose. Thyroid hormone also causes increased cholesterol synthesis and degradation as well as increased lipolysis. This results in a lowering of serum cholesterol [7,8]

1.1.5- Control of Thyroid Hormones Synthesis and Secretion:

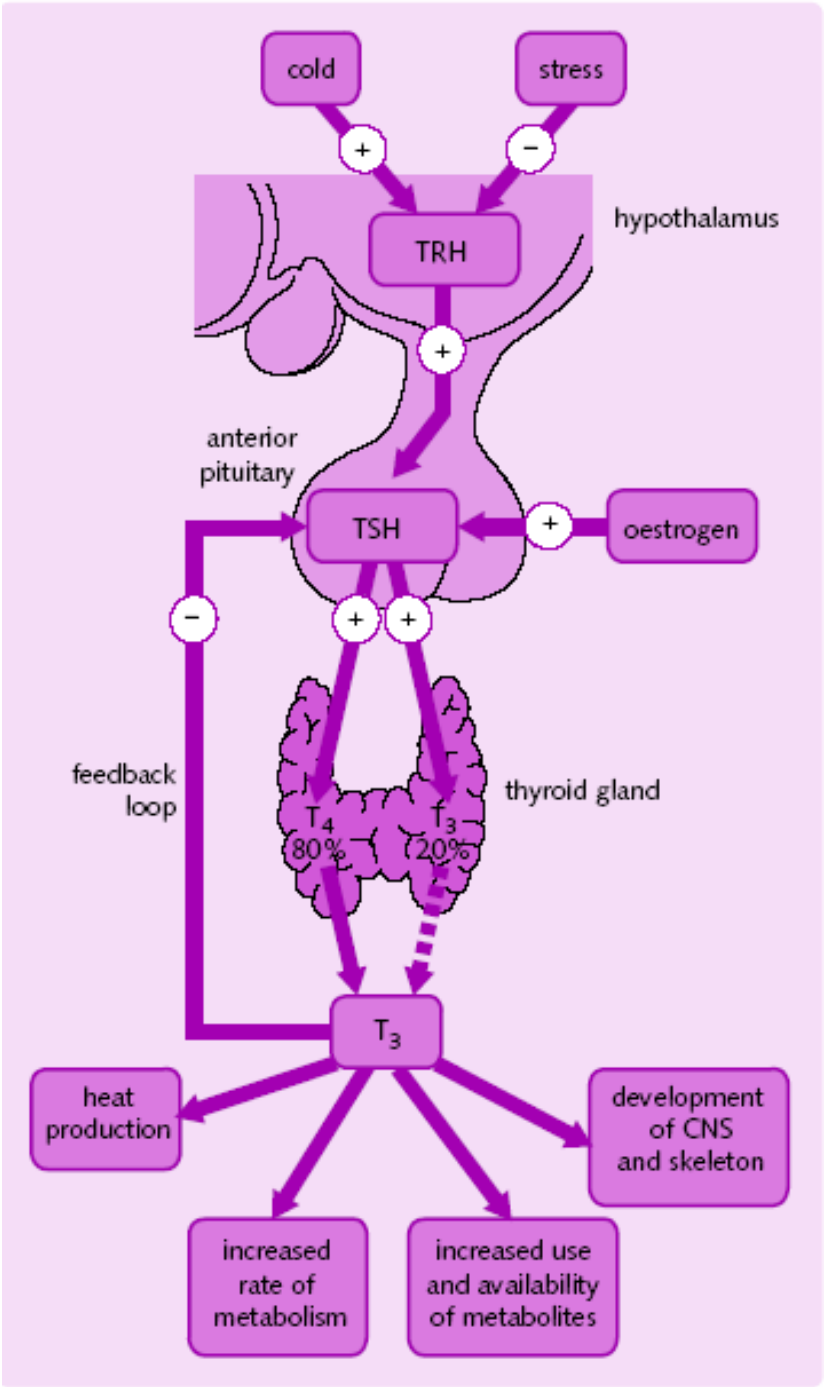
The thyroid hormones are synthesized and stored in the follicles and are dependent on an adequate iodine supply. The thyroid gland acts as a store of iodine .Thyroid hormone levels are regulated by a multiplex negative feedback loop with control from the hypothalamic-pituitary axis and autoregulation within the thyroid itself. The end product of this process is the production of the two thyroid hormones(figure 1.5) [9]

1-T4---a prohormone that acts as a plasma reservoir.

2-T3---the active hormone [10]

1.1.6- Feed back inhibition :

T3 receptors are found in the pituitary gland and the hypothalamus, where they inhibit transcription of the gene for TRH prohormone and the release of TSH, respectively. Excess T3 inhibits TSH release while a deficiency of T3 stimulates TSH release. This feed –back mechanism helps to maintain T3 levels, and therefore stabilizes metabolic rate(figure 1.5) [11]



Figure(1-5) Hormonal regulation of the thyroid hormones.[(T3) triiodothyronine;(T4) thyroxine; (TRH) thyrotrophin-releasing hormone;(TSH) thyroid-stimulating hormone] [12]

+ stimulation
_ inhibition

1.1.7-Thyroid hormones during pregnancy:

In the first trimester (3 months) of pregnancy, the fetus is completely dependent upon the mother for thyroid hormone. During the second and final trimesters of pregnancy, most fetuses are able to provide some thyroid hormone but also continue to rely upon their mothers for some hormone. Problems of brain development occur when either the mother is unable to provide for the fetus' needs in the first trimester, or neither the mother nor the fetus can provide for fetal needs during the remainder of pregnancy. Other things is the availability of iodine to the mother since iodine is an important requirement to provide for proper thyroid production by both the mother and the developing fetus [13]

1.1.8 -MATERNAL THYROID PHYSIOLOGY:

During pregnancy, maternal thyroid function is modulated by three independent but interrelated factors : (1) an increase in hCG concentrations that stimulate the thyroid gland, (2) significant increases in urinary iodide excretion, resulting in a fall in plasma iodine concentrations, and (3) an increase in thyroxine-binding globulin (TBG) during the first trimester, resulting in increased binding of thyroxine. In the aggregate, these factors may be responsible for the increased thyroid demand, or thyroid "strain" observed during pregnancy. [14]

1.1.9-Thyroid Disease in Mother During Pregnancy:

Hypothyroidism. can result from a thyroid gland defect or secondarily from insufficient thyrotropin-releasing hormone(TRH) secretion from the hypothalamus, or insufficient TSH secretion from the anterior pituitary, or insufficient iodine in the diet. in the latter case, excessive TSH secretion stimulates abnormal thyroid growth and the development of an endemic goiter . The hypothyroidism and goiter caused by iodine deficiency can be reversed by iodine supplements [15]

Hyperthyroidism occurs when the thyroid gland becomes overactive and produced excess thyroid hormones. It is a condition called Graves disease . In this disease ,the body's immune system begins to attack the very organs and tissues that its supposed to protect. This leads to excessive production of thyroid hormone by the thyroid gland. Additional symptoms include swelling of the neck due to enlargement of the gland ,and protrusion of the eyes. Hyperthyroidism can also be caused by hormone producing tumors in the pituitary gland .This cause is far less frequent. There is also a form of hyperthyroidism that develops soon after birth [16]

The most common symptoms include:

1- Effect on circulation :- high thyroid hormone level usually lead to a high heart rate.

2- Growth, weight and a ppetite :- Children who develop hyperthyroidism often start growing at a much faster rate than is normal for their age.

Increased a ppetite may also be present , although there is often weight loss that may be extreme.

3- Behavior problems :- a child may be restless and have poor concentration .They are often moody and may be unable to sleep well.

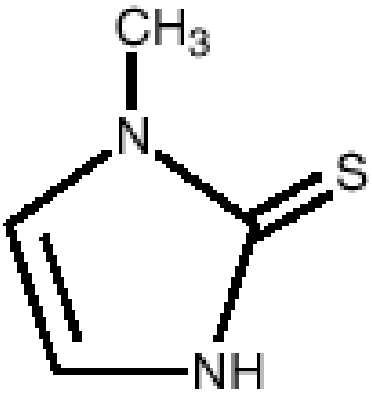
The amount of radiation may approach levels which can be harmful Later in pregnancy radioactive iodine can destroy the fetal thyroid [17]

1.1.10- **Treatment:**

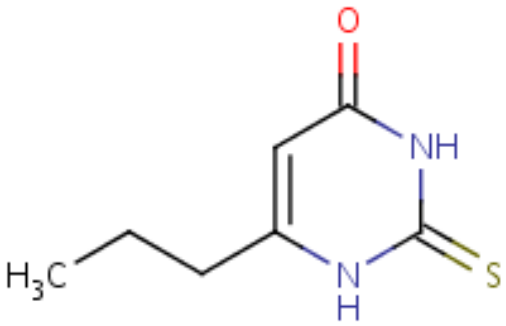
Choice for thyrotoxicosis during pregnancy is antithyroid medication, either propylthiouracil or methimazole (figure1.6), since radioactive iodine cannot be used. Propylthiouracil (PTU) remains the drug of choice, since it does not cress the placenta as well as methimazole

(1-methylimidazole -2 –thiol) differs chemically from the drugs of the thiouracil series primarily because it has a 5- instead of a 6-membered ring).The initial goal is to control the hyperthyroidism and then use the lowest medication dose possible to maintain the serum thyroid hormone levels in the high normal range. In this way the smaller doses of medications are used, and there seems to be little risk to the baby. If a mild allergy to one of these medications develops, the other medication may be substituted. If there is a problem with taking pills or more severe drug allergy, then an operation may be performed to remove most of the thyroid gland. This is usually done in the middle part (second trimester)of the pregnancy. Fortunately, it is rarely necessary [18]

The natural course of hyperthyroidism in pregnancy is for the disease to become milder or remit totally near term. In many patients antithyroid medications can be tapered to low levels or even discontinued. For those patients who are not so fortunate, it is important to maintain control of the hyperthyroidism throughout pregnancy to avoid severe thyrotoxicosis (thyroid storm) developing during labour and delivery. If this does develop, additional acute treatment with beta-adrenergic blocking drugs such as propranolol (Inderal) and high doses of no radioactive iodine are used . Long-term treatment with these agents is not advised in pregnancy [19]



Methimazole (Tapazole)



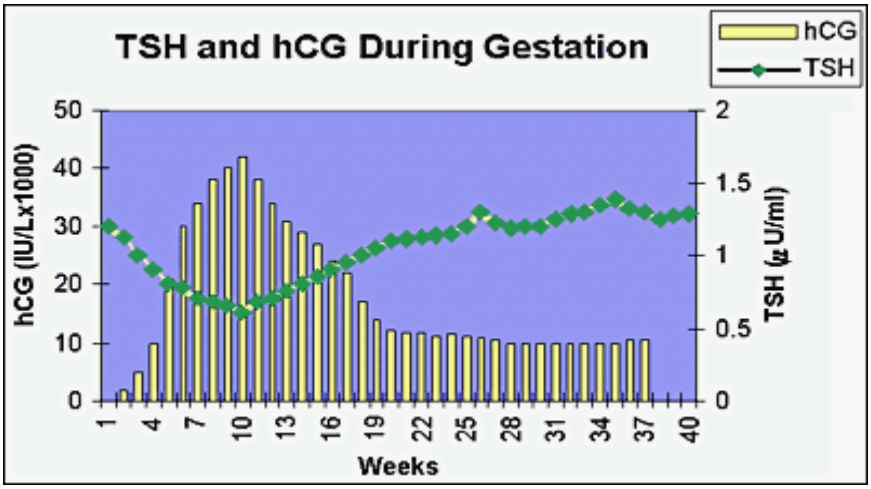
Propylthiouracil (PTU)

Figure (1-6): Show the structure of propylthiouracil and methimazole. [20]

1.2 - Other Hormones During Pregnancy:

1.2.1 Human chorionic gonadotropin

Human chorionic gonadotropin (hCG) is a peptide hormone responsible for the production of adequate concentrations of progesterone early in gestation. hCG concentrations increase dramatically during the first trimester of pregnancy [21] Structurally, the hCG peptide is composed of two chains, an alpha chain and a beta chain, the same structural composition of TSH ,Significantly, the alpha chain is identical to that of TSH, while the beta chains differ between the two molecules [22] This partial structural homology anticipates at least a partial overlap in function. The overall effect of hCG on the degree of thyroid stimulation and TSH suppression reflects an integration of both the amplitude and the duration of the hCG peak. Despite hCG-mediated stimulation of the thyroid gland, free (unbound) serum hormone concentrations generally remain within, or slightly above, the normal range during the first trimester. (Fig1.7) [23]



Figure(1-7). Thyroid-stimulating hormone (TSH) and human chorionic gonadotropin (hCG) during gestation. Note the reciprocal relationship between TSH and hCG [24]

1.2.2-Prolactin:

During pregnancy, prolactin secretion in normal women markedly increases from the beginning of pregnancy until delivery. The marked rise in serum prolactin concentrations, often into a range of 100 to 200 ng/ml or higher is the result of an actual increase in lactotroph number and capacity because of stimulation by estradiol. Estrogen both stimulates lactotroph number and prolactin secretion by a direct effect on prolactin transcription and also cell proliferation. [25] Several substances released from the hypothalamus to regulate prolactien secretion .There are two main releasing hormones: one increases prolactin secretion and one decreases prolactin. Serotonin and thyroid hormone help to increase prolactin release, whereas dopamine works to block prolactin release. [26].

1.2.2.1- Structure:

Prolactin is a single-chain polypeptide of 199 amino acids with a molecular weight of about 24,000 Daltons. Its structure is similar to that of growth hormone and placental lactogen. The molecule is folded due to the activity of three disulfide bonds. Significant heterogeneity of the molecule has been described, thus bioassays and immunoassays can give different results due to differing glycosylation, phosphorylation, sulfation, as well as degradation. The non-glycosylated form of prolactin is the dominant form of prolactin that is secreted by the pituitary gland [27]

Little prolactin is apparently the result of removal of some amino acids, while **big prolactin** can be the produced from the interaction of several prolactin molecules [28]

1.2.2.2- Effects of prolactin

Prolactin has many effects:

1- To stimulate the mammary glands to produce milk (lactation), the high levels of progesterone during pregnancy act directly on the breasts to stop ejection of milk. It is only when the levels of this hormone fall after childbirth that milk ejection is possible. [29]

2- To contribute to surfactant synthesis of the fetal lungs at the end of the pregnancy and immune tolerance of the fetus by the maternal organism during pregnancy [30]

3- To decrease normal levels of sex hormones — estrogen in women and testosterone in men [31]

1.2.2.3- Conditional affecting on Prolactin secretion :

Conditions elevated prolactin secretion resulting in Hyperprolactinaemia which is the presence of abnormally - high levels of prolactin in the blood. Normal levels are less than 580 mIU/L for women. The hormone prolactin is down regulated by dopamine and is unregulated by estrogen. [32] These condition are:

a- Prolactinoma

b- Excess thyrotropin-releasing hormone (TRH), usually in primary hypothyroidism

c- Many anti-psychotic medications

Conditions causing decreased prolactin level in the blood are:-

a- Bulimia

b- Excess dopamine

1.3- Biochemical Tests:

1.3.1- Blood Glucose:

Measures blood sugar-elevated levels associated with diabetes. Glucose is a six-carbon sugar that is the main source of energy for all of the cells in the body. The rate at which it is metabolized is controlled by insulin, which is secreted by the pancreas. Elevated fasting levels of glucose (>109 mg/dl) may be an early sign of diabetes or could indicate other problems such as hyperthyroidism. Low levels (hypoglycemia) could indicate too much insulin in the blood. Glucose transported via the bloodstream, it is tightly regulated in the human body. Normally, the blood glucose level is maintained between about 70 to 100 mg/dl. Glucose levels rise after meals and are usually lowest in the morning, before the first meal of the day. Failure to maintain blood glucose in the normal range leads to conditions of persistently high (hyperglycemia) or low (hypoglycemia) blood sugar. Diabetes mellitus, characterized by persistent hyperglycemia of several causes, is the most prominent disease related to failure of blood sugar regulation. [33] , blood glucose above 180 mg/dL increases the risk of miscarriage.

