STUDY ON PREVALENCE OF HYPOTHYROIDISM IN WOMEN WITH PREECLAMPSIA

A Dissertation submitted to
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in partial fulfillment of the regulations for the award of the degree of

M.S (OBSTETRICS & GYNAECOLOGY) – BRANCH – II

GOVERNMENT STANLEY MEDICAL COLLEGE

CHENNAI – 600 001

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CERTIFICATE

This is to certify thatthe dissertation entitled "**STUDY ON PREVALENCE OF HYPOTHYROIDISM IN WOMEN WITH PREECLAMPSIA"** is a bonafide work done by **Dr. UMADEVI. N,** at R.S.R.M. Lyingin Hospital, Stanley Medical College, Chennai. This dissertation is submitted to Tamil Nadu Dr.M.G.R Medical University in partial fulfilment of university rules and regulations for the award of M.S. Degree in Obstetrics and Gynaecology.

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DECLARATION

I Dr. UMADEVI. N, solemnly declare that this dissertation titled "**STUDY ON PREVALENCE OF HYPOTHYROIDISM IN WOMEN WITH PREECLAMPSIA"** is a bonafied work done by me at R.S.R.M. Lying in Hospital, Stanley Medical College, Chennai during January 2014 to December 2014 under the guidance and supervision of Prof. Dr.V.Kalaivani M.D.D.G.O., Professor and H.O.D of the Department of Obstetrics and Gynaecology.

This dissertation is submitted to the Tamil Nadu Dr.M.G.R Medical University, Chennai in partial fulfilment of university rules and regulations for the award of M.S. Degree (Branch – II) in obstetrics and Gynaecology.

Place: Chennai

Date: **Dr.N .UMADEVI**

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INTRODUCTION
A variety of endocrine disorders complicate pregnancy, thyroid dysfunction is one of the common conditions among them. Thyroid gland function is intimately related to reproductive performance in women. Ovulation failure and infertility can occur in severe hypothyroidism. Though ovulation and conception can occur in mild hypothyroidism ,there is an increased chance of abortion, preterm delivery, stillbirth.
Pregnancy is associated with major changes in hypothalamic pituitary thyroid axis ,iodine metabolism and the immune function. To meet the increased metabolic demands in pregnancy changes in thyroid physiology occur which is reflected by altered thyroid function tests. So thyroid disorders commonly occur in pregnancy.
The body undergoes extensive changes during pregnancy. There is renal loss of iodine ,relative iodine deficiency and there is also transfer of iodine to the fetus from mother. To compensate for this there is increased uptake of iodine from blood by the thyroid gland. This results in
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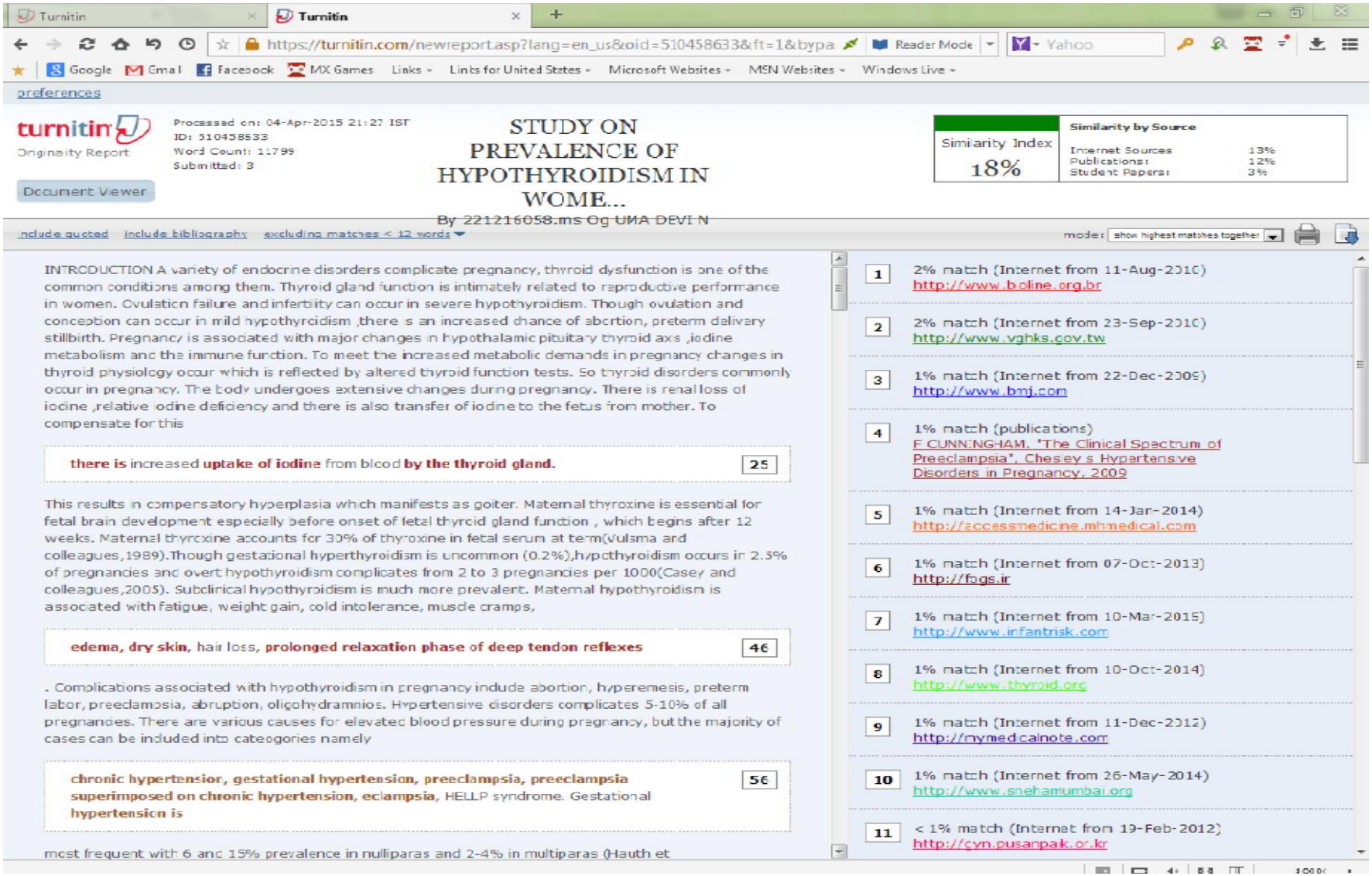
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there is increased uptake of iodine from blood by the thyroid gland.
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This results in compensatory hyperplasia which manifests as goiter. Maternal thyroxine is essential for fetal brain development especially before onset of fetal thyroid gland function , which begins after 12 weeks. Maternal thyroxine accounts for 30% of thyroxine in fetal serum at term(Vulsma and colleagues,1989).Though gestational hyperthyroidism is uncommon (0.2%),hypothyroidism occurs in 2.5% of pregnancies and overt hypothyroidism complicates from 2 to 3 pregnancies per 1000(Casey and colleagues,2005). Subclinical hypothyroidism is much more prevalent. Maternal hypothyroidism is associated with fatigue, weight gain, cold intolerance, muscle cramps,
edema, dry skin, hair loss, prolonged relaxation phase of deep tendon reflexes
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. Complications associated with hypothyroidism in pregnancy include abortion, hyperemesis, preterm labor, preeclampsia, abruption, oligohydramnios. Hypertensive disorders complicates 5-10% of all pregnancies. There are various causes for elevated blood pressure during pregnancy, but the majority of cases can be included into categories namely
chronic hypertension, gestational hypertension, preeclampsia, preeclampsia superimposed on chronic hypertension, eclampsia, HELLP syndrome. Gestational hypertension is
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most frequent with 6 and 15% prevalence in nulliparas and 2-4% in multiparas (Hauth et
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INTRODUCTION

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Pregnancy is associated with major changes in hypothalamic pituitary thyroid axis ,iodine metabolism and the immune function. To meet the increased metabolic demands in pregnancy changes in thyroid physiology occur which is reflected by altered thyroid function tests. So thyroid disorders commonly occur in pregnancy.

The body undergoes extensive changes during pregnancy. There is renal loss of iodine ,relative iodine deficiency and there is also transfer of iodine to the fetus from mother. To compensate for this there is increased uptake of iodine from blood by the thyroid gland. This results in compensatory hyperplasia which manifests as goiter. Maternal thyroxine is essential for fetal brain development especially before onset of fetal thyroid gland function , which begins after 12 weeks.