In pregnancy at the third trimester and near delivery the increase level of glucose in blood can cause the infant to be larger than normal and be at higher risk for complications after delivery. [34] Very high blood glucose levels over 250 mg/dL late in pregnancy increases the risk of stillbirth. These complications occur less frequently when blood glucose levels are carefully controlled. [35] Glucose in the mother's blood crosses the placenta to help provide energy for the fetus; thus, maternal hyperglycemia (high blood glucose levels) leads to fetal hyperglycemia as

well. In response to high glucose levels, the fetus produces large amounts of insulin, which lead to problems such as excessive fetal growth and low blood glucose levels after birth. [36]

1.3.1.2 **Gestational Diabetes**:

This type is discovered during pregnancy and usually disappears when the pregnancy is over. Women who have had gestational diabetes have a grater risk of developing Type 2 diabetes later in their lives.(37)

1.3.1.3-Treatment for gestational diabetes:

Treatment for gestational diabetes focuses on keeping blood glucose levels in the normal range. Treatment may include:

1- special diet
2- exercise
3- daily blood glucose monitoring
4- insulin injections (rarely) (38)

1.3.2 Uric acid:

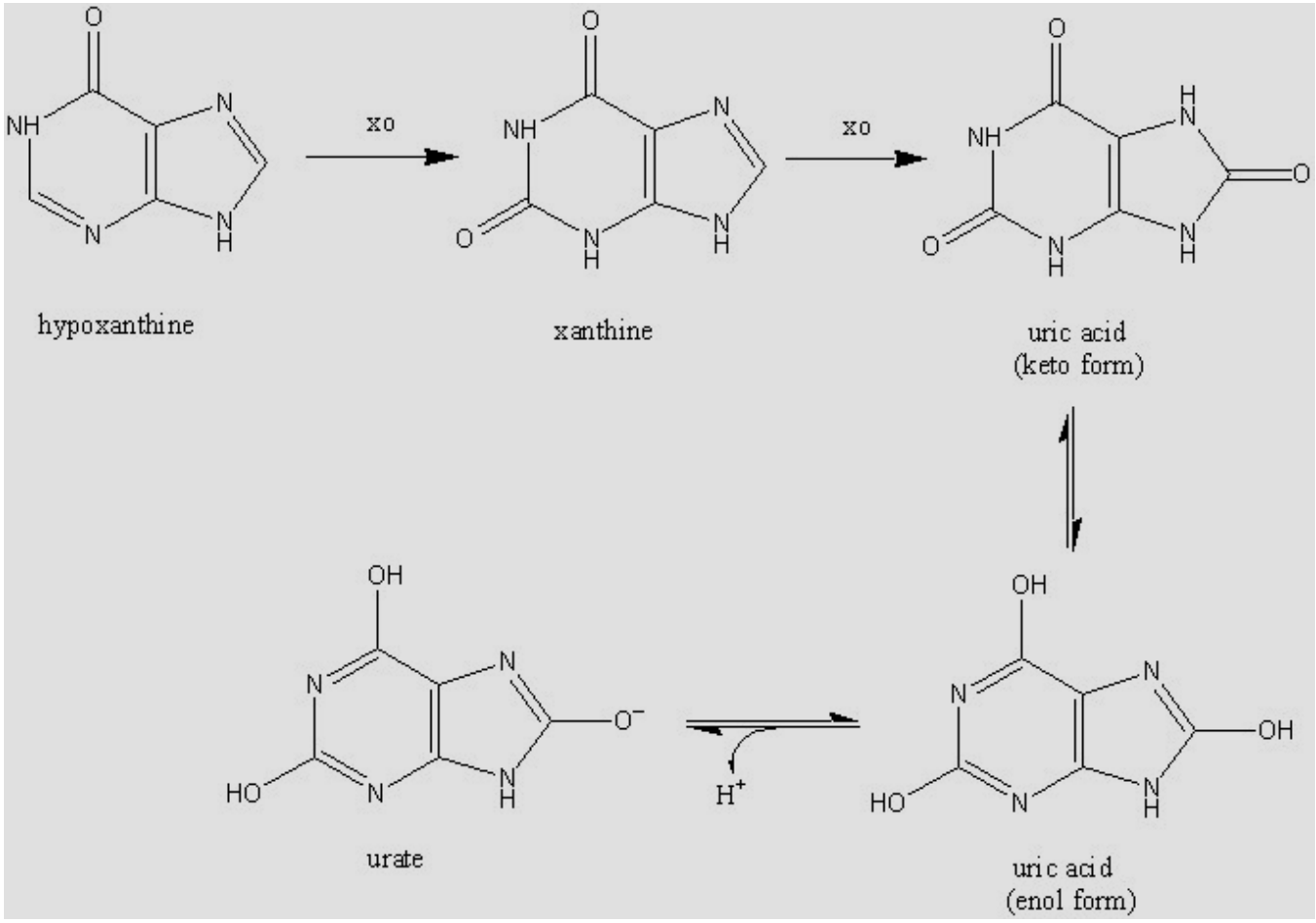
1.3.2.1 Metabolic processes

Uric acid (or urate) is an organic compound of carbon, nitrogen, oxygen and hydrogen with the formula $C_5H_4N_4O_3$.(39)

Xanthine oxidase oxidizes the oxypurines such as xanthine and hypoxanthine to uric acid. In humans and higher primates, uric acid is the final oxidation product of purine catabolism. In most other mammals, the enzyme uricase further oxidizes uric acid to allantoin. (40)fig(1.8).

Excess serum accumulation of uric acid can lead to a type of arthritis known as gout. Elevated (serum uric acid) level (hyperuricemia) can result from high intake of purine-rich foods, high fructose intake (regardless of fructose's low Glycemic Index (GI) value) and/or impaired excretion by the kidneys.(41) Saturation levels of uric acid in blood may result in one form of kidney stones when the urate crystallizes in the kidney. These uric acid stones are radiolucent and so do not appear on an abdominal x-ray. Their presence must be diagnosed by ultrasound for this reason. Some patients with gout eventually get uric kidney stones. (42)

During the second half of pregnancy is to be based on increases in serum uric acid concentrations then such increases will have to be carefully interpreted against the background of rising concentrations which occur as part of the physiological response to normal pregnancy (43)



Figure(1-8) **The formation of uric acid in the body** (44)

1.3.3- Total protein:

Protein in the plasma is made up of albumin and globulin. The globulin in turn is made up of α1, α2, β, and γ globulins.

These fractions can be quantitated using protein electrophoresis , But the total protein test is a faster and cheaper test that estimates the total of all fractions together. (45) The reference range for total protein is 60-85gm/L.Concentrations below the reference range usually reflect low albumin concentration, for instance in liver disease or infection. Concentrations above the reference range are found in paraproteinaemia,Hodgkin's lymphoma or leukemia Thyroid hormones inhibit TSH secretion at least in part by process that is inhibited by protein synthesis inhibitors. (46)

During pregnancy ,protein levels remained unchanged except for a small increase in protein antigen at 28-32 weeks gestation . Serum albumin concentration falls gradually from early pregnancy and this is related to ECF(Extra cellular Fluid) expansion. The concentrations of many other proteins increase, particularly placental proteins such as alkaline phosphatase of placental origin, transport proteins such as transferring ,hormone-binding glycoproteins such as thyroxin-binding globulin, and fibrinogen. (46)

1.3.4 Lipid profile:

A lipid profile is a measurement of various lipids that are found in the blood. This kind of blood test is often used to assess risk of heart disease. There are two common concerns people have about lipids in their diet: one is their high caloric value, which may lead to undesired weight gain. The other is their association with high total cholesterol levels, which are a risk factor for cardiovascular disease. Limiting the intake of fat and oil in the diet, especially saturated fats, may help keep cholesterol levels low and thus lower ones risk of heart disease. (48)

1.3.4.1 Cholesterol:

The name originates from the Greek chole- (bile) and stereos (solid), and the chemical suffix -ol for an alcohol, as researchers first identified cholesterol in solid form in gallstones by François Poulletier de la Salle in 1769. However, it is only in 1815 that chemist Eugène Chevreul named the compound "cholesterine".(49)

Most cholesterol in the body is synthesized by the body and some has dietary origin. Cholesterol is more abundant in tissues which either synthesize more or have more abundant densely-packed membranes, like, the liver, spinal cord and brain. cholesterol plays a central role in many biochemical processes, but is best known for the association of cardiovascular disease with various lipoprotein cholesterol transport patterns and high levels of cholesterol in the blood. ".(50)

cholesterol and other fats are insoluble in blood, they have to be transported in the circulatory system within lipoproteins, There is a large range of lipoproteins within blood, generally called, from larger to smaller size: very low density lipoprotein (VLDL), low density lipoprotein (LDL) and high density lipoprotein (HDL). (51)

During pregnancy cholesterol level is generally on a rise during pregnancy.(52)

1.3.4.1.1 Function

Cholesterol is required to build and maintain membranes; function of cholesterol , it regulates membrane fluidity over a wide range of temperatures. (53) The hydroxyl group on cholesterol interacts with the phosphate head of the membrane, while the bulky steroid and the hydrocarbon chain is embedded in the membrane. Some research indicates that cholesterol may act as an antioxidant. Cholesterol also aids in the manufacture of bile (which is helps digest fats), and is also important for the metabolism of fat soluble vitamins, including vitamins A, D, E and K. It is the major precursor for the synthesis of vitamin D and of the various steroid hormones. (54)

1.3.4.2 Triglyceride (TG):

Triglyceride plays an important role in metabolism as energy sources and transporters of dietary fat. They contain more than twice as much energy (9 kcal/gm) as carbohydrates and proteins. In the intestine, triglycerides are split into glycerol and fatty acids (this process is called lipolysis).(55)

Calories ingested in a meal and not used immediately by tissues are converted to triglycerides and transported to fat cells to be stored. Hormones regulate the release of triglycerides from fat tissue so they meet the body's needs for energy between meals. During pregnancy as expected, significant increases in total triglycerides were observed with advancing gestation. (56)

1.3.4.3 High density lipoprotein(HDL-cholesterol) :

High-density lipoproteins (HDL) form a class of lipoproteins, that carry cholesterol from the body's tissues to the liver. About thirty percent of blood cholesterol is carried by HDL. (57)

Some experts believe HDL remove excess cholesterol from plaques and thus slows their growth. HDL-cholesterol is known as "good" cholesterol because a high HDL level seems to protect against heart attack. Serum HDL-cholesterol levels did not change significantly during pregnancy .(58)

1.3.4.4 Low density lipoprotein(LDL-cholesterol) :

Low-density lipoprotein as the major cholesterol carrier in the blood. (59) If too much LDL-cholesterol circulates in the blood ,it can slowly build up in the walls of the arteries feeding the heart and brain. With other substances it can form plaque , a thick, hard deposit that can clog those arteries. This condition is known as atherosclerosis . (60)

Generally ,LDL transports cholesterol and triglycerides from the liver and small intestine to cells and tissues which are taking up cholesterol and triglycerides .Increasing level of LDL-cholesterol gradually as pregnancy proceeded, reached maximum values in the third trimester.(61)

1.3.4.5 Very low density lipoprotein (VLDL-cholesterol) :

Very-low-density lipoprotein (VLDL)cholesterol is one of the three major types of blood cholesterol combined with protein .The other two are high-density lipoprotein (HDL) cholesterol and low-density lipoprotein (LDL) cholesterol. Each type contains a specific combination of cholesterol, protein and triglyceride, a blood fat. VLDL cholesterol contains the highest amount of triglyceride. (62)

Like LDL cholesterol ,VLDL cholesterol is considered "bad" cholesterol because elevated levels are associated with an increased risk of coronary artery disease. There is no simple, direct way to measure VLDL cholesterol. So it is usually calculated as a percentage of triglyceride levels. (63)

1.4- Hemoglobin

Hemoglobin is the protein in red blood cells (RBCs) that gives the blood its red color. It binds to oxygen in the lungs, transports it throughout the body, and releases it to the cells and tissues. (64)

During pregnancy, a woman's hemoglobin must transport enough oxygen to meet both her and her baby's needs. Many pregnant women will experience some degree of anemia. (65)

Anemia can be caused by decreased RBC production, increased RBC destruction, or by increased RBC (blood) loss. Anemia in a pregnant mother can cause a fetus to receive too little oxygen to support normal development.The first baseline concentrations will be compared to later values to look for changes that could indicate increasing anemia.(66)

Often a hemoglobin will be run as part of a Complete Blood Count (CBC). The CBC also measures the actual number of RBCs, the number and type of white blood cells (WBCs), the number of platelets (cell fragments involved in blood clotting), and the hematocrit (the amount of solids versus liquids in the blood). Iron deficiency is the most common cause of anemia, but vitamin deficiency, kidney disease, inherited hemoglobin disorders, and other illnesses can also cause anemia. It is also possible to have a higher than normal hemoglobin level. This is usually caused by dehydration, but may also result from a variety of diseases. Treatment for the unexpected hemoglobin level will depend upon the medical cause of the problem normal value for women 12-13 gm/l. (67)

2-1-Chemicals

2-1-1-Hormones

2-1-1-1-T3(TRIIODOTHYRONINE):

Triiodothyronine(T3) levels were measured by the miniVIDAS Kit(VIDAS T3-Biomerieux-FRANCE).

2-1-1-2- T4(THYROXINE):

Thyroxine (T4)levels were measured by the miniVIDAS Kit (VIDAS T4-BIOMERIEUX-FRANCE).

2-1-1-3- TSH(THYROID-SITMULATING HORMONE):

Thyrotropin or thyroid-stimulating hormone (TSH) levels were measured by the miniVIDAS Kit (VIDAS TSH-BIOMERIEUX-FRANCE)

2-1-2- GLUCOSE:

Glucose was measured by (glucose Enzymatic colorimetric method kit-LOT (12328 E),LiNEAR Chemicals ,SPAIN.

2-1-3- URIC ACID:

URIC ACID was measured by (URICACID Enzymatic colorimetric method kit-LOT (12354 A),LiNEAR Chemicals ,SPAIN.

2-1-4-TOTAL PROTIEN:

Total Protein was measured by instrumental ATAGO(SuR-NE)Protein gm/100ml(made in japan).

2-1-5-LIPID PROFILE:

2-1-5-1- Cholesterol:

Cholesterol was measured by (cholesterol Enzymatic colorimetric method kit-LOT (12200C),LiNEAR Chemicals ,SPAIN.

2-1-5-2- Triglyceride:

Triglyceride was measured by (triglyceride Enzymatic colorimetric method kit-LOT (12327 E),LiNEAR Chemicals ,SPAIN.

2-1-5-3-HDL-Cholesterol:

HDL was measured by (HDL-CHOLESTEROL(DIFFERENTIAL PRECIPITATION) Enzymatic colorimetric method kit-LOT (1133010),LiNEAR Chemicals ,SPAIN.

2-1-5-4- VLDL-CHOLESTEROL:

VLDL was measured by using this equation : VLDL = $\frac{\text{Triglyceride}}{5}$

2-1-5-5- LDL-Cholesterol:

LDL was measured by Using this equation

LDL cholesterol = total cholesterol - (HDL cholesterol + VLDL)

This formula is valid only at TG concentration of less than 400 mg/100ml

2-1-6-Hemoglubine(Hb):

Hemoglobin(Hb)was measured by using capillary tube and instrument (MiCRO - HAEMATOCRiT CENTRIFUGE , HAWKSLEL , ENGLAND) .

2-2-Instruments:

The instrument used during this study were:

1- miniVidas device from miniVidas Company from Italy. Was used to measured the level of T3,T4,TSH.

Figure(2-1) Show the miniVidas instrument for musurement serum T3,T4 and TSH.

2-The measurements of glucose, uric acid ,cholesterol and lipid profile are done by using spectrophotometer model(CECIL-CE1011).

3-The measurements of Total Protein done by using instrumental ATAGO(SuR-NE)Protein gm/100ml (made in Japan).

Figure(2-2)show the instrument for measurement serum total protein.

4-The measurements of Hemoglobin by using MiCRO-HAEMATOCRiT CENTRIFUGE,HAWKSLEL,ENGLAND.

2-3-Subject Selection:

This study was conducted at the AL-ELWIA HOSPITAL Department of Clinical Chemistry and at the Central Public Health Laboratories Department of Hormones .All measurements were during the period (1/7/2007 to 8/4/2008)

Seventy Iraqi women were included in this study ;the study groups included Fifty pregnant women at three trimesters. And twenty non-pregnant as control group .All pregnant women introduced in this study were examined by their gynecology physician in the hospital .

All biochemical tests (glucose, uric acid, total protein, lipid profile ,Hemoglobin, Bilirubin,T3,T4,TSH) were examined in both the pregnant and non- pregnant groups.

Questionnaire form was filled by direct interview with all women&control in this study.

2.4- Blood Collection:

Ten milliliters of blood was taken from pregnants at three trimesters and non-pregnant women, some blood was used directly before separated to measured hemoglobin and the other part were placed in plane tube (no anti coagulant) left for (15 min) at room temperature, then centrifuged (at 2500 round/min for 10 min) to get the serum, which is stored at (-20^{o}c) unless used immediately. This part was divided to two part some of serum used to measured thyroid hormone (T3,T4,TSH).and other for measuring the biochemical parameters (glucose, uric acid, totalprotine ,lipid profile and Hemoglobin).

2-5-METHODS:

Hormones were measured by using miniVidas techniques.

2-5-1-miniVidus method:

The assay principle combines an enzyme immunoassay competition method with a final fluorescent detection Enzyme languid fluorescent assay (ELFA).The Solid Phase Receptacle (SPR)serves as the solid phase as well as the pipetting device for the assay .Reagents for the assay is ready-to use and predispensed in the sealed reagent strips. All of the assay steps are performed automatically by the instrument. The reaction medium is cycled in and out of the SPR several times . The sample is taken and transferred into the well containing the T3 antigen labeled with alkaline phosphatase (conjugate).Competition occurs between the antigen present in the sample and the labeled antigen for the specific anti-T3antibodies(sheep)coated on the interior of the SPR. Un bound components are eliminated during washing steps. During the final detection step, the substrate(4-Methyl-umbelliferyl phosphate) is cycled in and out of the SPR. The conjugate enzyme catalyzes the hydrolysis of this substrate into a fluorescent product (4-Methyl-umbelliferone)the fluorescence of which is measured at 450nm. The intensity of the fluorescence is inversely proportional to the concentration of antigen present in the sample . At the end of the assay. Results are automatically calculated by the instrument in relation to the calibration curve stored in memory, and then printed out. This method is used to measuring the T3,T4,TSH,hormones level are measuring by using miniVidas device

from mini Vidas Company from Italy the type of analysis is Immunophlorences.

2-6 Analysis of sample:

2-6-1-Hormones

2-6-1-1- Working procedure of T3-triiodothyronine:

T3-triiodothyronine levels were determined by the miniVidas

Kit-T3(VIDAS T3-Biomerieux-FRANCE).

The normal value (0.92-2.33)nmol/l

2-6-1-2- Working procedure of T4-THYROXINE:

T4-thyroxine levels were determined by the miniVidas

Kit-T4(.VIDAS T4-Biomerieux-FRANCE).

The normal value (60-120)nmol/l

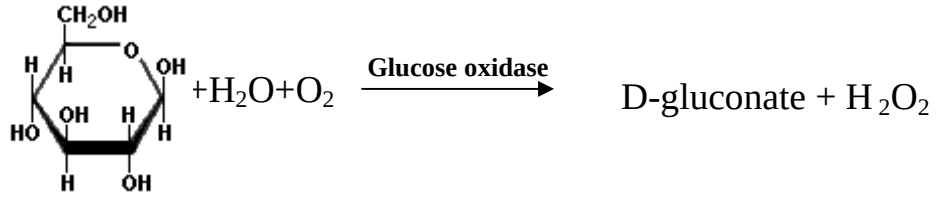
2-6-1-3-Working Procedure of TSH- Thyroid Stimulating Hormone:

TSH-Thyroid Stimulating hormone levels were determined by the miniVidas Kit (TSH Kit(VIDAS TSH -Biomerieux-FRANCE).

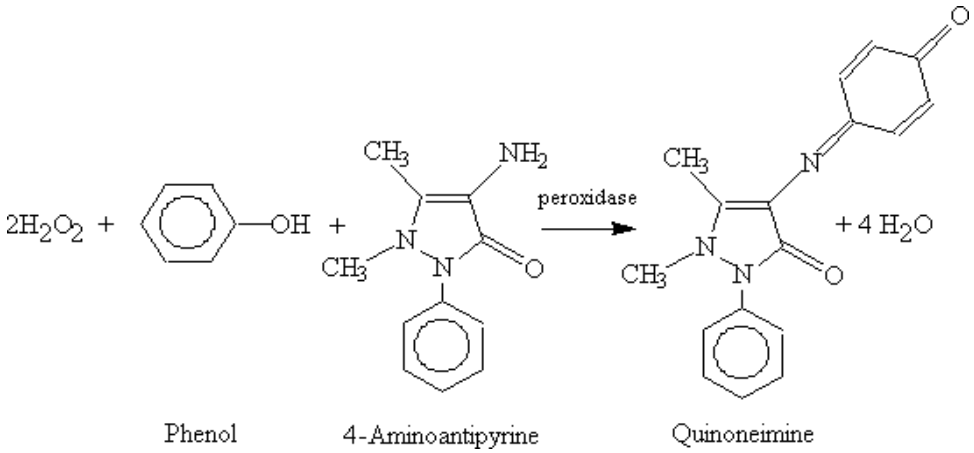
The normal value (0.25-5)μIU/ml

2-6-2-<u>Working procedure of glucose</u>:

Glucose was measured by (glucose Enzymatic colorimetric method kit-LOT (12328 E),LiNEAR Chemicals ,SPAIN .The principle of this method in the Tinder reaction, the glucose is oxidized to D-gluconate by the glucose oxidase(GOD).the hydrogen peroxide formed and reacts with phenol and 4-aminoantipyrine to form a red-violet quinoneimine dye as indicator, as the following equations:



ß-D-Glucose



Was determined spectrophotometrically at λ=500 nm , according to the following equation:

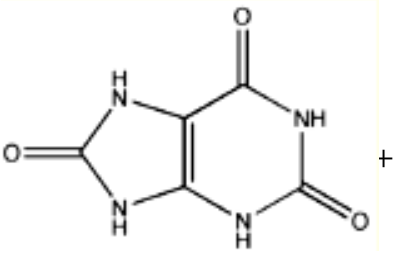
$$\text{The concentration of glucose (mg/dl)} = \frac{\text{Abs.of sample}}{\text{Abs.of standard}} * N$$

N =100 mg/dl concentration of standard glucose.

The normal range fasting is (70-105) mg/dl

2-6-3-<u>Working Procedure of Uric Acid</u> :

Uric Acid was measured by (Uric acid Enzymatic colorimetric method kit-LOT (12328 E) , LiNEAR Chemicals , SPAIN . The principle of this method is that uric acid oxidized by uricase to allantoin with formation hydrogen peroxide . The hydrogen peroxide formed and reacts with 4-aminoantpyrine (4-AA) and N-ethyl-Nsulphopropyl-m-anisidine(ADPS) to form a red-violet quinoneimine dye as indicator, as the following equations:



Uric acid

$$\text{Uric acid} + O_2 + 2H_2O \xrightarrow{\textbf{uricase}} \text{Allantoin} + H_2O_2$$

$$\text{4-aminoantipyrine} + \text{ADPS} \xrightarrow{\textbf{peroxidase}} \text{Quinoneimine} + 4H_2O$$

Was determined spectrophotometrically at λ=550 nm,according to the following equation:

$$\text{The concentration of uric acid (mg/dl)} = \frac{\text{Abs. of sample}}{\text{Abs. of standard}} * N$$

N = 6 mg/dl concentration of standard uric acid

The normal range for female is(2.6- 6.0)mg/dl (mailgrams per deciliter)

2-6-4-Working procedure of Total Protein :

Total Protein was measured by using instrumental ATAGO (SUR-NE) Protein-gm/100ml (made in Japan).Clinical refractometers Using the principles of light refraction.

The test was doing by refractometry.To do the test, a drop of serum is placed on the optical surface of the refractometer. A lid is lowered over the sample. Through an eyepiece the values were taken from a scale etched on the lens. The round optical field is split with dark above and light below at the point on the scale corresponding with the grams of total protein per 100 ml of blood. With practice this determination is quick and accurate enough.

The normal range is (6.0- 8.3) gm/dl (grams per deciliter).

2-6-5-<u>Measurment of lipid profile:</u>

2-6-5-1-<u>Working procedure of Cholesterol</u>:

Cholesterol Was measured by (cholesterol Enzymatic colorimetric method kit-LOT (12328 E),LiNEAR Chemicals ,SPAIN . The principle of this method use three enzymes: cholesterol esterase(CE),cholesterol oxidase(CO)and peroxidase(POD).in the presence of the former the mixture of phenol and 4-aminoantipyrine (4-AA) are condensed by hydrogen peroxide to form a red-violet quinoneimine dye as indicator, as the following equations:

$$\text{Cholesteryl esters} \xrightarrow{\text{Cholesterol esterase}} \text{Cholesterol + Fatty acid}$$

$$\text{Cholesterol}+O_2 \xrightarrow{\text{Cholesterol oxidase}} \text{Cholestenone}+ H_2O_2$$

$$\text{4-aminoantipyrine +Phenol} \xrightarrow{\text{peroxidase}} \text{Quinoneimine} + 4H_2O$$

Was determined spectrophotometrically at λ=500 nm,according to the following equation:

$$\text{The concentration of cholesterol (mg/dl)} = \frac{\text{Abs. of sample}}{\text{Abs. of standard}} * N$$

N = 200 mg/dl concentration of standard cholseterol.

The normal values of serum cholesterol are: 200 mg/dl.

2-6-5-2-<u>Working procedure of Triglycerides:</u>

Triglycerides Was measured by (Triglyceride Enzymatic colorimetric method kit-LOT (12328 E),LiNEAR Chemicals ,SPAIN .The principle of this method is based on the enzymatic hydrolysis by lipoprotein lipase (LPL) triglyceride to glycerol and fatty acids(FFA),the glycerol is phosphorylated by adinosintriphosphate(ATP) in the presence of glycerolkinase (GK) to form glycerol-3-phosphate (G-3-P) and adenosine diphosphate (ADP).G-3-P is oxidized by glycerophosphate oxidase (GPO) to form dihydroxyacetone phosphate (DHAP) and hydrogen peroxide .a red chromogen is produced by the peroxidase (POD)catalyzed as the following equations:

$$\text{Triglycerides} + 3\,H_2O \xrightarrow{\text{Lipoprotein lipase}} \text{Glycerol} + 3\ \text{FFA}$$

$$\text{Glycerol} + \text{ATP} \xrightarrow{\text{glycerolkinase}} \text{Glycerol-3-P} + \text{ADP}$$

$$\text{Glycerol-3-P} + O_2 \xrightarrow{\text{Glycerophosphate oxidase}} \text{DHAP} + H_2O_2$$

$$\text{4-aminoantipyrine} + \text{4 Phenol} \xrightarrow{\text{Hydrogen peroxide}} \text{Quinoneimine} + H_2O$$

Was determined spectrophotometrically at λ=500 nm,according to the following equation:

$$\text{The concentration of triglyceride (mg/dl)} = \frac{\text{Abs. of sample}}{\text{Abs. of standard}} * N$$

N = 200 mg/dl concentration of standard triglyceride. The normal values of serum triglyceride are : 150 mg/dl

2-6-5-3-<u>Working procedure of HDL</u>:

HDL was measured by (HDL-CHOLESTEROL(DIFFERENTIAL PRECIPITATION) Enzymatic colorimetric method kit-LOT (1133010),LiNEAR Chemicals ,SPAIN. The serum level of HDL-cholesterol was determined by precipitation of VLDL-cholesterol and LDL-cholesterol by the addition of phosphotungastic acid in the presence of magnesium ions (phosphotungestic acid 4 gm/dl,$MgCl_2.6H_2O$ 10gm/dl,PH 6.2.The supernatant obtained after centrifugation contains only HDL cholesterol on which the cholesterol fraction was done by the same enzymatic method mentioned in cholesterol.

The normal value (40-90)mg/dl

2-6-5-4-<u>Working procedure of LDL and VLDL</u>:

Serum level of LDL-C was calculated according to Friedwald formula. In 1972 Friedwald et al., had put a formula to calculate VLDL-C is present in serum at a concentration equal to one fifth of the Triglyceride concentration.

$$\text{VLDL} = \frac{\text{TG}}{5}$$

Therefore :

LDL cholesterol = Total cholesterol – [HDL cholesterol +VLDL]

The formula is only valid at triglyceride concentration of less than 400 mg/100 ml.

The normal value of LDL (40-130)mg/dl .

The normal value of VLDL (0-40)mg/dl.

2-7-**Working Procedure of Hemoglubin :**

Hemoglubin was measured directly after taking the sample from the pregnant , Hemoglubin was measured by using instrument MiCRO-HAEMATOCRiT CENTRIFUGE,HAWKSLEL, ENGLAND. The normal value (12-15)g/dl.

2-8-General Procedure of hormones in miniVidus:

This method was used to measured (T3,T4,TSH).

Number sufficient coatedtubes (standers, control, serum samples).

1-Allow reagents to come to room temperature for at least 30 minutes.

2-Use one ("T3","T4","TSH") strip and one ("T3","T4","TSH") SPR from the kit for each sample, control or calibrator to be tested

3- Select ("T3","T4","TSH") to enter the test code.

4-pipette 100µl for "T3",and pipette 200µl for "T4","TSH"respectively of sample, calibrator, or control in to the sample well.

5- Insert the VIDAS SPRs and strips into the positions indicated on the screen .

6-All the assay steps are performed automatically by the instrument . The assay will be completed within approximately 40 minutes.

2-9- Statistical analysis:

Statistical analysis was performed by the SPSS 12.01 statistical Package for social sciences and also Excel 2003. Data analysis was done Using chi-sequare test For tables with frequencies.

All values were expressed as Mean ± Standard Deviation of the mean (M**±SD) .**

Statistical analysis were performed using students t-test to estimate the difference between the groups pregnant and control .ANOVA-test to compare the different between groups and it is control groups and correlation regression ,taking ($P<0.05$) as the lowest limit of significance.

P value <0.05 was regarded as statistically significant.

Chapter Three Results

Fifty sample of pregnant women and twenty sample of non- pregnant as (control group) were studied . The age of the pregnant women ranges between (16-50) years. with a (mean ±SD) at three trimester:

The first trimester there was (29.27 ± 7.93) years,

The second trimester was (24.81 ±6.06) years,

The third trimester with mean age (27.15 ± 6.54) years .

The age of non- pregnant women ranges between (17-54)years with a mean ±SD (35.25 ± 10.27)years.

3.1- Thyroid hormone and biochemical tests:

3.1.1- Serum (T3)

Serum (T3) was measured using (miniVIDAS) method . Table (3-1) shows the mean ±SD of T3 at the firsttrimister was (2.22±0.33) nmol/l, at the second trimester was (3.01±0.81)nmol/l , and at the third trimester was (2.66±0.72)nmol/l.

All were higher than mean ±SD (1.95±0.29)nmol/l,of control group.

3.1.2-Serum (T4)

Serum (T4) was measured using (miniVIDAS) method. table (3-1) shows the mean ±SD of T4 at the firsttrimister was (96.62±12.88) nmol/l , at the second trimester was(106.54±20.66) nmol/l , and at the third trimester was (115.17±18.04) nmol/l.

All were higher than mean ± SD(77.42±10.55) nmol/l, of control group.

3.1.3-Serum (TSH)

Serum (TSH) was measured using (miniVIDAS) method. table (3-1) shows the mean ± SD of TSH at the firsttrimister was (1.06±0.75)µIU/l , at the second trimester was (0.83±0.52) µIU/ml , and at the third trimester was (1.52±1.50) µIU/l .

All were lower than mean ±SD (1.67±1.13) µIU/ml , of control group.