Maternal thyroxine accounts for 30% of thyroxine in fetal serum at term(Vulsma and colleagues,1989).Though gestational hyperthyroidism is uncommon (0.2%),hypothyroidism occurs in 2.5% of pregnancies and overt hypothyroidism complicates from 2 to 3 pregnancies per 1000(Casey and colleagues,2005). Subclinical hypothyroidism is much more prevalent.

Maternal hypothyroidism is associated with fatigue, weight gain, cold intolerance, muscle cramps, edema, dry skin, hair loss, prolonged relaxation phase of deep tendon reflexes . Complications associated with hypothyroidism in pregnancy include abortion, hyperemesis, preterm labor, preeclampsia, abruption, oligohydramnios.

Hypertensive disorders complicates 5-10% of all pregnancies. There are various causes for elevated blood pressure during pregnancy, but the majority of cases can be included into cateogories namely chronic hypertension, gestational hypertension, preeclampsia, preeclampsia superimposed on chronic hypertension, eclampsia., HELLP syndrome. Gestational hypertension is most frequent with 6 and 15% prevalence in nulliparas and 2-4% in multiparas (Hauth et al.,2000;Buchbinder et al 2002). Preeclampsia is identified in 3.9% of all pregnancies.

Although pregnancy is associated with mild hyperthyroxinemia, preeclamptic women have a high incidence of hypothyroidism that might

correlate with the severity of preeclampsia. On the other side preeclampsia occurs in 16.7% of subclinical cases and43.7% of overt hypothyroidism. The changes in thyroid gland in pregnancy is accounted by high circulating estrogen.

There are controversies in the mechanism of hypothyroidism in preeclampsia which are accounted by decreased plasma protein concentration and high levels of endothelin and soluble fms like tyrosine kinase.

The aim of my present study is to study the prevalence of hypothyroidism in women with preeclampsia. By looking for the association we can make screening universal instead of target screening in antenatal patients and we can start early thyroxine supplementation in affected individuals.

AIM OF THE STUDY

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- To study the prevalence of hypothyroidism in women with preeclampsia and to compare them with age matched controls.
- To look for the correlation between hypothyroidism and severity of pre eclampsia.
- To analyse the association between onset of preeclampsia and hypothyroidism

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The thyroid gland is an endocrine gland in the anterior region of the neck . Its main function is to secrete triiodothyronine(T3) and thyroxine (T4).The production of thyroid hormones by the gland is under the control of thyrotropin (TSH),which in turn is controlled by TRH. The thyroid gland undergoes tremendous changes during pregnancy.

EFFECTS OF PREGNANCY ON THE THYROID GLAND:

Pregnancy has a goitrogenic effect on the thyroid gland by 40 to 100% to compensate for the maternal and fetal demands (Small ridge and associates 2005). There is an increase in Thyroxine binding globulin(TBG) in early pregnancy to reach twice the non pregnant levels by 16-20 weeks ,due to increased estrogen and reduced clearance by sialylation of TBG. The increase in TBG inturn produces an increase in total T3 and total T4, whereas the free hormone levels remain unchanged. To maintain a constant free T4 level in the plasma, the production of T4 increases by 30-50%[1].

PREGNANCY AS A GOITROGENIC FACTOR:

In pregnancy due to the increased vascularity and glandular hyperplasia of the thyroid gland ,there is a moderate enlargement of the gland . The mean thyroid volume increases from 12 ml in the first trimester to 15 ml at delivery[2].Serumthyrotropin concentration is inversely related to thyroid volume. Such enlargement is not pathological. Any goiter in pregnancy should be investigated since there is no significant thyromegaly in normal pregnancy.

IODINE AND PREGNANCY:

The iodine excretion in the urine increases during pregnancy due to increased glomerular filtration rate and plasma clearance. On the other hand the requirement of iodine increases due to increase in serum thyroxine levels and for the transfer of thyroxine to the fetus in the first trimester and iodine later on ,once the fetus starts synthesizing thyroid hormones. Women with sufficient iodine reserve can cope up with this, with little impact on the thyroid function. Those with deficient reserve will have a fall in thyroid hormone levels ,resulting in increase in TSH with enlargement of thyroid and goiter formation in the mother and fetus.

Serum thyroglobulin is a good marker that correlates well with the thyroidal stimulation due to iodine deficiency. The goiter formed in pregnancy regress only partly after pregnancy ,especially in breast feeding women. To prevent this goiter formation and to attain a normal intrathyroidal stores of 10-20mg ,the women should have atleast 150µg /day iodine.

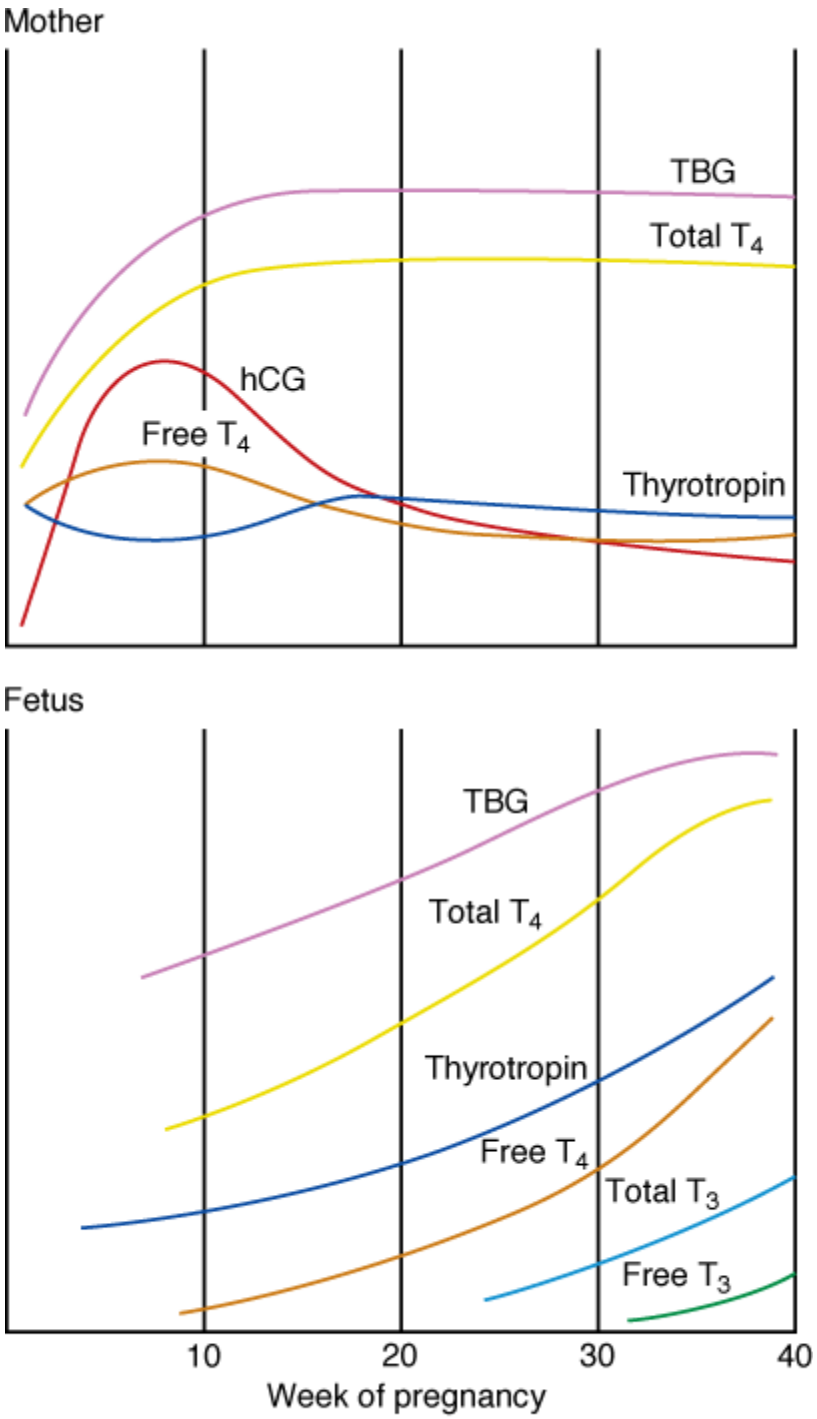
The American Thyroid Association (ATA) has recommended supplementation of pregnant and lactating women with 150µg/day of iodine added to prenatal vitamins. WHO recommends that during pregnancy and lactation, this daily intake should be increased to 250µg/day. As per the Institute of Medicine, daily iodine intake varies from 220µg/day in pregnancy and 290 µg/day in lactation.

The maximum upper limit for iodine is 600-1100 µg/day for adults and pregnant women >19 yrs. Tend and co-workers in 2006 [2.] have suggested that excessive iodine intake >300 mg/day may lead to subclinical hypothyroidism and autoimmune thyroiditis.