3.1.4- Serum Glucose

Table(3-1) shows that a mean ±SD at the first, second, third trimester was (72.27±9.75),(89.64±12.23),(86.44±34.48) mg/dl respectively less than mean ±SD (92.19±24.82) mg/dl of the control group.

3.1.5- Serum uric acid

Table (3-1) shows that a mean ±SD at the first and second trimester have mean ±SD(3.28±1.60),(3.02±1.59) mg/dl respectively closes to the mean ±SD(3.58±1.19) mg/ dl of control group, but at the third trimester the mean ±SD(4.04±1.66) mg/dl higher than mean ±SD of the control group.

3.1.6- Serum total protein

Table (3-1) shows that mean ±SD at the first trimester was close mean ±SD(7.23±0.60) mg/dl to control group, while at second and third trimester the mean ±SD(6.75±0.67),(6.81±0.59) mg/dl respectively was less than mean ±SD(7.21±0.66) mg/dl of control group.

3.1.7- lipid profile

3.1.7.1- Cholesterol

Table (3-1) shows that mean ±SD at the first trimester was(107.56±32.96) mg/dl less than mean ±SD(118.59±27.36) mg/dl of control group but at the second and third trimester the mean ±SDwere (143.64±32.42), (153.19±28.08) mg/dl .

Respectively higher than mean ±SD of the control group.

3.1.7.2- Triglyceride

Table(3-1) shows that at the first and second trimester were (90.09±38.74) , (129.64±64.92) mg/dl respectively less than mean ±SD (160.84±98.28) mg/dl of control group. while at the third trimester the mean ±SD was (224.07±120.80) mg/dl higher than mean ±SD of the control group.

3.1.7.3- HDL (high-density lipoprotein)

Table(3-1)show that at the first , second and third trimester were (50.55 ±15.36) mg/dl, (45.73±14.71) , (46.35±13.34) mg/dl respectively higher than mean ±SD (41.0±13.00) mg/dl of control group .

3.1.7.4- LDL(low density lipoprotein)

Table(3-1) shows that at the first trimester was (37.02±33.42) mg/dl less than mean ±SD(45.42±28.75)mg/dl of the control group ,while at the second trimester and third trimester were (71.98±30.43) ,(62.03±32.43) mg/dl respectively higher than mean ±SD of the control group.

3.1.7.5- VLDL(very low density lipoprotein):

Table(3-1) shows that mean ±SD at the first and second trimester was (18.38±7.71),(25.94±12.97) mg/dl respectively less than mean ±SD of the control group, while at the third trimester was (44.83±24.71) mg/dl higher than mean ±SD was (32.17±19.65) mg/dl of the control group.

3.1.7.6-CHO/HDL and LDL/HDL

Table(3-1)shows that mean ±SD at the firsttrimister of the ratio CHO/HDL and ratio LDL/HDL were (2.34±1.09), (0.89±0.89) mg/dl respectively less than mean ±SD (3.29±1.74), (1.24±1.24) mg/dl respectively of the control group, while at the second and third trimester were less mean ±SD (3.46±1.43), (3.53±1.08), (1.87±1.23), (1.57±0.96) mg/dl respectively to the mean ±SD of control group.

CHO/HDL the normal value below > 4.5mg/dl

LDL/HDL the normal value below >3.5 mg/dl .

3.2- Hemoglobin (Hb)

Table(3-1)shows that mean ±SD at the firsttrimister and second trimester were (10.81±0.87),(10.91±1.22) mg/dl respectively less than mean ±SD (11.81±0.75) mg/dl of the control group but at the thirdtrimister have the closes mean ±SD(11.3±1.28) mg/dl to the mean ±SDof the control group.

Table(3-1)The mean and standerd divation of thyroid hormone and biochemical parameter for both pregnant and non- pregnant women

Parameter	Glucose mg/dl	Uric acid mg/dl	Total Protein mg/dl	Cholesterol mg/dl	Triglyceri mg/dl	HDL mg/dl	VLDL mg/dl	LDL mg/dl	CHOL/ HDL mg/dl	LDL/ HDL mg/dl	Hb mg/dl	T3 nmol/l	T4 nmol/l	TSH µIU/l
FIRST TRIMISTER	72.27 ± 9.75	3.28 ± 1.60	7.23 ± 0.60	107.56 ± 32.96	90.09 ± 38.74	50.55 ± 15.36	18.38 ± 7.71	37.02 ± 33.42	2.34 ± 1.09	0.89 ± 0.89	10.81 ± 0.87	2.22 ± 0.33	96.62 ± 12.88	1.06 ± 0.75
SECOND TRIMISTER	89.64 ± 12.23	3.02 ± 1.59	6.75 ± 0.67	143.64 ± 32.42	129.64 ± 64.92	45.73 ± 14.71	25.94 ± 12.97	71.98 ± 30.43	3.46 ± 1.43	1.87 ± 1.23	10.91 ± 1.22	3.01 ± 0.81	106.54 ± 20.66	0.83 ± 0.52
THIRD TRIMISTER	86.44 ± 34.48	4.04 ± 1.66	6.81 ± 0.59	153.19 ± 28.08	224.07 ± 120.80	46.35 ± 13.34	44.83 ± 24.71	62.03 ± 32.43	3.53 ± 1.08	1.57 ± 0.96	11.31 ± 1.28	2.66 ± 0.72	115.17 ± 18.04	1.52 ± 1.50
CONTROL	92.19 ± 24.82	3.58 ± 1.19	7.21 ± 0.66	118.59 ± 27.36	160.84 ± 98.28	41.0 ± 13.00	32.17 ± 19.65	45.42 ± 28.75	3.29 ± 1.74	1.24 ± 1.24	11.81 ± 0.75	1.95 ± 0.29	77.42 ± 10.55	1.67 ± 1.13

3-3 Biochemical parameter during pregnancy:

3-3-1- Hormones:

Serum hormones concentration using (MiniVidas) to measuring T3,T4,TSH. Were compared between the study groups and control groups. using analysis of variance t-test of significant as table(3-2) . The elevated levels of thyroid hormone pregnant were found to have significantly higher levels in serum T4 ($p<0.001$) at the first trimesters but not significantly T3,TSH($P=0.122$),($P=0.138$) respectively compared with control group.

Twenty pregnant women in the second trimesters and control using analysis of variance t-test of significant as table (3-2).The elevated level of thyroid hormones in pregnant were found to have significantly higher levels in serum T3,T4, ($p<0.001$) and TSH($P<0.05$) compared with control group.

Twenty pregnant women in the third trimesters as Table(3-2).The elevated levels of thyroid hormone in pregnant were found to have significantly higher levels in serum T3,T4 ($P<0.001$) but non significantly for TSH $P=(0.724)$between the third trimester and control group.

3-3-2-Biochemical test during pregnancy:

Serum of glucose, uric acid, total protein,lipd profile, and hemoglobin were compared between the study groups(pregnant at the firsttrimister) and control groups,using analysis of variance t-test of significant as Table(3-3) shows that Hemoglobin have significantly higher ($P<0.001$) but glucose significantly lower ($P<0.05$) compared with control group. But non significantly in serum uric acid($P=0.178$),Total

protein(P=0.061),lipid profile were not significantly at the first trimester for pregnant women compared with control group.

As Table (3-4) in this study shows that at the second trimester the cholesterol,LDL,Hemoglobin were have significantly lower (P<0.05), but non significant in serum glucose(p=0.826),uric acid(P=0.233), Total protein (P=0.346) ,Triglyceride (P=0.229), HDL (P=0.287) , VLDL (P=0.229) , CHO/HDL (P=0.846), LDL/HDL (P=0.404) during the second trimester for pregnant women compared with control group. Also at the third trimester Table (3-5)shows that significantly higher were found in serum cholesterol, triglyceride, LDL (P<0.001),and significantly lower were found in serum total protein, VLDL (P<0.05), but non significant in serum glucose (P=0.406) ,uric acid (p=0.114) ,HDL (P=0.410) ,CHO/HDL (P=0.431), LDL/HD(P=0.518), Hemoglobin (P=0.684) at the third trimester for pregnant women compared with control group.

Table (3-2) comparison of hormones levels between the pregnant women in three trimester and its control not pregnant women's.

Hormones	Firsttrimister mean ± SD	Control mean ± SD	P-value
T3(nmol/ml)	2.22± 0.33	1.95±0.29	0.122
T4(nmol/l)	96.62±12.88	77.42±10.55	0.003**
TSH(µIU/ml)	1.06±0.75	1.67±1.13	0.138
Hormones	**Secondtrimister mean ± SD**	**Control mean ± SD**	**P-value**
T3(nmol/ml)	3.01±0.81	1.95±0.29	0.006**
T4(nmol/l)	106.54±20.66	77.42±10.55	0.001**
TSH(µIU/ml)	0.83±0.52	1.67±1.13	0.023*
Hormones	**Thirdtrimister mean ± SD**	**Control mean ± SD**	**P-value**
T3(nmol/ml)	2.66±0.72	1.95±0.29	0.017*
T4(nmol/l)	115.17±18.04	77.42±10.55	0.000**
TSH(µIU/ml)	1.52±1.59	1.67±1.13	0.724

Table (3-3) comparism of biochemical parameter (glucose, uric acid, total protein,lipid profile and hemoglobin) between the pregnant At the First trimester and its control.

parameters	Firsttrimister mean ± SD	Control mean ± SD	P-value
Glucose (mg/dl)	72.27±9.75	92.19±24.82	0.013*
Uric acid(mg/dl)	3.28±1.60	3.58±1.19	0.178
Total protein(mg/dl)	7.23±0.60	7.21±0.66	0.061
Cholesterol (mg/dl)	107.56±32.96	118.59±27.36	0.659
Triglyceride (mg/dl)	90.09±38.74	160.84±98.28	0.066
HDL(mg/dl)	50.55±15.36	41.0±13.00	0.126
LDL(mg/dl)	37.02±33.42	45.42±28.75	0.071
VLDL(mg/dl)	18.38±7.71	32.17±19.65	0.975
CHO/HDL(mg/dl)	2.34±1.09	3.29±1.74	0.267
LDL/HDL(mg/dl)	0.89±0.89	1.24±1.24	0.988
Hemoglobin (g/dl)	10.81±0.87	11.81±0.75	0.003**

****=highly significant at P<0.001. *=significant difference at P<0.05.**

Table (3-4) comparism of biochemical parameter (glucose, uric acid, total protein, lipid profile and hemoglobin)between the pregnant At the Second trimester and its control.

parameters	secondtrimister mean ± SD	Control mean ± SD	P-value
Glucose(mg/dl)	89.64 ±12.23	92.19±24.82	0.826
Uric acid(mg/dl)	3.02 ±1.59	3.58±1.19	0.233
Total protein(mg/dl)	6.75 ±0.67	7.21±0.66	0.346
cholesterol(mg/dl)	143.64 ±32.42	118.59±27.36	0.039*
triglyceride(mg/dl)	129.64 ±64.92	160.84±98.28	0.229
HDL(mg/dl)	45.73 ±14.71	41.0±13.00	0.287
LDL(mg/dl)	71.98±30.43	45.42±28.75	0.013*
VLDL(mg/dl)	25.94 ±12.97	32.17±19.65	0.229
CHO/HDL(mg/dl)	3.46±1.43	3.29±1.74	0.846
LDL/HDL(mg/dl)	1.87±1.23	1.24±1.24	0.404
Hemoglobin(g/l)	10.91±1.22	11.81±0.75	0.024*

Table (3-5) comparism of biochemical parameter (glucose, uric acid, total protin,lipid profile and hemoglobin)between the pregnant At the Third trimester and its control

parameters	Thirdtrimister mean ± SD	Control mean ± SD	P-value
glucose(mg/dl)	86.44±34.48	92.19±24.82	0.406
Uric acid(mg/dl)	4.04±1.66	3.58±1.19	0.114
Total protein(mg/dl)	6.81±0.59	7.21±0.66	0.043*
cholesterol(mg/dl)	153.19±28.08	118.59±27.36	0.001**
triglyceride(mg/dl)	224.07±120.80	160.84±98.28	0.008**
HDL(mg/dl)	46.35±13.34	41.0±13.00	0.410
LDL(mg/dl)	62.03±32.43	45.42±28.75	0.008**
VLDL(mg/dl)	44.83±24.71	32.17±19.65	0.033*
CHO/HDL(mg/dl)	3.53±1.08	3.29±1.74	0.431
LDL/HDL(mg/dl)	1.57±0.96	1.24±1.24	0.518
hemoglobin	11.31±1.28	11.81±0.75	0.684

****=highly significant at P<0.001.** ***=significant difference at P<0.05.**

3.4-**Relation between thyroid hormone and other biochemical parameter at the first trimester for pregnant women**

The correlation test done by using Excel 2003,according to the program ± (0.15-0.35) consider as weak correlation, ± (0.35-0.5)consider as correlation ± (0.5-1) consider as strong correlation. The possible correlations at the firsttrimister between Thyroid Hormone (T3,T4,TSH) and Biochemical parameters were investigated by the value of correlation coefficient. Result for the pregnant women at the first trimester

Table(3-6) Shows that There is a highly significance at P-value ($P<0.001$) with a positive correlation between cholesterol with LDL ($r=0.835$),and triglyceride with VLDL($r=0.988$),and HDL with Hb($r=0.790$),and LDL with ratio CHO/HDL ($r=0.817$), also with ratio LDL/HDL($r=0.885$),and between ratio CHO/HDL with ratio LDL/HDL ($r=0.974$). And a highly significance negative correlation between ratio CHO/HDL with Hb ($r=-0.852$),and ratio LDL/HDL with Hb ($r=-0.800$). And a significance at ($P<0.05$) positive correlation between cholesterol with height ($r=0.640$), glucose ($r=0.662$) ,Uric acid ($r=0.702$) , ratio CHO/HDL($r=0.599$), ratio LDL/HDL($r=0.620$), also significant negative correlation between HDL with CHO/HDL ($r = -0.708$) ,

LDL/HDL($r=-0.677$) ,and also TSH with glucose($r=0.667$),

uric acid($r=0.619$),cholesterol($r=0.597$).also T3 negative correlation not significance with glucose($r=-0.399$), uric acid($r=-0.372$), also T4 with glucose($r=-0.482$), also positive correlation not significant between TSH with triglyceride($r=0.522$),but TSH have weak negative correlation with T3($r=-0.235$),T4($r=-0.312$).

Table(3-6) Correlation between Thyroid Hormone and other Biochemical parameters at The FIRSTTRIMISTER of Pregnant Woman

	firsttrimist	Age	weight	height	glucose	uricacid	totalproten	cholesterol	triglyceride	HDL	LDL	VLDL	CHO/HDL	LDL/HDL	HB	T3	T4
age	-0.08																
Weight	0.308	0.162															
Height	0.47	-0.398	0.084														
Glucose	-0.081	0.102	0.051	0.254													
uric acid	-0.099	-0.242	0.315	0.146	0.457*												
totalproten	0.263	0.159	0.553	0.258	-0.308	-0.021											
cholesterol	0.253	-0.339	0.206	0.640*	0.662*	0.702*	-0.208										
triglyceride	-0.159	0.418	0.5	-0.05	0.287	0.489*	0.086	0.365									
HDL	0.381	0.197	0.449	0.183	-0.203	-0.181	0.119	0.003	0.106								
LDL	0.197	-0.44	-0.108	0.549	0.657*	0.58*	-0.213	0.835**	0.02	-0.465							
VLDL	-0.16	0.425	0.443	-0.071	0.324	0.435	-0.002	0.35	0.988**	0.105	0.007						
CHO/HDL	0.023	-0.332	-0.093	0.413	0.42*	0.472	0.015	0.599*	0.251	0.708*	0.817**	0.233					
LDL/HDL	0.164	-0.375	-0.129	0.498	0.44	0.416	0.02	0.620*	0.082	0.677*	0.885**	0.063	0.974**				
HB	0.181	0.311	0.127	-0.119	-0.076	-0.46	-0.161	-0.284	-0.172	0.790**	-0.569	-0.103	0.852**	0.800**			
T3	0.571	-0.249	0.358	0.356	-0.399	-0.372	0.335	-0.06	-0.022	0.444	-0.241	-0.011	-0.108	-0.094	0.191		
T4	0.314	-0.154	0.161	-0.058	-0.482*	-0.177	-0.22	0.02	0.003	0.357	-0.159	0.002	-0.189	-0.186	0.203	0.295	
TSH	0.159	0.214	0.11	0.027	0.667*	0.619*	-0.268	0.597*	0.522	-0.12	0.512	0.529*	0.383	0.367	-0.235	-0.235	-0.312

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** = Strong Correlation is significant at P<0.001. * =Correlation is significant at p<0.05.

3.5- Relation between thyroid hormone and other biochemical parameters at the second trimester for pregnant women

Clinical investigation from the pregnant women at the second trimester as Table(3-7) shows that a highly significant positive correlation at ($P<0.001$) between cholesterol with LDL($r=0.761$),also triglyceride with VLDL($r=1.000$), and ratio CHO/HDL with LDL($r=0.815$),and between LDL/HDL with LDL ($r=0.832$) ,CHO/HDL ($r=0.984$),and between TSH with height ($r=0.765$), Also there are highly significant negative correlation between total protein with secondtrimister ($r=-0.747$) , ratio LDL/HDL with HDL($r=-0.824$),also ratio CHO/HDL with HDL($r=-0.811$),

And there is significantly positive correlation at ($p<0.05$) between cholesterol with triglyceride ($r=0.672$),T3 ($r=0.631$),VLDL ($r=0.671$),

Also between T3 with VLDL($r=0.630$),and between T4 with T3($r=0.646$).also there are positive correlation but non significant between Hb with glucose($r=0.493$),and TSH with glucose($r=0.522$),uric acid ($r=0.539$),and also there is a negative correlation but non significant between T3 with uric acid ($r=-0.431$),total protein($r=-0.463$),and T4 with total protein ($r=-0.499$), and TSH with triglyceride(-0.535), T3($r=-0.521$).

And also weak negative correlation between T3 with glucose ($r=-0.305$), and weak positive correlation between TSH with T4($r=0.058$).

Table(3-7)Correlation between Thyroid Hormone and other Biochemical parameters at The Secondtrimister of pregnant women.

	2ndtrimist	Age	weight	height	glucose	uricacid	totalproten	cholester	triglyceri	HDL	LDL	VLDL	CHO/HDL	LDL/HDL	HB	T3	T4
age	0.137																
weight	0.152	0.336															
height	0.291	-0.392	0.257														
glucose	-0.02	0.29	0.241	0.424													
uric acid	0.275	0.173	0.107	0.392	0.116												
totalproten	0.747**	0.135	0.163	-0.424	-0.051	-0.412											
cholesterol	0.428	0.389	0.471	-0.09	-0.298	0.209	-0.28										
triglyceride	0.41	0.059	0.197	-0.411	-0.47*	-0.125	-0.259	0.672*									
HDL	0.189	0.381	0.364	-0.26	0.019	-0.018	0.283	0.036	0.236								
LDL	0.19	0.206	0.242	0.205	-0.127	0.284	-0.324	0.761**	0.175	-0.545							
VLDL	0.41	0.06	0.198	-0.412	-0.469	-0.126	-0.259	0.671*	1.000**	0.237	0.174						
CHO/HDL	0.1	-0.026	-0.024	0.163	-0.03	-0.021	-0.452	0.441	0.11	0.811**	0.815**	0.109					
LDL/HDL	0.02	-0.009	-0.002	0.228	0.011	0.019	-0.379	0.391	-0.04	0.824**	0.832**	-0.041	0.984**				
HB	0.133	0.443	0.614*	0.39	0.493*	0.21	-0.068	0.194	-0.301	-0.157	0.411	-0.3	0.356	0.421*			
T3	0.351	-0.39	-0.302	-0.097	-0.305	-0.431	-0.463	0.298	0.631*	-0.213	0.151	0.630*	0.37	0.241	-0.49		
T4	0.429	-0.527	-0.341	0.479	0.028	-0.245	-0.499	-0.097	-0.007	-0.206	0.00	-0.008	0.154	0.119	-0.22	0.646*	
TSH	-0.1	-0.191	0.309	0.765**	0.522*	0.539*	0.026	-0.253	-0.535*	0.018	-0.05	-0.535	-0.26	-0.165	0.341	-0.521	0.058

** =Strong Correlation , * =Correlation

3.6-**Relation between thyroid hormone and other biochemical parameters at the Third trimester for pregnant women**

Clinical investigation from pregnant women at the third trimester Table(3-8)shows that thirdtrimister is highly significant($P<0.001$)positive correlation between triglyceride with VLDL(r=1.000),and also LDL with cholesterol(r=0.702), ratio LDL/HDL(r=0.693), and uric acid with T3(r=0.542),age (r=0.620), and also between glucose with height (0.517),and a highly significant negative correlation between HDL with ratio CHO/HDL(r=-0.775),also glucose with thirdtrimister(r=-0.574).

And there are found significant positive correlation at ($P<0.05$), between glucose with triglyceride (r=0.464) ,VLDL(r=0.463) ,also between cholesterol with total protein (r=0.393) , ratio CHO/HDL (r=0.467) , ratio LDL/HDL (r=0.404) , and also between triglyceride with uric acid (r=0.382),LDL(r=0.470),and between VLDL with uric acid (r=0.381), and between ratio CHO/HDL with LDL(r=0.450) , ratio LDL/HDL (r=0.397) , there was a significant negative correlation between glucose with T3(r=-0.414),TSH(r=-0.418), also triglyceride with ratio LDL/HDL(r=-0.451),T4(r=-0.384), and between VLDL with LDL (r=-0.472), ratio LDL/HDL(r=-0.452),T4(r=-0.384).There are positive correlation but non significant between glucose with uric acid(r=0.321), and total protein with triglyceride (r=0.359).There was a weak negative correlation not significant between TSH with uric Acid (r=-0.142),and also weak positive correlation not significant between T4with Hb(r=0.121),T3(r=0.147),and also TSH with T3(r=0.176).

Table(3-8)Correlation between Thyroid Hormone and other Biochemical parameters at The THIRDTRIMISTER of pregnant women.

	thirdtrimis	Age	Wight	height	glucose	uricacid	totalproten	cholester	triglyceri	HDL	VLDL	LDL	CHO/HDL	LDL/HDL	HB	T3	T4
thirdtrimis																	
age	0.124																
Wight	0.081	0.436*															
height	0.525**	0.275	0.359														
glucose	0.574**	0.118	0.057	0.517**													
uricacid	-0.146	0.620**	0.154	-0.127	0.321												
totalproten	0.155	0.061	0.348	-0.056	-0.035	0.013											
cholesterol	-0.233	0.051	0.189	0.189	0.088	0.06	0.393*										
triglyceride	-0.306	0.399*	0.464*	-0.275	0.464*	0.382	0.359	0.171									
HDL	0.125	-0.373	-0.263	0.14	-0.014	-0.34	0.156	0.093	-0.31								
VLDL	-0.305	0.398*	0.463*	-0.275	0.463*	0.381	0.359	0.169	1.000**	-0.31							
LDL	-0.025	-0.099	-0.073	0.312	-0.264	-0.093	0.009	0.702**	0.470*	-0.1	-0.472*						
CHO/HDL	-0.139	0.358	0.396*	0.038	-0.01	0.323	0.109	0.467*	0.368	0.775**	0.367	0.450*					
LDL/HDL	0.194	-0.074	-0.014	0.402*	-0.351	-0.044	-0.142	0.404*	0.451*	-0.017	-0.452*	0.693**	0.397*				
HB	-0.225	0.027	0.076	-0.175	0.354	0.089	0.011	0.176	-0.186	0.096	-0.188	0.253	0.102	0.315			
T3	0.237	-0.101	0.305	0.222	0.414*	0.542**	0.16	0.038	-0.04	-0.19	-0.04	0.141	0.169	-0.023	-0.303		
T4	0.112	0.167	-0.338	-0.092	-0.152	0.091	-0.292	-0.132	-0.384	-0.116	-0.384	0.221	0.02	0.056	0.121	0.147	
TSH	-0.034	-0.18	-0.139	0.082	0.418*	-0.142	0.156	-0.094	-0.108	0.063	-0.108	-0.027	-0.088	-0.046	-0.064	0.176	-0.122

** =Strong correlation , * =correlation

3.7-Relation between Thyroid Hormone and other Biochemical parameters of the non-pregnant women:

Results for the non-pregnant women as Table(3-9) .The highly significant positive correlation at ($P<0.001$) was found between triglyceride with VLDL(r=1.000),and cholesterol with LDL(r=0.681), between LDL with ratio CHO/HDL(r=0.742), ratio LDL/HDL(0.746), between ratio LDL/HDL with ratio CHO/HDL(r=0.891).

Also highly significant negative correlation between ratio CHO/HDL with HDL(r=-0.790) , and between ratio LDL/HDL with HDL(r=-0.627), TSH with T3(r=-0.632).There was significant positive correlation at ($P<0.05$) between glucose with weight (r=0.49),Hb(r=0.511), between uric acid with triglyceride (r=0.514), VLDL(r=0.514) , and also height with triglyceride(r=0.557) ,VLDL(r=0.557) ,between total protein with age(r=0.523).

There was significant negative correlation between uric acid with Total protein (r=-0.514) ,also there was a positive correlation non significant between cholesterol with triglyceride(r=0.456) ,There was a negative correlation non significant between T4 with Hb(r=-0.394),TSH with T4(r=-0.35),there was a weak positive correlation between TSH with total protein(r=0.238),and weak negative correlation between T4 with triglyceride (r=0.181).

Table(3-9)Correlation between Thyroid Hormone and other Biochemical parameters at The Control group(not pregnant women.).

	age	Wight	hight	glucose	uricacid	totalproten	cholester	triglyceri	HDL	VLDL	LDL	CHO/HDL	LDL/HDL	HB	T3	T4
weight	0.272															
height	0.096	0.24														
glucose	0.263	0.49*	-0.178													
uric acid	-0.025	0.318	0.294	-0.081												
totalproten	0.523*	0.196	-0.315	0.258	0.580*											
cholesterol	0.044	0.423*	-0.148	0.327	0.267	0.05										
triglyceride	0.106	0.383	0.557*	0.113	0.514*	-0.202	0.456*									
HDL	0.202	-0.153	-0.161	0.27	-0.417*	0.288	-0.09	-0.099								
VLDL	0.106	0.383	0.557*	0.113	0.514*	-0.202	0.456*	1.000**	-0.099							
LDL	-0.123	0.21	-0.448*	0.111	0.09	0.056	0.681**	-0.205	-0.47*	-0.205						
CHO/HDL	-0.092	0.308	-0.064	-0.074	0.389	-0.041	0.571*	0.231	0.790**	0.231	0.742**					
LDL/HDL	-0.036	0.277	-0.157	-0.098	0.197	0.172	0.513*	0.038	0.627**	0.038	0.746**	0.891**				
HB	0.387	0.384	-0.009	0.511*	0.029	0.255	-0.004	-0.109	-0.062	-0.109	0.098	0.099	0.101			
T3	-0.139	0.342	-0.135	0.047	-0.154	0.294	0.097	-0.442*	-0.235	-0.442*	0.501*	0.225	0.426*	0.252		
T4	-0.073	-0.055	0.028	-0.07	0.007	-0.003	0.224	0.181	-0.199	0.181	0.179	0.281	0.22	-0.394	-0.06	
TSH	0.321	-0.185	-0.174	-0.02	-0.125	0.238	-0.067	0.048	0.446*	0.048	-0.298	-0.202	-0.252	0.001	0.632**	-0.35

**** =Strong Correlation**
*** = Correlation**

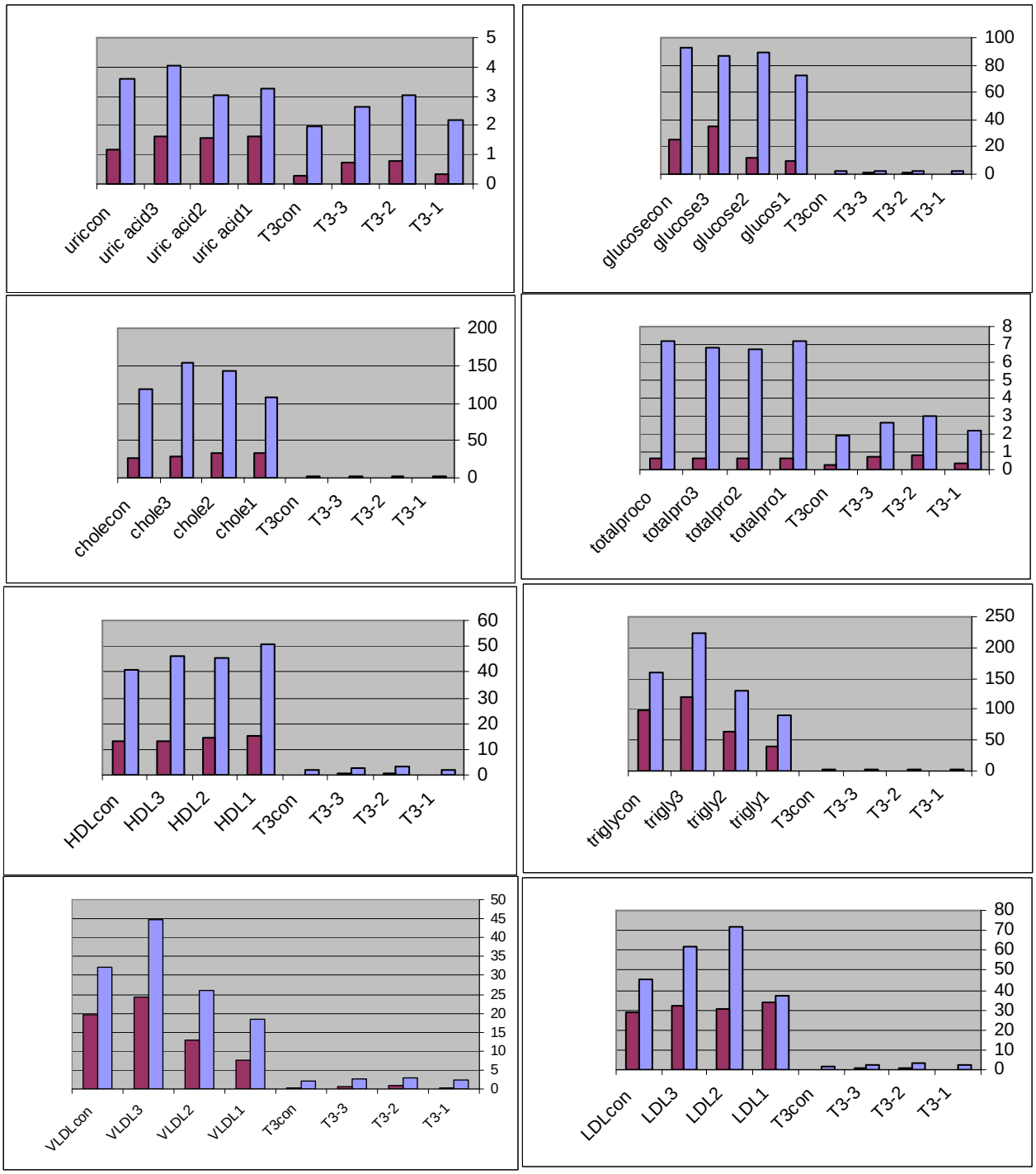
3.8-The Correlation between T3,T4 and TSH with biochemical parameters in pregnant at three trimester and non pregnant women .

In this study figures (3-1) shows that mean ± SD for T3 hormone in pregnant women at there three trimester were higher than the T3 level in control group. while it was found that the (glucose , Hemoglobin) at three trimester lower than the control group. Also it was found that the (uric acid &cholesterol &triglyceride &CHO/HDL &VLDL) . in the third trimester was higher than the (uric acid &cholesterol &triglyceride & CHO/HDL &VLDL) level in the first and secondtrimister as well as in control group. And also it was found that the (total protein &HDL) in the first trimester was higher than the(total protein &HDL) level at the second and third trimester as well as control group .And also it was found (LDL & LDL/HDL) in the second trimester was higher than the (LDL& LDL/HDL) level at the first and third as well as control group.