RELATIVE CHANGES IN MATERNAL THYROID FUNCTION:

The thyroxine binding globulin increase and attains its peak at 20 weeks and stabilise at twice the baseline values thorough out the rest of the pregnancy. Between 6 and 9 weeks there is a sharp increase of TotalT4 and attains plateau at 18 weeks. There is a gradual peak of Free serum T4 along with hCG and they come back to normal. Upto 18 weeks there is a pronounced rise in total T3 and then it plateaus.

Glinoer and associates in 1990 [2] found that the secretion of T4 and T3 is not similar in all pregnant woman .Around one third of women experience relative hypothyroxinemia ,with preferential T3 secretion and higher albeit normal, serum thyrotropin levels.



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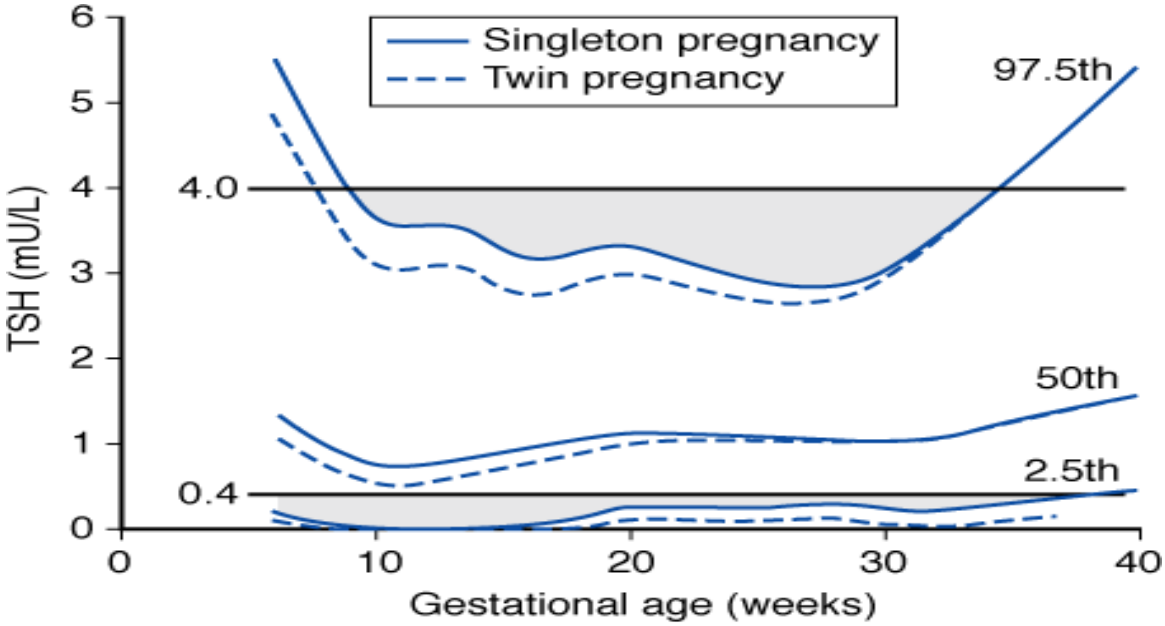
RELATION BETWEEN hCG AND TSH:

The changes in serum TSH and human chorionic gonadotropin (hCG) vary with gestational age. The alpha subunit of the two glycoproteins are identical, although the beta subunits are similar there is a difference in their amino acid sequence. Due to this structural similarity hCG has thyroid stimulating property. An example, 1 microU of hCG was equivalent to 0.0013 microU of TSH. hCG reaches peak at 8 to 10 weeks producing transient increase in free T4.

In more than 80% of pregnant women the thyrotropin levels decrease whereas the levels still fall into the normal non pregnant range. Serum TSH concentrations are transiently low or undetectable in 10 to 20 percent of normal women [3,4,5]. The normal fall in TSH in pregnant women may lead to a false diagnosis of subclinical hyperthyroidism. This fall in TSH also leads to the failure to identify early hypothyroidism. To obviate this misdiagnosis gestational age specific TSH values were developed by Dashe and co workers[2] in 2005

Relationship between TSH and gestational age in weeks.



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These changes in thyroid regulations are not reflected by maternal thyroid status. As pregnancy advances the decline in hCG secretion leads to decline in serum free T4 and T3 concentrations and there is a rise of serum TSH slightly to or within the normal range..Though the BMR increases by as much as 25 %, if the fetal surface area is taken into consideration the observed BMI rates are similar to non pregnant women.

PHYSIOLOGICAL CHANGES IN THE THYROIDGLAND OF THE FETUS-:

By the end of the first trimester ,the pituitary thyroid system is functional. As early as 11 weeks the TSH,T4,TBG can be detected in fetal serum. The placenta concentrates iodine on the fetal side and by 12 weeks there is more strong iodine concentration by the fetal thyroid than maternal thyroid. But there is a little hormone production till 18-20 weeks. The fetal thyroid hormone plays an important role in the development of all organs especially the brain.

Congenital hyperthyroidism caused by maternal thyroid stimulating antibodies is manifested by perceptual motor difficulties, hyperactivity and reduced growth. The placenta prevents further passage of maternal thyroid hormones to the fetus by rapidly deiodinating maternal T4 and T3 to form reverse T3. Initially it was thought that thyroid hormones are not essential

for fetal growth, but Vulsma and colleagues have shown that small amount of maternal T4 prevent antenatal cretinism to manifest in fetus with thyroid agenesis[1].

The fetus with congenital hypothyroidism does not develop the stigma of cretinism until after birth. Since administration of thyroid hormones will prevent this ,all newborns are tested for rise in TSH. At term serum TSH concentration is higher, whereas there is low serum T4 concentrations. The serum T3 concentration is only one half of the concentration in the mother. The serum TSH concentrations increase to 50-80 mU/L after birth and within 2 days it falls to 10-15 mU/L. The T3 and T4 concentrations in serum rise to levels are that are slightly higher than those in normal adults.

PLACENTAL TRANSFER :

The placental transfer of thyroid hormones is controversial. There is passage of TSH-receptor antibodies via the placenta and leads to hypo or hyperthyroidism. There is little transfer of TSH .Exogenously administered TRH can stimulate fetal TSH secretion due to the transplacental passage of thyrotropin releasing hormone.

NORMAL VALUES OF TSH:

The trimester specific reference range for TSH and serum free T4 is recommended by the American Thyroid Association(ATA) for management of pregnancy and postpartum period due to the pregnancy associated changes in thyroid .The commercial labs should provide their method specific reference range.

If the lab does not provide their standard range the following range is used.

For TSH:

I trimester =0.1-2.5mU/L

II trimester=0.2-3.0mU/L

III trimester=0.3-3.0 mU/L

The value of 2.5mU/L was chosen since it is not only close to 97.5th percentile but also higher values are associated with higher fetal morbidity.

Free T3=1.7 to 4.2 pg/ml

Free T4=0.7 to 1.8ng/dl

There are some studies showing a decrease in free T4 during pregnancy, others report no change or even an increase[7,8]. During pregnancy it may be unreliable to measure direct free T4. Measurement of free T4 using liquid chromatography/tandem mass spectrometry in the dailysate or ultrafiltrate of serum samples appears to be the most reliable, and when this method is used, free T4 concentrations were shown to decrease gradually with advancing gestational age, particularly between the first and second trimester [9,10].

Trimester-specific and method specific ranges of serum free T4 should be used, if available[11].When there is a discrepancy between TSH and free T4,total T4 should be measured. The total T3 and T4 are 1.5 fold higher than non pregnant women. Thus pregnancy specific range should be used.

THYROID DISORDERS:

1.Hypothyroidism- → Subclinical hypothyroidism
1.Hypothyroidism- → Overt hypothyroidism

2.Hyperthyroidism

HYPOTHYROIDISM:

The most common cause of hypothyroidism in iodine sufficient areas is chronic autoimmune (Hashimotos thyroiditis),whereas iodine deficiency itself acts as a cause for hypothyroidism. Other causes are prior iodine ablation, diseases of pituitary and hypothalamus.

Overt hypothyroidism - TSH above trimester specific range in association with low free T4 concentration. It complicates 2 to 3 pregnancies per 1000.

Subclinical hypothyroidism – TSH above trimester specific range with normal free T4 concentration. The incidence of subclinical hypothyroidism is about 5 % in women between 18 -45 years.

Diez and Iglesias in 2004, did a prospective study on 93 pregnant women with subclinical hypothyroidism reported that the TSH returned back to normal levels in one third of women In the remaining two thirds, those with TSH levels between 10 -15mU/L developed overt hypothyroidism at a rate of 19 per 100 patient years. Those women with TSH <10mU/L developed overt hypothyroidism at a rate of 2 per 100 patient years[2].