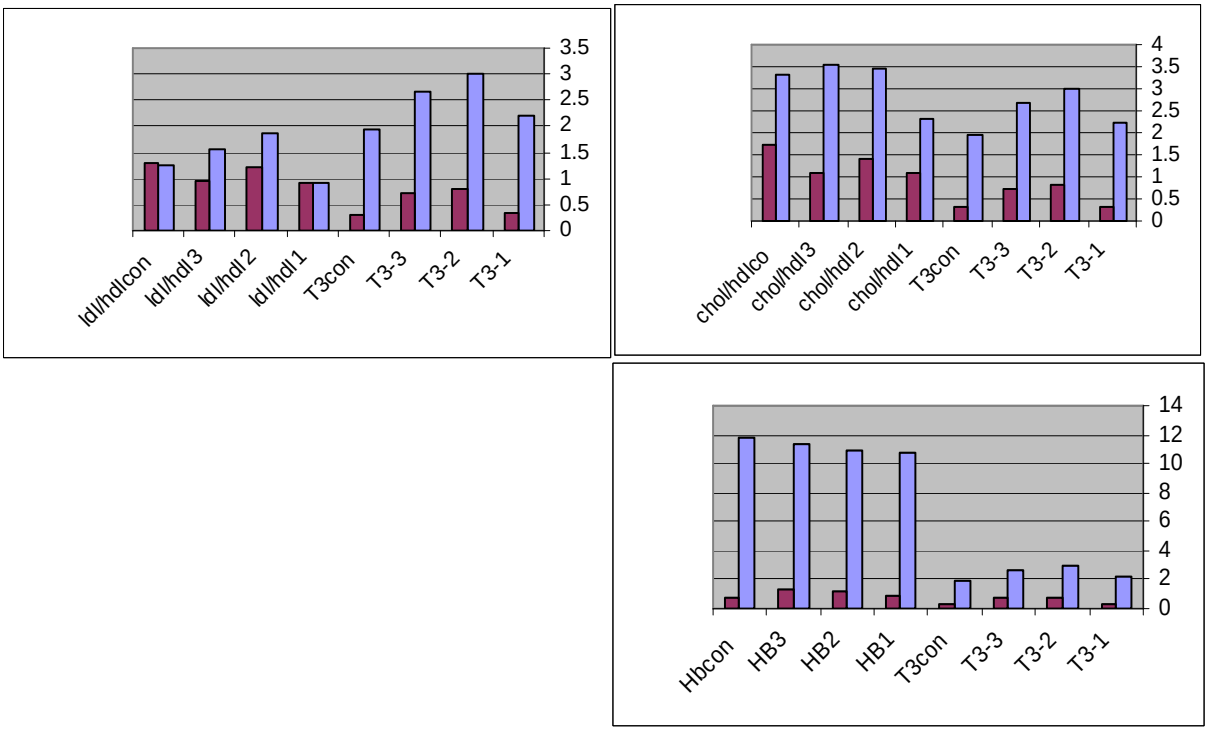
Figure(3-2) showes that mean ± SD for T4 (thyroxin) hormone in pregnant women at three trimester were higher than the T4 level in control group. while it was found that the (glucose) at three trimester were lower than the (glucose, Hemoglobin) level in the control group. And also it was found that the (uric acid & Total protein & CHO/HDL& LDL/HDL)were all in three trimester closes to the control group. and also it was found that the (cholesterol & triglyceride &VLDL)at third trimester was higher than second and first trimester as well as control group.

While HDL at the first trimester was higher than second and third trimester as well as the control group. And also LDL at the second trimester higher than first and third trimester as well as control group.

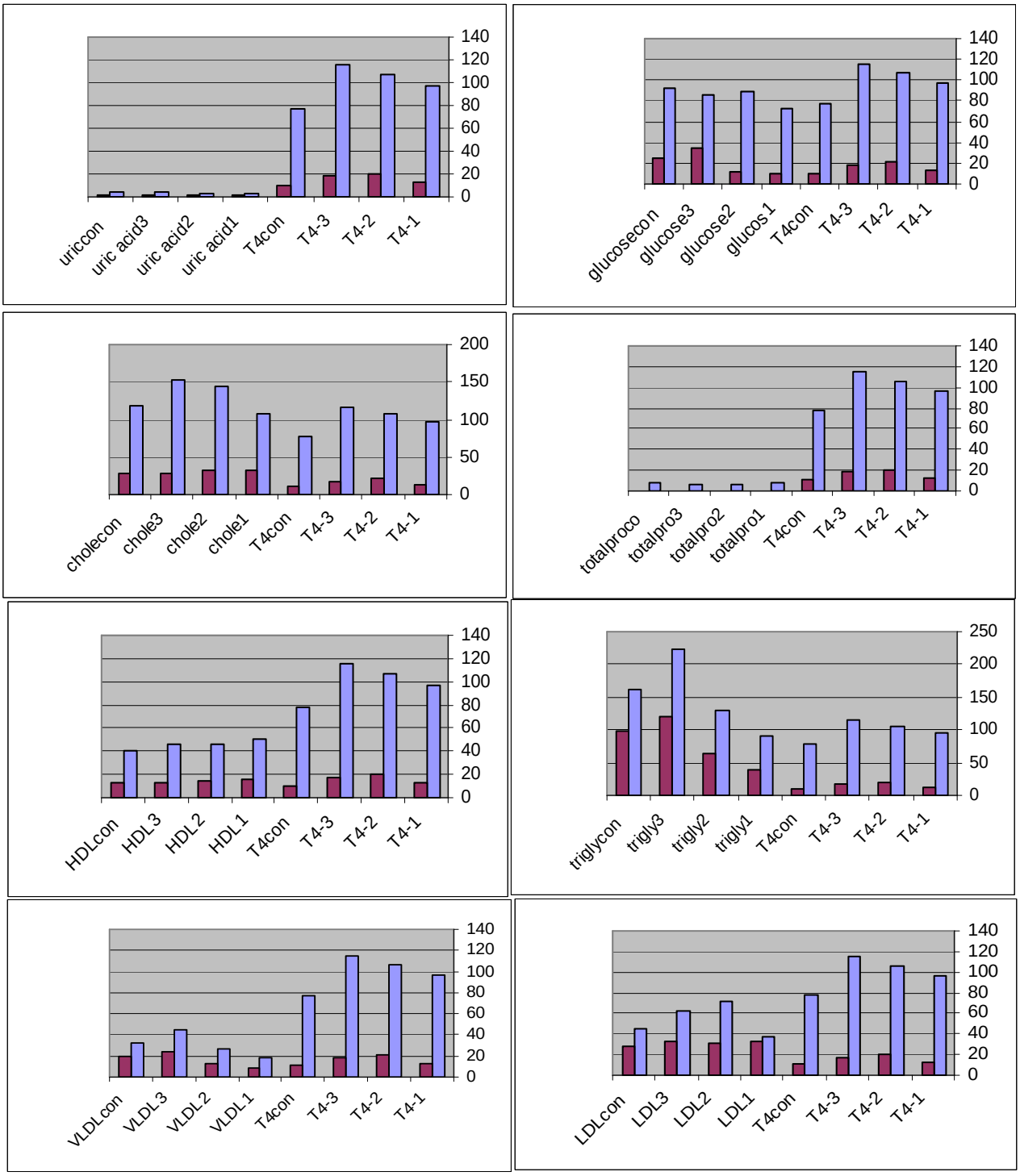
Figure (3-3) showes that the mean ± SD for TSH hormone in pregnant women at there three trimester were lower than the TSH level in control group .while(glucose , Hemoglobin) at three trimester lower than the control group . and also(uric acid & cholesterol& triglyceride & VLDL& CHO/HDL) at third trimester higher than first and second trimester as well as control group. (total protein&HDL) at the first trimester higher than the second and third trimester as well as control group. And also(LDL&LDL/HDL) at second trimester higher than the first and third trimester as well as control group.



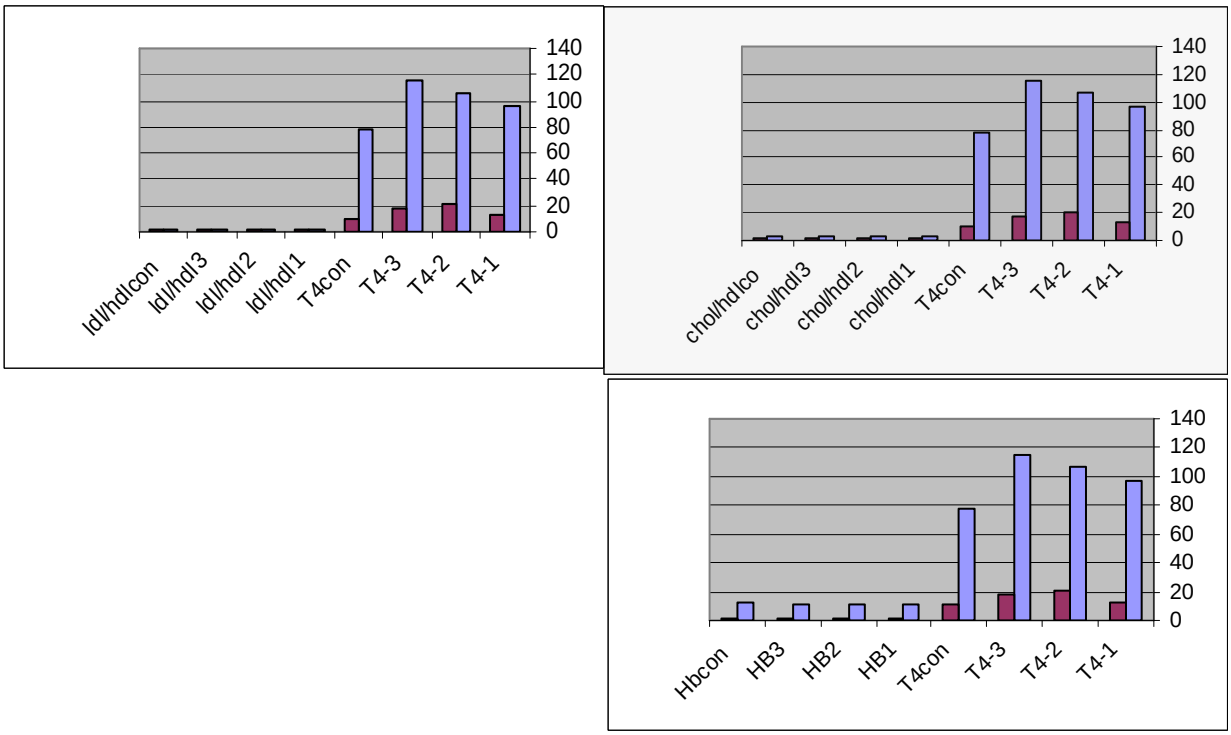
Figure(3-1)Histogram of **(mean ± SD)**
for Triiodthyronine (T3) and biochemical parameters.



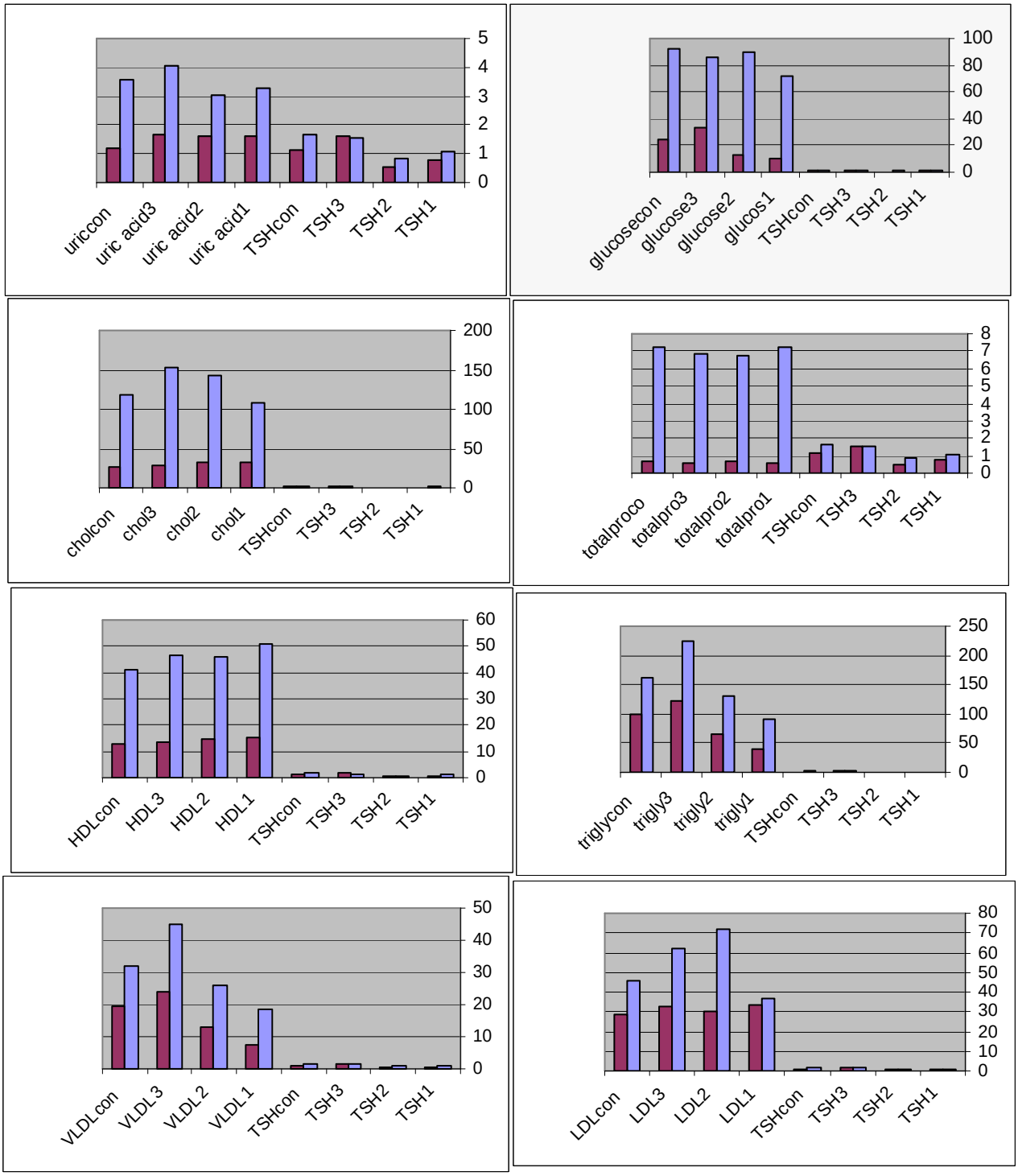
Figure(3-1)Histogram of **(mean ± SD)**
the Triiodthyronine (T3) and biochemical parameters.



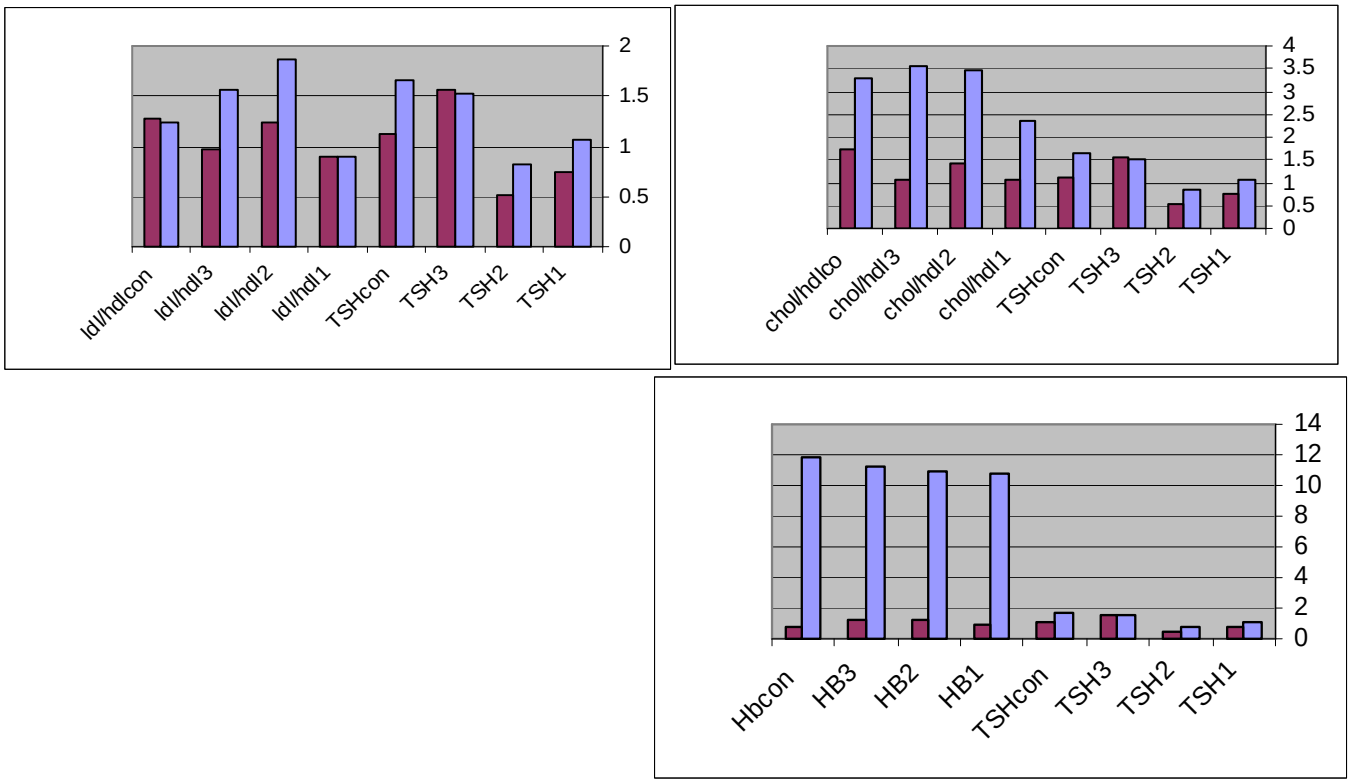
Figure(3-2)Histogram of**(mean ± SD)**
for the thyroxin (T4) and biochemical parameters.



Figure(3-2)Histogram of **(mean ± SD)**
for the thyroxin (T4) and biochemical parameters.



Figure(3-3)Histogram of **(mean ± SD)**
for the thyroid –stimulating hormone (TSH) and biochemical parameters.



Figure(3-3)Histogram of **(mean ± SD)**
for the thyroid –stimulating hormone (TSH) and biochemical parameters.

Chapter Four Discussion

Discussion:

Pregnancy results in a number of important physiological and hormonal changes that alter thyroid function. These changes mean that laboratory tests of thyroid function must be interpreted with caution during pregnancy. Thyroid function tests change during pregnancy due to the influence of two main hormones: human chorionic gonadotropin (hCG), the hormone that is measured in the pregnancy test and estrogen, the main female hormone. HCG can weakly turn on the thyroid and the high circulating hCG levels in the first trimester may result in a slightly low TSH. When this occurs, the TSH will be slightly decreased in the first trimester and then return to normal throughout the duration of pregnancy . Estrogen increases the amount of thyroid hormone binding proteins in the serum which increases the total thyroid hormone levels in the blood since more than 99% of the thyroid hormones in the blood are bound to these proteins.

The thyroid gland can increase in size during pregnancy (enlarged thyroid = goiter). However, pregnancy-associated goiters occur much more frequently in iodine-deficient areas of the world. This is usually only a 10-15% increase in size and is not typically apparent on physical examination by the physician.(71)

4-1- Hormones :

For the first 10-12 weeks of pregnancy, the baby is completely dependent on the mother for the production of thyroid hormone. By the end of the first trimester, the baby's thyroid begins to produce thyroid hormone on its own. The baby, however, remains dependent on the mother for ingestion of adequate amounts of iodine, which is essential to make the thyroid hormones.

Zargham N, 2005 finding that serum total T4 level in pregnant during three trimester were significantly ($p<0.001$) higher compared to non-pregnant women. (71)

Corinne R, 2004 finding that Plasma concentrations of Total T4and total T3 concentrations increase sharply in early pregnancy and plateau early in the second trimester at concentrations 30-100% greater than non pregnancy values (72) .The etiology of this increase in total circulating thyroid hormones involves, primarily, increased concentrations of plasma thyroxin binding globulin (TBG) . Another proposed mechanism for this increase in total thyroid hormone concentrations is production of type III deiodinase from the placenta .This enzyme, which converts T4 to reverse T3, and T3 to diiodotyrosine (T2),has extremely high activity during fetal life (73,74).

Glinoer D,1997 said that Increased demand for T4 and T3 has been suggested to increased production of these hormones with, ultimately, increased concentrations in the circulation during pregnancy ,and this increased risk of miscarriage, spontaneous abortion, fetal death may occur as a result of chromosomal abnormalities. (75).

Total T3,total T4 at the third trimester significantly higher (P<0.001) than control group .This study agrees with study done by Mocan *et al* 2001 .They mentioned that serum TSH levels declines in pregnant women compared with non pregnant women.

Uncontrolled maternal hyperthyroidism during pregnancy has been associated with fetal tachycardia (fast heart rate), small for gestational age babies, prematurity, stillbirths and possibly congenital malformations. .(76)

4.2- Biochemical's profile :

Tables(3-1),(3-3),(3-4),(3-5) shows that glucose has lower mean at three trimesters. And significant(P<0.05) at the first trimester with control group while at the second and third it was found not significantly. This Results agrees with Boden *et al* ,(77) who mentioned that glucose at the first trimester was decrease during pregnancy, insulin needs often drop during the first three months, because the growing baby uses some of mother glucose.

Spellacy *et al* .(78) said that pregnant also may have morning sickness and eat than usual. This can decrease blood glucose level and insulin requirements and this study agree with our study.

Jovanovich *et al*.(79)said that glucose at the second and third trimester increase camper with first trimester then insulin needs will increase .Because the placenta begins to make hormones that interfere with the work of insulin. Therefore needs more insulin to overcome the effects of the placenta. At the third trimester baby will grow quickly. The extra food along with the effect of placental hormones will increase insulin needs.

Tables (3-1),(3-3),(3-4),(3-5) shows that serum uric acid at the third trimester higher level than first and second trimester all compared with control group , it is at three trimesters were not significantly our study agree with Simmonds *et al* [80] said that raised levels of uric acid in pregnant in the third trimester and not significantly during three trimesters, is regarded as evidence of the extent of fetal uric acid production and clearance by the maternal circulation.

BROWN *et al* ,said that the blood uric acid level falls in early and in moddle pregnancy and rises to normal values in late pregnancy. We have also demonstrated in the present study a normal daily urinary urate excretion in early pregnancy and an enhanced renal loss of urate in middle and late pregnancy.(81)

D.Robertson Smith. found that increased renal excretion of urate in pregnancy it is unlikely that this is the sole factor responsible for the changes which have been observed in the serum uric acid concentration for the following two reasons. First, in early pregnancy the serum uric acid is low while there is no change in the renal excretion of urate Secondly, when the serum uric acid concentration is normal in late pregnancy ,a high renal loss of urate obtains. It is unlikely that diminished uric acid production is responsible for the hypouricaemia .(82A). Our findings may have the following explanation : initially early hypervolaemia of pregnancy, such as has been shown to exist by Hytten and Paintin, as early as 12 weeks, may produce a dilution hypouricaemia; the finding of a normal 24-hour urinary urate excretion in the presence of a low serum uric acid concentration indicates that the renal clearance of uric acid must be

raised at this time. Increased uric acid clearance will then be responsible for the low uric acid levels found .In middle pregnancy the serum uric acid level is even lower but the 24-hour urinary urate excretion is above normal .These facts suggest a further increase in uric acid clearance by the kidneys in this stage of pregnancy .It is also probable that in middle pregnancy significant transfer of uric acid from the growing fetus to the maternal blood stream is beginning to occur . In late pregnancy the normal serum uric acid levels possibly indicate that the high renal uric acid clearance is not sufficient to clear from the mothers blood the increasing amounts of uric acid produced by the fetus. .(82B)

Tables (3-1),(3-3),(3-4),(3-5) shows that total protein during three trimester there are no change at the first trimester and decrease level at second and third trimester this compared with control group, and significantly ($P<0.05$) at third trimester only .

Our study agree with Hancock. said that the plasma levels of total protein during pregnancy decreases from 10^{th} to the 28^{th} week of pregnancy; a further decrease was evident at the 37^{th} week. Because low albumin concentration total protein decreases during pregnancy.(83)

In liver disease or acute infection concentrations of total protein below the reference rang usually reflect low albumin concentration . Concentrations above the reference rang are found in paraproteinaemia, Hodgkin's lymphoma or leukemia. These findings support the concept that embryonic and placental development are closely related in the first trimester of human pregnancy, placental biological functions persisting only for a limited period of time after embryonic demise(84)

4.3-lipid profile during three trimesters:

Tables (3-1),(3-3),(3-4),(3-5) shows that increases in serum lipids are common during the second half of pregnancy. Many pregnant women had serum concentrations of total cholesterol which in non-pregnant women would be associated with an increased risk of coronary heart disease(CHD) . We have also shown that the concentrations of LDL and HDL cholesterol are increased in pregnancy. The increases in total and LDL cholesterol and triglyceride are similar to those reported by others(85,86) but the increase in HDL cholesterol has not been widely observed. Our results for HDL cholesterol agree with those of Desoye et al(87)and with those of Piechota and Staslewski.(86) Other workers have reported little change in HDL cholesterol during pregnancy.(88,89) High plasma concentrations of endogenous oestrogens are well known to lower LDL cholesterol, the mechanism whereby pregnancy induces hyperlipidaemia has not been fully elucidated. The complementary and opposing actions of the individual pregnancy hormones and their changing concentrations during pregnancy would be expected to lead to pronounced alterations in lipoprotein metabolism as gestation progresses. Desoye *et al* found a positive correlation between changes in the lipid and lipoprotein concentrations and the changes in the concentrations of the pregnancy hormones oestradiol, progesterone, and human placental lactogen (HPL) during gestation .(87) Triglyceride was also positively correlated with increasing concentrations of insulin in the second trimester of gestation .Oestrogen seems to be responsible for most of the alterations in lipoprotein metabolism during pregnancy, but its actions are complemented and opposed by the other pregnancy hormones, and in late

pregnancy by increasing insulin resistance. Oestrogens can increase the concentration of plasma triglyceride by stimulating hepatic production of the triglyceride-rich very low density lipoproteins(VLDL)(90) and by inhibition of hepatic and a dipose tissue lipoprotein lipases.(91) Oestrogens increase the concentration of HDL cholesterol by directly stimulating. Production of LDL is stimulated by oestrogen(92) but the net effect is to reduce plasma concentrations as the clearance of LDL is enhanced owing to increased activity of the hepatic LDL receptors.(93) The role of progesterone in pregnancy associated hyperlipidaemia is questionable. Progestogens have been shown to oppose the actions of oestrogens on lipoprotein metabolism, leading to increased concentrations of LDL cholesterol and decreased concentrations of HDL cholesterol.(94) Some authors have suggested that the oestrogen: progesterone ratio, which is low in early and in very late pregnancy is important in the balance of alterations in lipoprotein metabolism through-out pregnancy.(87,92) The actions of exogenous progestogens on lipoprotein metabolism seem ,however, to depend on the androgenicity of the preparations used(94,95) Natural progesterone is not androgenic and has not been shown to affect lipoprotein concentrations(95) and so many not be involved in the alterations in lipoprotein metabolism during pregnancy. In late pregnancy rising concentrations of prolactin inhibit a dipose tissue lipoprotein lipase activity(96) resulting in a rise in the concentration of plasma triglyceride .HPL has lipolytic activity which may increase substrate supply of free fatty acids for the increased maternal hepatic VLDL production . The hypertriglyceridaemia in late pregnancy may be further enhanced by hyperinsulinaemia due to insulin resistance which is known to be

associated with higher concentrations of triglyceride .(97) Hypoalbuminaemia is a further possible contributory cause of hyperlipidaemia in pregnancy, but our subjects did not have sufficiently low serum albumin concentrations to result in secondary hyperlipidaemia. It has been suggested that the alterations in lipid metabolism during gestation may be important in the control of delivery to and uptake of nutrients by the fetus ,particularly during rapid fetal weight gain in the second half of gestation .During early pregnancy adipose stores are enlarged and used by the mother in late gestation so as to spare glucose for the fetus. Maternal hyperlipidaemia may therefore have a beneficial influence on fetal development. Whether the maternal hyperlipidaemia has any pathological importance is not known.(98) A recent study has shown that lipid concentrations after delivery are higher, and that the total cholesterol to HDL cholesterol ratio is higher in women who have had five or more pregnancies compared with those who have had only one. Pregnancy may also precipitate sever chylomicronaemia in women with the rare familial lipoprotein lipase deficiency or with other causes of hypertriglyceridaemia. Such women may be previously undiagnosed and may present with acute pancreatitis during pregnancy.

Our study also agree with Kostner *et al*.and Tan *et al.*)(99) said that The hyperlipidemia of pregnancy is accompanied by an increase in the plasma cholesterol esterification rate for the first half of gestation. During the second half of gestation there is little change in the plasma cholesterol esterification rate, in the presence of a continuing increase in plasma lipid levels.

Farjad Shaker , reported that in humans pregnancy there was a statistically significant increase in the concentrations of serum cholesterol and triglyceride in advanced age . NOGUCHI reported that in humans the concentrations of LDL and VLDL increased and the concentration of HDL decreased with increasing age

Castelli , Said that Cholesterol cant dissolve in the blood .It has to be transported to and form the cells by carriers called lipoproteins. Low-density lipoprotein, or LDL is Known as "bad" cholesterol. High-density lipoprotein, or HDL, is Known as "good" cholesterol. These two types of lipids, along with triglyceride, make up total cholesterol count, which can be determined through a blood test. When too much LDL(bad)cholesterol circulates in the blood, it can slowly build up in the inner walls of the arteries that feed the heart and brain. Together with other substances, it can form plaque, a thick, hard deposit that can narrow the arteries and make them less flexible. This condition is known as atherosclerosis. If a clot forms and blocks a narrowed artery, heart attack or stroke can result .

About one-fourth to one-third of blood cholesterol is carried by high-density lipoprotein (HDL).HDL cholesterol is Known as "good" cholesterol, because high levels of HDL serum to protect against heart attack

The cholesterol levels show an overall 53% increase during pregnancy, two thirds of which occurred within the second trimester. .. The cholesterol decrease 15% within 12 to 24 hours of delivery, but at day 5 of postpartum some rebound occurs and al 6 to 7 week of postpartum the women had still a significant raised cholesterol levels. Many diseases can

occur during pregnancy and require diagnosis in the context of expected physiological changes..(100)

Triglyceride at the third trimester higher mean and highly significant (P<0.001) compared with control group while at first and second lower mean and not significantly ,this study agree with Jung *et al.*, said that Serum triglyceride concentration showed very significant (P<0.001) increase in the third trimester of normal pregnancy than in the non pregnant women. The principle modulator of this hypertriglyceridemia is oestrogen as pregnancy is associated with hyperoestrogenaemia. Oestrogen induces hepatic biosynthesis of endogenous triglycerides, which is carried by VLDL (101). This process may be modulated by hyperinsulinism found in pregnancy . in our study which corroborated with the findings of many workers.

Increased triglyceride , found in pregnancy induced hypertension, There is a general agreement that thyroid hormones influence the mechanism of triglyceride but the results are controversial to study of Diamant *et al*.and Thorklid, ; O'Brien *et al*.,and Mukhopadhyay *et al*., .(102,103,104,105)

Berti *et al* ;said that Most of the articles related to plasma lipids in pregnancy agree that both increase during pregnancy, as well as lipoprotein.(106)

In our own study, the mean value of HDL-C was higher at three trimester of normal pregnancy over the non pregnant women, but statistically the alteration was not significant (P>0.05). this study was reported by many workers (107) . Oestrogen is responsible for induction of triglyceride and HDL .