SCREENING FOR HYPOTHYROIDISM IN PREGNANCY :

Routine screening of all pregnant women for thyroid disorders is still an area of controversy. The American Thyroid Association (ATA), the Endocrine society, the American College of Obstetrics and Gynaecology (ACOG) favour targeted screening instead of universal screening. The ATA recommends screening in pregnant women when they are,

1. Symptomatic / from an area with moderate to severe iodine deficiency.
2. positive personal/family history of thyroid disorders.
3. history of miscarriages /preterm delivery/ head and neck irradiation
4. BMI > 40
5. thyroid peroxidase antibody (TPOAb) positive / type 1 diabetes
6. age > 30 years / history of infertility

In women who fulfill the screening criteria ,first serum TSH is measured, if it is above the trimester specific range then free T4 is measured to identify the degree of hypothyroidism. In those women with subclinical hypothyroidism ,thyroid peroxidase antibody should be measured. This helps to make decision in women with boderline TSH and in predicting postpartum thyroid dysfunction.

We miss upto one third of the cases with hypothyroidism with this approach. Universal screening will be a cost effective approach if one thinks that the treatment will improve the IQ of the newborn.

In one study 0f 4562 women ,there were no difference in adverse outcome among women divided to universal screening and case finding group. Low risk women in universal screening group who were diagnosed as hypothyroid and treated have few adverse outcomes than among low risk women in case finding group who were not treated. This however failed to reach statistical significance due to large number of adverse events occurring even in women with euthyroid status.

OVERT HYPOTHYROIDISM:

It is difficult to diagnose hypothyroidism clinically during pregnancy, due to the overlapping of features between hypothyroidism and normal pregnancy itself. It is characterized by fatigue, muscle cramps, weight gain, cold intolerance ,constipation .Other features include dry skin, edema, hair loss, prolonged relaxation phase of deep tendon reflexes.

PREGNANCY OUTCOME WITH OVERT HYPOTHYROIDISM:

MATERNAL:

Overt hypothyroidism constitutes 0.3-0.5% of screened women. The factors contributing to this low % are some hypothyroid women are anovulatory[12], and there are increased risk of spontaneous first trimester abortions[13,14,15].

These include preeclampsia, gestational hypertension, placental abruption, anaemia, heart failure, non reassuring fetal heart tracing, preterm delivery, low birth weight babies, increased caesarean section rate[16], postpartum haemorrhage.

FETAL:

Increased perinatal morbidity and mortality, neuropsychological impairment, low IQ, sepsis, respiratory distress syndrome, cardiomyopathy, neonatal hyperthyroidism.

SUBCLINICAL HYPOTHYROIDISM:

It is more common when compared to overt hypothyroidism occuring in 2.0 -2.5% of screened women in united states.

1)Pregnancy outcome:

Some studies state that women with subclinical hypothyroid are also at an increased risk of preeclampsia, placental abruption ,preterm labor, pregnancy loss[17-21]. In a study of 17,298 women registered for prenatal care ,there is two fold higher risk of preterm birth in 2.3% subclinical hypothyroid women ,defined as TSH above 97.5th percentile for gestational age.

In another study comparing pregnancy outcome in women with negative antithyroid peroxidase antibody , there is increased pregnancy loss rate in women with serum TSH between 2.5 -5.0mU/L than those with TSH < 2.5mU/L in the first trimester (6.1 Vs 3.6%) [22].

There are limited studies[23] available stating that there is worse pregnancy outcome in women undergoing IVF with preconception TSH > 2.5 mU/L, whereas the FASTER trial didn't find significant adverse outcomes in subclinical hypothyroidism.

2) Cognitive impairment:

There are some observational studies indicating the association of subclinical hypothyroidism with impaired cognitive development in children[24,25].In one report,15 % of women of women with TSH > 98th percentile have mean IQ score was 85% or less ,whereas only 5% of women have TSH within normal limits[26].

Further additional randomized trials are needed to determine whether screening and treatment of subclinical hypothyroidism earlier in pregnancy (prior to 13 weeks) has any benefit on neurocognitive outcomes.An analysis of maternal thyroid function at delivery of preterm babies < 34 weeks ,and later neurocognitive development at 5.5 years reported a significant decrements in cognitive performance for each mU/L increment in TSH[27].There are some studies stating subclinical hypothyroidism in older patients causes hypertension, heart failure and atherosclerotic vascular disease.

ISOLATED HYPOTHYROXINEMIA:

It is defined as lower maternal free T4 concentration in the 5th or 10th percentile of the reference range in association with normal TSH. The effects of isolated hypothyroxinemia on pregnancy is unclear. According to one study there is no adverse outcomes associated with this condition[28.]

According to the FASTER trial, women with isolated hypothyroxinemia have increased odds ratio of 1.62 for preterm labor, 1.97 for macrosomia, 1.70 for gestational diabetes .Children whose mother had isolated low FT4 have decreased mean intelligence and behavior scale compared with women who had normal TSH values[24,29,30,31]. The children IQ was not different between those who born to low T4 mothers who did or didn't receive treatment before 20 weeks of gestation[32].

TREATMENT OF HYPOTHYROID ;

The treatment recommendations as per American Thyroid Association (ATA) and the Endocrine society are as follows;

Candidates eligible for treatment:

- pregnant women with overt hypothyroidism which is newly diagnosed.

- pregnant women with subclinical hypothyroidism with positive TPO-Ab titres (as per ATA).

In women with subclinical hypothyroidism without positive TPO-Ab, the ATA found insufficient evidence to support for or against for treating them. Therefore the benefits of treating women without TPO-Ab is to be established by further studies.

The goal of treatment is to maintain trimester-specific reference range for mother's serum TSH (0.1 to 2.5 mU/L, 0.2 to 3 mU/L, and 0.3 to 3 mU/L for the first, second, and third trimesters).

The treatment of choice for hypothyroidism is synthetic thyroxine (T4).Tthose with moderate and severe hypothyroidism should be started with almost full replacement doses(1.6mcg/kg/day),while patients with TSH< 10mU/L can be started on approximately 1 mcg/kg/day.

Ideally T4 should be taken on empty stomach in morning, one hour before breakfast. If the TSH is above the trimester specific range ,the dose should be increased by12-25mcg/day. After starting treatment TSH should be repeated every 4 weeks once, in the first half of pregnancy and less frequently in later half of pregnancy.

It is suggested that same formulation of T4 to be used. Generics is avoided because of the potential for frequent interchange of preparations by the pharmacy. Follow-up biochemical monitoring (TSH) should be performed six weeks later, if the preparation is changed .This is done to consider the need for retitration of the dose .

PREEXISTING HYPOTHYROIDISM:

50-85 % of Women with pre existing hypothyroidism need dose increments during pregnancy [33-35]. During pregnancy there may be increase in dose requirements to as much as 50% and the increase occurs usually as early as 5th week of gestation .This is accomplished by increasing the dose from once daily dosing to nine doses per week.

In a retrospective study ,60% required levothyroxine dose increase (34% during the first trimester)[35].In a prospective study of 20 patients, 47% required increase in thyroxine dose[34].

Unlike normal women, those with pre-existing hypothyroidism or subclinical hypothyroidism are unable to cope with the increased demand occurring in normal pregnancy especially difficult in those who had prior ablation, surgical treatment for graves disease.

There is an increase in T4 requirement in pregnancy because of increase in T4 pool, increase in TBG, weight gain, transfer of iodine to fetus, placental deiodinase activity and reduced gastrointestinal absorption due to iron in prenatal vitamins[33].

The goal of preconception TSH is <2.5 mU/L[36].According to one study, only 17% of women with preconception TSH <1.2mU/L required dose increments whereas 50% of women required dose increments with preconception TSH between 1.2 and 2.4 mU/L [37].

The TSH should be measured immediately after conception and then once in every 4 weeks or 4 weeks after change in T4 dose and atleast one in each trimester. Adjustment of the dose should be done every 4 weeks to achieve normal TSH .The T4 dose can be reduced to prepregnancy levels, but this should be confirmed by measuring TSH 4-6 weeks whether the reduction was appropriate[33,38].

CONGENITAL HYPOTHYROIDISM:

It is caused by agenesis or dysgenesis of thyroid gland, congenital dyshormonogenesis, iodine deficiency in endemic areas.

THYROID PEROXIDASE ANTIBODIES:

Women with TPO-Ab are at increased risk of subclinical hypothyroidism in first trimester and thyroiditis in the postpartum period. The decision to treat euthyroid women with TPO-Ab with thyroxine or to monitor for the development of hypothyroidism is controversial.