Dalmacio *et al.,* said that It is important to determine the HDL-C because elevation has a protective effect against atherosclerosis while its reduction aggravates the atherosclerosis processes.(108)

Tan and Kung *et al* , said that Low levels of HDL(less than 40 mg/dl) also increase the risk of heart disease. Medical experts think that HDL tend to carry cholesterol away from the arteries and back to the liver, where its passed from the body. Some experts believe that HDL removes excess cholesterol from arterial plaque , thus slowing its buildup.(109)

In present study, serum VLDL-C level at third trimesters have higher mean and significantly ($P<0.05$) during pregnancy in comparison to non-pregnant women, which is perhaps due to hypertriglyceridemia leading to enhanced entry of VLDL that carries endogenous triglyceride into circulation.(110)

LDL-C level increases and highly significant ($P<0.001$) at second and third trimester and this study also reported by many workers (111,112) This finding is in agreement with that of previous reports (Thorkild and Orbie, Sucic *et al*., de-Castero *et al*., Iguma *et al*.,and Jung *et al*.) which demonstrate that LDL-C clearance is reduced in hypothyroidism. The mechanism responsible for that had been attributed to the decrease in the number of LDL-C receptors. On the other hand, thyroid hormones enhance LDL-C receptors expression (99,100,101,102)

We have also calculated the ratios CHO-C:HDL-C; LDL-C:HDL in present study there was no significant at three trimester in normal pregnant as compared to non-pregnant women.(113)

The researchers found no difference in the rate of birth defects but women with very low cholesterol were more likely to give birth to babies

with a smaller head size. (113) High levels of LDL cholesterol or bad cholesterol in the blood can however increase the risk of heart diseases .

4-4 **Hemoglubin during threetrimister**

Tables (3-3),(3-4),(3-5) shows that hemoglobin was significantly ($p<0.05$)at the first and second trimester while it is not significant at third trimester .and this study agree with Kelley *et al* , said that there is maternal anemia during pregnancy was associated with preterm birth.

And also observed the strongest increased risk of preterm birth among women with more severe anemia when hemoglobin decreases depending on week of gestation due to Dilutional anaemia of pregnancy . At third trimester there is no change in level and not significantly and this study was agreed with our study (114)

Conclusions:

In this study it found that

1- During three trimester of pregnancy ,both serum levels of T3&T4 were higher than serum TSH level this may produce hyperthyroidism if not controlled symptoms.

2- Pregnancy at third trimester have increase level in all biochemical tests except glucose and total protein they have lower level.

3-uric acid at third trimester increased and this can be due to blood volume.

4-Total protein decreases during second and third trimester and this can be due to albumin decreases.

5- more correlation between lipid and lipoprotein and glucose, uric acid, total protein, bilirubin , hemoglubin associated with thyroid hormone especially at second trimester during pregnancy than first and third trimester this is due to physiological changes during pregnancy .

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List of abbreviations

ATP	Adenosine triphosphate.
α	Alpha.
Ab	Antibody.
Ag	Antigen.
ß	Beta.
CVD	Cardio – vascular disease.
r	Correlation coefficient.
dL	Deciliter.
DIT	Diiodotyrosine.
GI	Glycemic Index
HDL-C	High density lipoprotein – cholesterol.
hCG	Human chorionic gonadotropin
IDD	Iodine deficiency disorders.
K cal	Kilo calories.
LPL	Lipoprotein lipase.
LDL-C	Low density lipoprotein-cholesterol.
Ml	Milliter.
Min	Minute.
MW	Molecular weight.
MIT	Monoiodotyrosine.
P	Probability level.
r-T_3	Reverse triiodothyronine.
RBC	Red blood cells
SD	Standard deviation.
Tg	Thyroglobulin.
TSH	Thyroid-stimulating hormone.
TRH	Thyrotropin releasing hormone.
TBG	Thyroxin binding globulin.
T_4	Thyroxin.
TC	Total Cholesterol.
T_3	Triiodothyronine.
TG	Tryglyceride.
5`D-I	Type I 5 – deiodinase.
5`D-II	Type II 5 – deiodinase.
VLDL-C	very Low density lipoprotein-cholesterol

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Recommendations:

1- Determination of trace elements(e.g. Copper, Zinc, Irone, manganese, calcium , sodium)concentration in pregnancy during three trimester and compared with non pregnant women.

2- Studying some of enzymes (e.g. alkaline phosphatase),(Gamma Glutamyl Transferase) ,(Alanine Aminotransferase),
(Aspartate Aminotransferase) and Superoxide dismutase(SOD) in serum of women before, during and after pregnancy.

3 - More studies have to be measured to correlate the function of thyroid gland with there enzymes.

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Chapter One

Introduction

Chapter Two

Materials & Methods

Chapter Three

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Chapter six
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Chapter Five

Conclutions & Recommendations

الخلاصة

المقدمة :

حالة الحمل هي متعلقة بالتغيرات الفيزيولوجيه والتي تساعد على بقاء الجنين والتحضير للولادة .تقسم مده الحمل إلى ثلاث مراحل كل مرحله تشمل ثلاث أشهر من خلالها توصف التغيرات التي تحصل إثناء فتره الحمل وهي مهمة في معرفه التغيرات الطبيعية التي تحصل إثناء الحمل لتساعد في تشخيص المشاكل الطبية للحامل مثل زيادة في نشاط الغدة الدرقية أو سكر الحمل أو فقر الدم والتغيرات الكيميائية الحياتية عند الحامل تعكس بصوره واضحة وممتازة عن حاله غير الحمل عند النساء ،عدة هرمونات تفرز إثناء الحمل قسم منها يختفي بعد الولادة والأخر يتغير في مستواه حتى بعد الولادة ،هرمونات الغدة الدرقية وظائفها إثناء الحمل تتغير خاصة خلال الأشهر الثلاث الأولى حيث يعتمد الجنين بصوره كلية على هرمونات الدرقية للام وهي تؤثر على عمليات ايض الكاربوهيدرات والبروتينات والدهون ، فتقل حساسية الانسجه للاستجابة للأنسولين مما يودي إلى انخفاض مستوى الكلوكوز عند إلام ناتج سكر الحمل وكذالك فقر الذي يأتي نتيجة الزيادة الحاصلة في حجم الدم إلى حوالي ٤٠% وهذا أيضا يسبب نقص في مستوى الالبومين أيضا التغيرات الهرمونية والتغيرات الكيمياويه تظهر الاختلاف الواضح بين حاله الحمل وغير الحمل .

طريقه العمل:

في هذه الدراسة تم دراسة مصل ٥٠ امرأة حامل خلال الثلاث فترات من أشهر الحمل تراوحت أعمارهن بين (١٦-٥٠)سنه ومصل ٢٠ امرأة متزوجة غير حامل تراوحت أعمارهن بين (١٧-٥٤)سنه اعتبرت كمجموعه سيطرة. تم اخذ النماذج من مستشفى العلوية للولادة ومختبر الصحة المركزي وخلال الفترة من ٢٠٠٧/٧/١ إلى ٢٠٠٨/٤/٨ . حيث تم قياس هرمونات الغدة الدرقية وهي(T3,T4,TSH) بواسطة استخدام الفلوره المناعية باستخدام جهاز miniVidus في مصل النساء الحوامل وخلال الثلاث مراحل من فتره الحمل ، وكذالك تم قياس مستوى الكلوكوز وحامض اليوريك والبروتين الكلي والكوليستيرول والترايكليسرايد والبروتينات الناقلة للدهون وهي البروتين الناقل عالي الكثافة HDL والبروتين الناقل قليل الكثافة LDL والبروتين الناقل ذو الكثافة الواطئة جدا VLDLونسبه الكوليستيرول/البروتين عالي الكثافة وكذالك نسبه البروتين منخفض الكثافة /البروتين عالي الكثافة وأيضا الهيموكلوبين تم قياس النماذج بواسطة جهاز , spectrophotometer, في مصل كل من النساء الحوامل وغير الحوامل .

الخلاصة

الهدف من الدراسة:

١- قياس مستوى هرمونات الدرقية (T3,T4,TSH) خلال الأشهر الثلاث الأولى والأشهر الثلاث الثانية والأشهر الثلاث الثالثة عند النساء الحوامل وكذالك عند النساء غير الحوامل.

٢- قياس المتغيرات الكيميائية الحياتية وتشمل الكلوكوز وحامض اليوريك والبروتين الكلي والكوليستيرول والترايكليسرايد والبروتينات الناقلة للدهون وهي البروتين الناقل عالي الكثافة HDL والبروتين الناقل قليل الكثافة LDL والبروتين الناقل ذو الكثافة الواطئة جدا VLDLونسبه الكوليستيرول/البروتين عالي الكثافة وكذالك نسبه البروتين منخفض الكثافة /البروتين عالي الكثافة وأيضا الهيموكلوبين عند النساء الحوامل وغير الحوامل .

٣- معرفه العلاقة بين هرمونات الغدة الدرقية والمتغيرات الكيميائية الحياتية عند الحوامل وخلال الثلاث فترات من الحمل وكذالك القياس عند النساء الغير الحوامل.

النتائج:

ا- في الأشهر الثلاث الأولى كان هناك ارتفاع في مستوى هرموني الدرقية T3,T4 ونقصان في مستوى هرمون TSH. خلال الثلاث فترات من الحمل ويرجع هذا الى اختلاف في تركيز البروتينات ألحامله (Thyroxin Binding Globulin and Prealbumin -TBG) جميع المتغيرات كانت ذات مستوى منخفض عدا مستوى إل HDL كان عالي أما مستوى البروتين الكلي لم يعاني تغير خلال هذه الفترة .أيضا وجد T4 كانت له احتماليه عاليه جدا ($P<0.001$) بينما T3,TSH لم تكن لهما احتماليه . كذالك الكلوكوز و الهيموكلوبين كانت لهم احتماليه اقل من ($P<0.05$) والباقي لم تظهر احتماليه.

أيضا يعتبر الكلوكوز هو مصدر الطاقة للجنين والذي ينتقل عبر المشيمة عن طريق الانتشار السهل لذالك يزداد إفراز الأنسولين خلال الحمل نتيجة نقصان في مستوى الكلوكوزوهذا يؤدي إلى استخدام الجنين كميه اكبر من الأحماض الدهنيه،خلال فتره الحمل زيادة معدل الفلتره يرتفع معدل التصفية (GFR) إلى حوالي ٥٠% ونقص في ألقدره على طرح الماء يؤدي إلى الاستقاء في الكاحل والساق وهذا يؤدي إلى زيادة في مستوى حامض اليوريك خلال الأشهر الاخيره من الحمل ،يزداد حجم الدم إلى حوالي ٤٠-٥٠% وتبدأ هذه الزيادة في الثلث الأول من الحمل وتزيد بسرعة في الثلث الثاني ثم تستقر في الأسبوع الثلاثين من الحمل ،هذه الزيادة ضرورية لإمداد كميه اضافيه من الدم للرحم ،والاحتياجات الايضيه للجنين وزيادة نشاط الأعضاء الأخرى وخاصة الكلى وهذه الزيادة في حجم الدم ينتج عنها انخفاض في تركيز الهيموكلوبين ،الألبومين،وبروتينات البلازما الأخرى.

الخلاصة

وجد هنالك علاقة موجبه قويه بين هرمون TSH مع ,cholesterol, uric acid, VLDL , glucose. أيضا وجدت علاقة سالبه قويه بين T4 مع glucose .

هنالك علاقة موجبه قويه بين الكلوكوز وحامض اليوريك في نفس الوقت علاقتهما مع الكوليستيرول وعلاقة الثلاثة مع البروتين الدهني واطئ الكثافة. في حين الترايكيسرايد له علاقة مع حامض اليوريك والبروتين الدهني واطئ الكثافة جدا.

الهيموكلوبين له علاقة موجبه قويه مع البروتين عالي الكثافة .

ب- في الأشهر الثلاث الثانية كانت هنالك ارتفاع مستوى هرموني الدرقيةT3,T4 ونقصان في مستوى هرمون TSH . الكلوكوز ارتفع عن مستوى الشهر الأول لكن مازال منخفض عن مجموعه السيطرة أيضا حامض اليوريك والبروتين الكلي والترايكليسرايد والبروتين الدهني واطئ الكثافة جدا والهيموكلوبين كانت ذا مستوى منخفض في حين الكوليستيرول والبروتين عالي والواطئ الكثافة كانت عاليه المستوى . أيضا وجد T3,T4 لهم احتماليه عاليه جدا ($P<0.001$) في حين TSH له احتماليه اقل من ($P<0.05$) ، أيضا الكوليستيرول والبروتين واطئ الكثافة والهيموكلوبين أظهرت احتماليه اقل من ($P<0.05$)

وجد هنالك علاقة موجبه قويه بين هرمون TSH مع uric acid , glucose

وأيضا بينT4 و T3. وعلاقة بين T3 والتراي كليسرايد وكذالك مع البروتين الدهني الواطئ الكثافة جدا وجد أيضا علاقة سالبه قويه بينTSH والتراي كليسرايد .

أيضا هنالك علاقة موجبه قويه بين الترايكيسرايد والكوليستيرول . البروتين واطئ الكثافة له علاقة مع الكوليستيرول . البروتين واطئ الكثافة جدا مع الكوليستيرول والتراي كليسرايد . الهيموكلوبين له علاقة مع الكلوكوز . أيضا هنالك علاقة سالبه قويه بين الترايكليسرايد والكلوكوز.

ج- في الأشهر الثلاث الثالثة كانت هنالك ارتفاع مستوى هرموني الدرقيةT3,T4 ونقصان في مستوى هرمون TSH . مستوى الكلوكوز والبروتين الكلي منخفض في حين باقي المتغيرات كانت مرتفعه المستوى مقارنه بمجموعه السيطرة .

أيضا وجدT4 له احتماليه عاليه جدا ($P<0.001$) في حين T3 له احتماليه اقل من ($P<0.05$) ،في حين الTSH لم يظهر احتماليه.

أيضا الكوليستيرول والبروتين واطئ الكثافة والترايكليسرايد لهم احتماليه عاليه جدا($P<0.001$) في حين البروتين الكلي والبروتين الدهني الواطئ الكثافة جدا أظهرت

الخلاصة

احتماليه اقل من ($P<0.05$) . وجد هنالك علاقة موجبه قويه بين هرمون TSH مع glucose.

وعلاقه بين T3 والكلوكوز وحامض اليوريك. الكوليستيرول مع البروتين الكلي.الترايكليسرايد مع الكلوكوز. وكذالك البروتين الدهني الواطئ الكثافه جدا مع الكلوكوز والترايكليسرايد. وكذالك البروتين واطئ الكثافه له علاقه مع الكوليستيرول والترايكليسرايد.

د- اظهرت النتائج بالنسبه الى النساء الغير حوامل بان هنالك علاقه موجبه قويه بين هرمون ال TSH والبروتين عالي الكثافه وكذالك مع هرمون T3.

ايضا هرمون T3 لـه علاقه موجبه قويه مع البروتين واطئ الكثافه وعلاقه سالبه قويه مع الترايكليسرايد والبروتين واطئ الكثافه جدا. ايضا هنالك علاقه موجبه قويه بين البروتين الكلي مع حامض اليوريك. الترايكليسرايد مع حامض اليوريك والكوليستيرول. البروتين عالي الكثافه له علاقه سالبه قويه مع حامض اليوريك. البروتين واطئ الكثافه جدا علاقه موجبه مع حامض اليوريك والكوليستيرولووالترايكليسرايد. البروتين واطئ الكثافه علاقه موجبه مع الكوليستيرول وعلاقه سالبه مع البروتين عالي الكثافه. الهيموكلوبين مع الكلوكوز علاقه موجبه قويه.

دراسة
بعض التغيرات الكيميائية الحياتية وهرمونات الغده الدرقية عند ا لنساء الحوامل

رسالة مقدمة إلى

كلية العلوم جامعة النهرين

كاستكمال جزئي لمتطلبات نيل درجة ماجستير
علوم في الكيمياء الحياتية

من قبل

سهاد عبد العزيز إبراهيم

بكالوريوس علوم كيمياء - جامعة بابل

رجب١٤٢٩هـ ٦ تموز٢٠٠٨م

جمهورية العراق
وزارة التعليم العالي والبحث العلمي
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