PREGNANCY HYPERTENSION:

Among all the pregnancies hypertensive disorders complicate 5 to 10 percent .The hypertensive disorders ,haemorrhage and infection form a deadly triad and they greatly contribute to maternal morbidity and mortality rates . Preeclampsia syndrome alone or superimposed on chronic hypertension is most dangerous.

Preeclampsia occurs in 3.9% of all pregnancies. In developed countries ,preeclampsia constitutes greater percentage than other three leading causes abortion —8 percent, hemorrhage —13 percent, and sepsis—2 percent. 16% of maternal mortality rates were due to hypertensive disorders(2)

Classification of hypertensive disorders:

According to NHBPEP—National High Blood Pressure Education Program (2000), hypertensive disorders in pregnancy are classified into:

1. Gestational hypertension—previously known as pregnancy-induced hypertension. If there is no development of preeclampsia syndrome and by 12 weeks of postpartum if hypertension resolves , it is renamed as transient hypertension

2. Preeclampsia and eclampsia syndrome

3. Preeclampsia superimposed on chronic hypertension

4. Chronic hypertension

Diagnosis of Hypertensive Disorders Complicating Pregnancy

Gestational Hypertension:

- Systolic BP ≥ 140 or diastolic BP ≥ 90 mm Hg for the first time during pregnancy
- No proteinuria
- BP returns to normal before 12 weeks postpartum
- Final diagnosis made only in postpartum

May have other signs or symptoms of preeclampsia, for example, epigastric discomfort or thrombocytopenia

Preeclampsia:

Minimum criteria:

- BP ≥ 140/90 mm Hg after 20 weeks gestation
- Proteinuria ≥ 300 mg/24 hours or ≥ 1+ dipstick

Increased certainty of preeclampsia:

- BP ≥ 160/110 mm Hg
- Proteinuria 2.0 g/24 hours or ≥ 2+ dipstick

- Serum creatinine >1.2 mg/dL unless known to be previously elevated
- Platelets < 100,000/μL
- Microangiopathic hemolysis—increased LDH
- Elevated serum transaminase levels—ALT or AST
- Persistent headache or other cerebral or visual disturbances.
- Persistent epigastric pain

Eclampsia:

- Seizures that cannot be attributed to other causes in a woman with preeclampsia

Superimposed Preeclampsia On Chronic Hypertension:

- New-onset proteinuria ≥ 300 mg/24 hours in hypertensive women but no proteinuria before 20 weeks gestation
- A sudden increase in proteinuria or blood pressure or platelet count < 100,000/ μL in women with hypertension and proteinuria before 20 weeks gestation

Chronic Hypertension:

- BP ≥ 140/90 mm Hg before pregnancy or diagnosed before 20 weeks' gestation, not attributable to gestational trophoblastic disease

or

Hypertension first diagnosed after 20 weeks' gestation and persistent after 12 weeks postpartum

(ALT = alanine aminotransferase; AST = aspartate amino transferase; BP = blood pressure; LDH = lactate dehydrogenase)

This classification helps in differentiating o preeclampsia and eclampsia from other hypertensive disorders, since the above two are potentially more ominous

DIAGNOSIS:

Hypertension is defined as systolic blood pressure >140 mmHg and diastolic blood pressure >90 mmHg on 2 occasions atleast 6 hours apart. Diastolic blood pressure is measured by Korotkoff phase V. Previously a rise of 30mmHg of systolic or 15mmHg of diastolic blood pressure from mid pregnancy values was used to diagnose hypertension, even when the absolute values were below 140/90mm Hg. Its not recommended now

since evidence shows that such women were not associated with poor pregnancy outcomes.

GESTATIONAL HYPERTENSION:

Gestational hypertension is diagnosed when blood pressure reaches for the first time after mid pregnancy to 140/90 mm Hg or greater after midpregnancy, but without *proteinuria*. Almost 50% of these women develop preeclampsia syndrome subsequently, which includes signs such as proteinuria and thrombocytopenia or symptoms such as epigastric pain or headache. If by 12 weeks postpartum, the blood pressure returns to normal and if there is no evidence of proteinuria, gestational hypertension is redefined as *transient hypertension*.

Proteinuria is the marker for endothelial damage.10% of eclamptic seizures develop before overt proteinuria is identified.

PREECLAMPSIA:

Preeclampsia is otherwise known as pregnancy specific syndrome which affects virtually all the organ systems of the body. Preeclampsia is more than gestational hypertension with proteinuria. Proteinuria is defined as 24 hour urinary protein of >300mg/24 hours or urine protein:creatinine ratio ≥0.3 or persistent 30mg/dl or 1+ protein in dipstick (Lindheimer and

colleagues, 2008a).The more severe the hypertension or proteinuria or the presence of indicators for severity ,the more is the certainity for development of preeclampsia.

INDICATORS FOR SEVERITY OF PREECLAMPSIA:

Headaches or *visual disturbances* such as *scotomata* can be premonitory symptoms of eclampsia. *Epigastric or right upper quadrant pain* frequently accompanies hepatocellular necrosis, ischemia, and edema that stretch Glisson's capsule. This characteristic pain is frequently accompanied by elevated serum hepatic transaminase levels.

The worsening preeclampsia may also be characterised by thrombocytopenia. The platelet activation, aggregation, microangiopathic hemolysis induced by severe vasospasm leads to thrombocytopenia. The more profound the signs of severity,the more likely delivery will be indicated. It may be misleading to classify as mild and severe because those classified as mild may progress to severe preeclampsia.

As per ACOG guidelines preeclampsia is divided into mild and severe based on the following indicators.

Indicators of Severity of Gestational Hypertensive Disorders		
Abnormality	**Nonsevere**	**Severe**
Diastolic blood pressure	<110 mm Hg	≥ 110mm Hg
Systolic blood pressure	<160 mm Hg	≥ 160 mm Hg
Proteinuria	≥ 2+	≥ 3+
Headache	Absent	Present
Visual disturbances	Absent	Present
Upper abdominal pain	Absent	Present
Oliguria	Absent	Present
Convulsion (eclampsia)	Absent	Present
Serum creatinine	Normal	Elevated
Thrombocytopenia (<100,000/μL)	Absent	Present
Serum transaminase elevation	Minimal	Marked
Fetal-growth restriction	Absent	Obvious
Pulmonary edema	Absent	Present

ECLAMPSIA:

The appearance of convulsions in a preeclamptic women with seizures not contributing to any other reasons. The seizures may be generalised and may appear before, during and after labour. As per parkland hospital status, delayed postpartum eclampsia continues to occur in less than 10% of pregnancies.(Alexander and co-workers, 2006; Brown and colleagues, 1987).

PREECLAMPSIA SUPERIMPOSED ON CHRONIC HYPERTENSION:

All chronic hypertension cases predispose to the development of eclampsia and preeclampsia. The diagnosis of chronic hypertension can be difficult if the patient is first seen >20 weeks ,since there is normally mid trimester fall in blood pressure, which again rise to original hypertensive level in the third trimester. If chronic hypertension is accompanied by proteinuria, then superimposed preeclampsia is diagnosed. Superimposed preeclampsia commonly may develop earlier in pregnancy than "pure" preeclampsia. Superimposed disease is often more severe and fetal-growth restriction most often accompanies it .

INCIDENCE AND RISK FACTORS OF PREECLAMPSIA:

Young and nulliparous women are more prone for preeclampsia whereas chronic hypertension usually occurs in older women. According to Sibai and Cunningham (2009), the incidence of preeclampsia in nulliparous populations ranges from 3 to 10 percent. The incidence of preeclampsia in multiparas is variable. Other risk factors include maternal age >35 years, obesity, multifetal gestation, African American ethnicity (Agudelo and Belizan, 2000; Sibai and colleagues, 1997; Walker, 2000).

There is a progressive relationship between preeclampsia and maternal weight. For women with a body mass index (BMI) < 20 kg/m^2 the risk is 4.3 percent whereas in those with a BMI > 35 kg/m^2 the risk is 13.3 %. In twins the incidence of preeclampsia is 13% Vs 5% in singletons. (Sibai and co-workers, 2000). The incidence is unrelated to zygosity (Maxwell and associates, 2001).

Though there are adverse pregnancy outcomes with smoking during pregnancy, there is decreased risk of hypertension in pregnant women who are smoking. (Bainbridge and associates, 2005; Zhang and colleagues, 1999). This is due to the fact that smoking upregulates placental adrenomedullin expression, which regulates volume homeostasis.

Placenta previa has also been reported to reduce the risk of hypertensive disorders in pregnancy (Ananth and colleagues, 1997). Those women who are normotensive during first pregnancy are less prone for preeclampsia in subsequent pregnancies.

INCIDENCE OF ECLAMPSIA:

Due to improved prenatal care the incidence of eclampsia decreases. According to the Royal College of Obstetricians and Gynaecologists (2006), in the United Kingdom, it approximates 1 in 2000.

ETIOPATHOGENESIS:

There is increased likelihood to develop gestational hypertensive disorders in the following women;

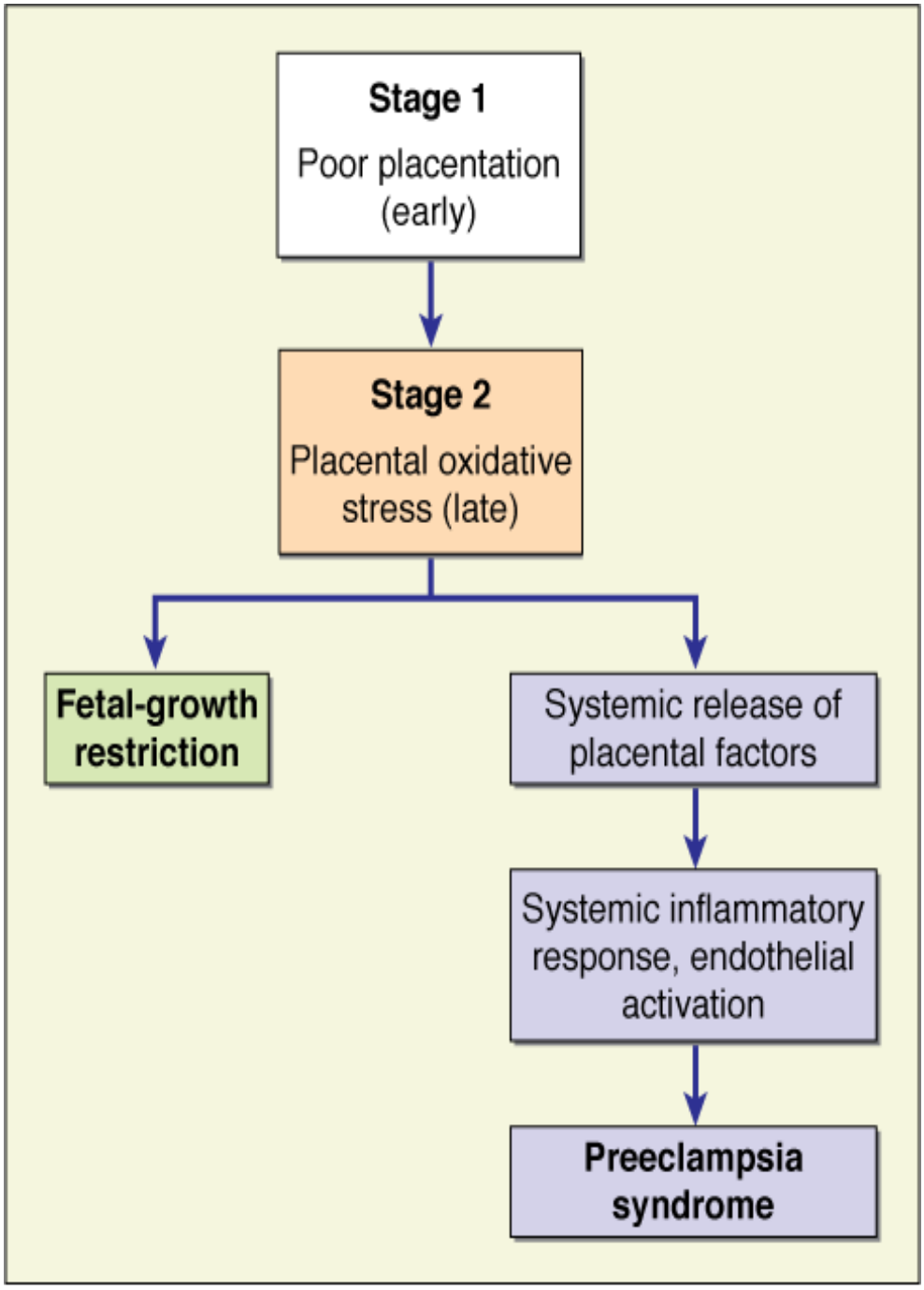
- First time exposure to chorionic villi
- Exposure to a more abundance of chorionic villi, as with twins or hydatidiform mole
- Have prior renal or cardiovascular disease
- When there is a genetic predisposition in pregnancy to hypertension.

A fetus is not a requisite for preeclampsia. The cascade of events that leads to the preeclampsia syndrome is characterized by a host of abnormalities that result in vascular endothelial damage and subsequent vasospasm, transudation of plasma, and further ischemic and thrombotic sequelae.

PREECLAMPSIA AS TWO STAGE DISORDER:

Following the observations that abnormal interfaces between maternal, paternal, and fetal tissues may cause preeclampsia, have led to hypotheses that the syndrome is a two-stage disorder.

According to Redman and colleagues (2009), stage 1 is caused by faulty endovascular trophoblastic remodeling that downstream causes the stage 2 clinical syndrome. Stage 2 is susceptible to maternal factors like diabetes, cardiac or renal disease, obesity and hereditary influences .Preeclampsia is clinically a spectrum of continuum disease.



Source: Cunningham FG, Leveno KJ, Bloom SL, Hauth JC, Rouse DJ, Spong CY: *Williams Obstetrics, 23rd Edition:* http://www.accessmedicine.com

ETIOLOGY:

1. Abnormal trophoblastic invasion of uterine vessels during placental implantation.

2. There is maladaptive immunological tolerance between maternal, paternal (placental), and fetal tissues

3. There is maternal maladaptation to normal cardiovascular or inflammatory changes of pregnancy

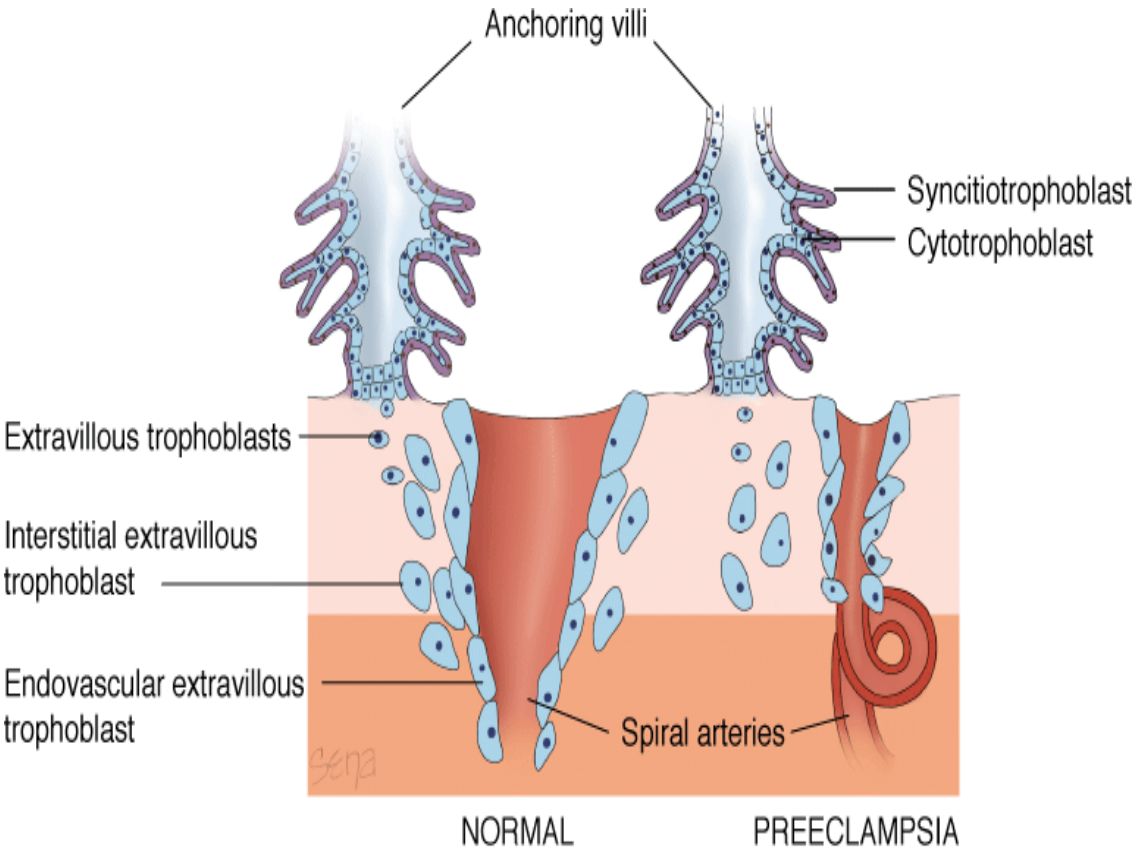
4. Genetic factors such as epigenetic influences and predisposing inherited genes.

ABNORMAL TROPHOBLAST INVASION:

In normal implantation, as shown in the figure below the uterine spiral arterioles are remodelled by the endovascular trophoblastic invasion . The vascular endothelial and muscular linings are replaced by these trophoblasts to increase the vessel diameter. There is only superficial invasion of the veins.

There may be *incomplete trophoblastic invasion* in preeclampsia. Because of the incomplete invasion only the decidual vessels, become lined with endovascular trophoblasts but not the myometrial vessels. Since there is no loss of the endothelial lining and musculoelastic tissue in the

deeper myometrial arterioles ,their mean external diameter is only 50% of the diameter in normal placental vasculature. (Fisher and colleagues, 2009). Madazli and associates (2000) showed that the severity of the hypertensive disorder is proportional to the magnitude of defective trophoblastic invasion of the spiral arteries.



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Thus the abnormally narrow spiral artery reduces placental blood flow, which inturn decreases placental perfusion leading to ischemia ,causing release of placental debris leading to systemic inflammatory response.

IMMUNOLOGICAL FACTORS

There is maternal tolerance to fetal and placental antigens. Loss of this tolerance is another explanation for preeclampsia.

Inherited Immunogenetic Factors in preeclampsia that May affect Genotype and Phenotype Expression
"Immunization" from a prior gestation
Inherited haplotypes for HLA-A, -B, -D, -Ia, -II
Inherited haplotypes for NK-cell receptors—also called killer-immunoglobulin-like receptors—KIR
Possibly shared susceptibility genes with diabetes and chronic hypertension

ENDOTHELIAL CELL ACTIVATION:

The endothelial injury is thought to be provoked by metabolic factors, anti angiogenic and other inflammatory mediators . The activated state of leucocytes in maternal circulation causes endothelial injury.(Faas, 2000; Gervasi, 2001)TNF-α, ILs may also leads to this oxidative stress. This oxidative stress produces reactive oxygen species and free radicals that inturn leads to self-propagating lipid peroxides formation (Manten and associates, 2005).

These free oxygen radicals damage the endothelial cells, impair nitric oxide production and interfere with the balance between the prostaglandins. Other manifestations of oxidative stress include lipid laden foamy macrophages as seen in atheroma, thrombocytopenia caused by microvascular coagulation and increased capillary permeability causing edema and proteinuria.

Many researchers have believed that antioxidants might prevent the occurrence of preeclampsia .But it‘s not proven so far.

NUTRITIONAL FACTORS:

John and co-workers (2002) showed that a diet high in fruits and vegetables with antioxidant activity is associated with decreased blood pressure. Zhang and associates (2002) reported there is double the incidence of preeclampsia in those women whose daily intake of calcium was less than 85mg. Villar and associates (2006) reported the supplementation of calcium in those women whose dietary intake of calcium was low, was associated with lower perinatal mortality rates, but no effect on the incidence of preeclampsia.

GENETIC FACTORS:

Preeclampsia is a multifactorial polygenic disorder. Ward and Lindheimer (2009) found that the following candidate genes are commonly investigated for causation.

Gene (Polymorphism)	Function Affected	Chromosome	Biological Association
MTHFR (C677T)	Methylene tetrahydrofolate reductase	1p36.3	Vascular diseases
F5 (Leiden)	Factor V Leiden	1q23	Thrombophilia—may coexist with other thrombophilic genes
AGT (M235T)	Angiotensinogen	1q42-q43	Blood pressure regulation, linked to essential hypertension
HLA (Various)	Human leukocyte antigens	6p21.3	Immunity
NOS3 (Glu 298 Asp)	Endothelial nitric oxide	7q36	Vascular endothelial function
F2 (G20210A)	Prothrombin (factor II)	11p11-q12	Coagulation—weakly associated, studied with other thrombophilic genes
ACE (I/D^{at}Intron 16)	Angiotensin converting enzyme	17q23	Blood pressure regulation

Because of the heterogenecity of preeclampsia, and the interaction of genetic and environmental factors for complex phenotypic expression ,it is doubtful whether any one of the candidate genes will be found responsible

Other genetic factors are

1. Multiple genotypes: maternal and paternal (fetal and placental)
2. Subgroups: associated disorders such as diabetes and characteristics such as parity
3. Genomic ethnicity: frequency of polymorphisms, founder effect, selection and genetic drift.
4. Gene-gene interaction: specific alleles or products of two or more genes affect one another and thus the phenotype
5. Epigenetic phenomena: variations in expression of a functional stable gene, for example, monozygotic twin differences
6. Gene-environmental interactions—these are infinite

PATHOGENSIS OF PREECLAMPSIA SYNDROME.

VASOSPASM:

Because of vasoconstriction there is increased resistance and subsequent hypertension .Due to endothelial damage there is interstitial leakage with subendothelial deposition of platelets and fibrinogen .With decreased blood flow there is ischemia of surrounding structures with necrosis ,haemorrhage and further end organ damage.

ENDOTHELIAL CELL ACTIVATION:

Intact endothelium has anticoagulant properties, and the vascular smooth muscle response to agonists is blunted by the endothelial cells by releasing nitric oxide. Damaged or activated endothelial cells may produce less nitric oxide and produce substances that favour coagulation and increase sensitivity to vasopressors. (Gant and co-workers, 1974).

INCREASED PRESSOR RESPONSE :

There is loss of refractoriness to renin angiotensin II which precedes the onset of preeclampsia.

PROSTAGLANDINS:

The decreased pressor response in normal pregnancy is mediated by increased prostacyclin .In preeclampsia there is decreased production of prostacyclin and increased thromboxane production with decreased prostacyclin: thromboxane ratio.

NITRIC OXIDE:

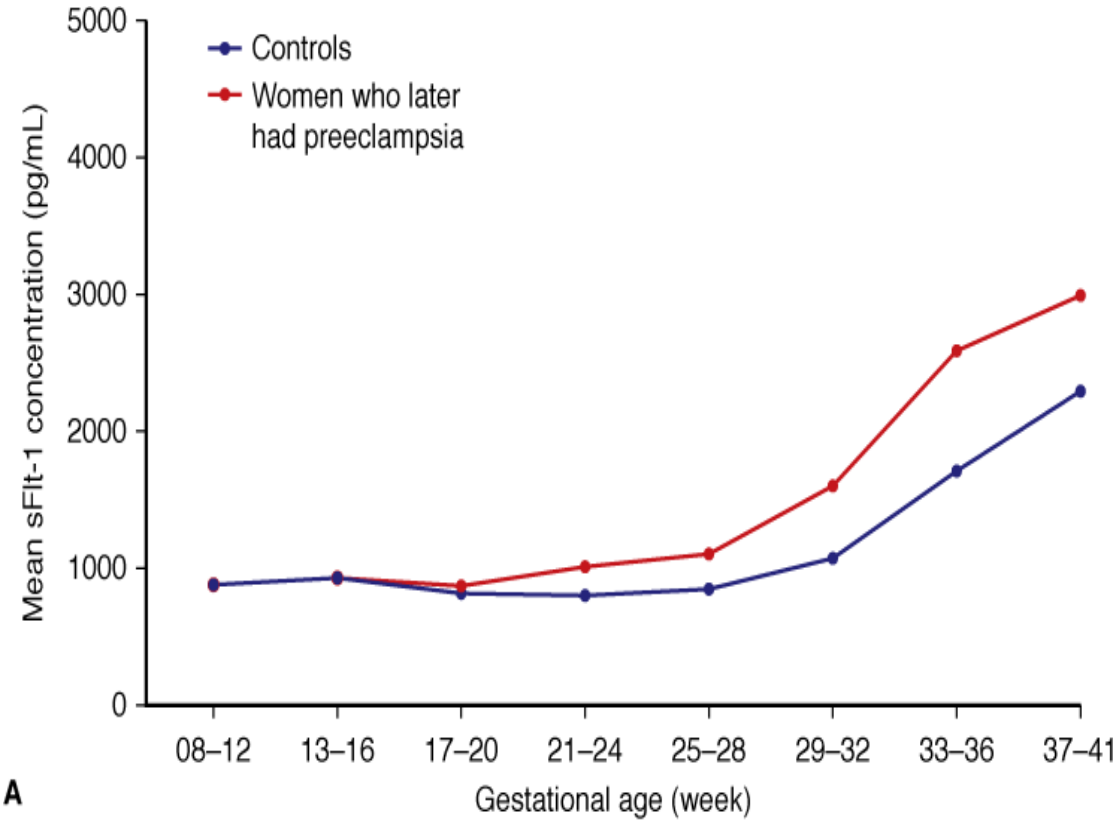
NO is a potent vasodilator produced by endothelial cells from L-ARGININE. Decreased nitric oxide production is associated with increased mean arterial pressure, decreased heart rate ,reverses the pregnancy induced refractoriness to vasopressor. The fetoplacental perfusion is characterised by the low pressure vasodilated state which is maintained by the nitric oxide The relation between nitric oxide and preeclampsia is unclear. It is associated with decreased nitric oxide production.

ENDOTHELINS:

Endothelins-1 (ET- 1) is the isoform produced by human endothelial cells. According to Taylor and Roberts (1999), the placenta is not the source of increased ET-1 concentrations, and they likely arise from systemic endothelial activation.

ANGIOGENIC ANTIANGIOGENIC PROTEINS:

The worsening hypoxia at the uteroplacental interface stimulates excessive amounts of antiangiogenic factors that leads to angiogenic imbalance. Trophoblastic tissue of women destined to develop preeclampsia overproduces at least two antiangiogenic peptides that enter into the maternal circulation (Karumanchi and colleagues, 2009) namely soluble fms like tyrosine kinase and soluble endoglins.



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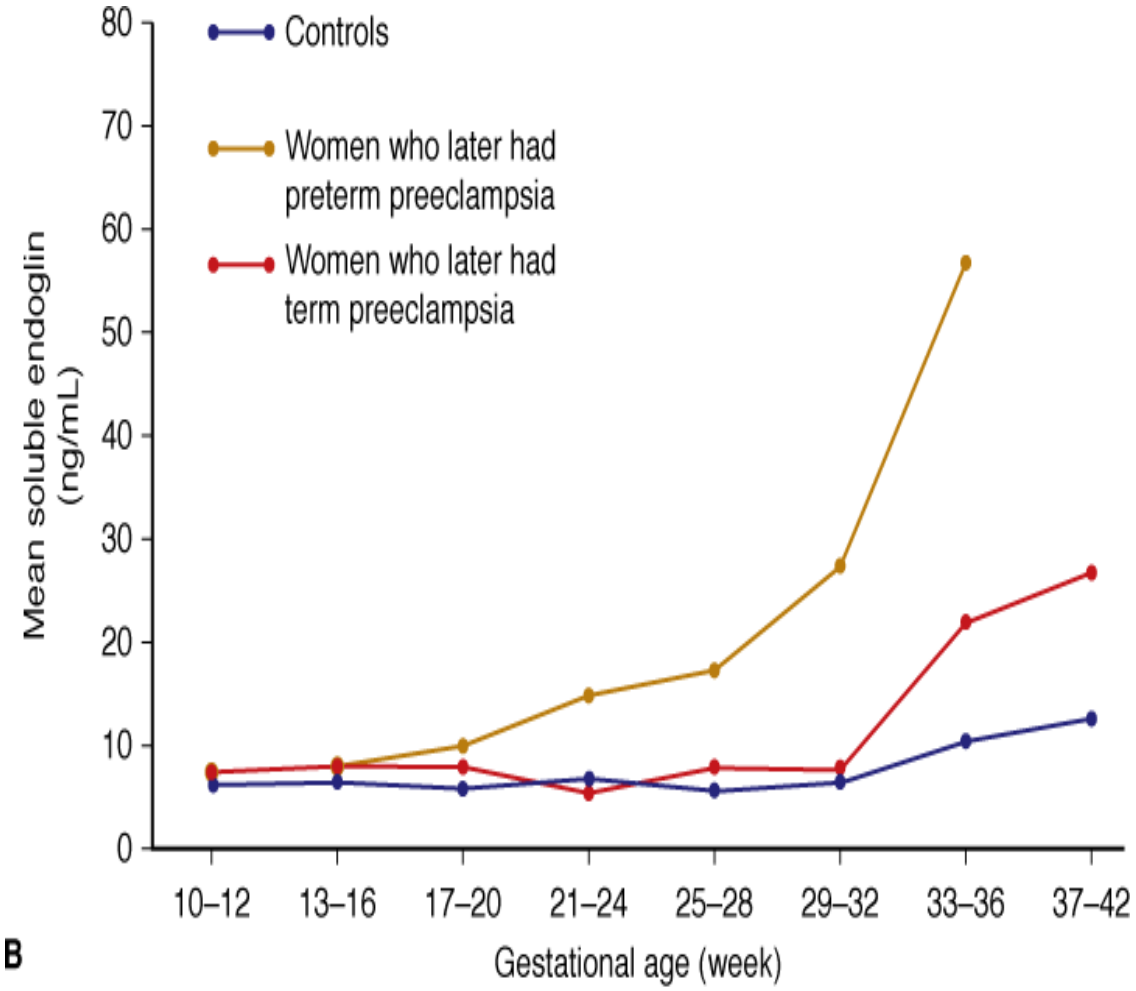

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PATHOPHYSIOLOGY OF PREECLAMPSIA:

The changes in preeclampsia evolve gradually beginning from early weeks of gestation to manifest in later half .They usually involve multiple organ systems.

CARDIOVASCULAR CHANGES:

The changes are due to increased cardiac afterload caused by hypertension, increased preload, endothelial activation leading to extravasation of intravascular fluid into extracellular space especially the lungs. Though there is a hyperdynamic cardiac state in all women, intravenous fluid infusions influence the filling pressures. In most women especially, aggressive hydration leads to hyperdynamic ventricular function . Importantly, this is also accompanied by elevated pulmonary capillary wedge pressures. In some of these women, inspite of normal ventricular function pulmonary edema may develop, because of an alveolar endothelial-epithelial leak that inturn is compounded by decreased oncotic pressure from a low serum albumin concentration (American College of Obstetricians and Gynecologists, 2002a).

The hyperdynamic ventricular function was largely a result of low wedge pressures and not a result of augmented myocardial contractility measured as left ventricular stroke work index. By comparison, women

given appreciably larger volumes of fluid commonly had filling pressures that exceeded normal, but their ventricular function remained hyperdynamic because of increased cardiac output. Thus aggressive fluid administration to women with severe preeclampsia causes normal left sided filling pressure to become substantively elevated and increase an already normal cardiac output to supranormal levels.

Blood volume:

Hemoconcentration is the hallmark of eclampsia.during last weeks of gestation the average blood volume is 5000ml compared to 3500ml in non pregnant women. This anticipated normal excess of 1500ml is lost in preeclampsia. This is due to generalised vasoconstriction that follows endothelial activation and leakage of plasma into interstitial space due to increased permeability. In preeclamptic women depending on its severity hemoconcentration is usually not marked . There is normal blood volume in gestational hypertension.

For women with severe hemoconcentration, it was taught that an sudden fall in hematocrit indicates resolution of preeclampsia. In this situation, hemodilution follows endothelial healing with return of interstitial fluid into the intravascular space. Though its partially correct, it is important to realise that a substantive cause of this fall in hematocrit is

usually the consequence of blood loss at delivery**.** It may also be partially the result of increased erythrocyte destruction. Vasospasm and endothelial leakage of plasma may persist for a variable amount of time after delivery as the endothelium undergoes repair. As this takes place, vasoconstriction reverses, and as the blood volume increases, the hematocrit usually falls. Thus, women with eclampsia:

- Are unduly sensitive to vigorous fluid therapy administered in an attempt to increase the contracted blood volume to normal pregnancy levels.
- Are sensitive to amounts of blood loss which is considered as normal for a normotensive women during delivery.

BLOOD AND COAGULATION ABNORMALITIES:

Platelet abnormalities:

Thrombocytopenia, platelet surface alterations(platelet bound and circulating antibodies). *Maternal thrombocytopenia in hypertensive women is not a fetal indication for caesarean delivery*. Kenny and associates (2009) reported other changes such as platelet activation with increased degranulation, thromboxane A_2 release, and decreased lifespan.

Hemolysis :

Severe preeclampsia is accompanied by hemolysis with elevated lactate dehydrogenase , with peripheral smear findings of schistocytosis, spherocytosis , reticulocytosis. These derangements result in part from microangiopathic hemolysis caused by endothelial disruption with platelet adherence and fibrin deposition. Sanchez-Ramos and colleagues (1994) described increased erythrocyte membrane fluidity with HELLP syndrome, and Cunningham and co-workers (1995) postulated that these changes were due to serum lipid alterations. Erythrocytic membrane changes, increased adhesiveness, and aggregation may also facilitate a hypercoagulable state (Gamzu and co-workers, 2001; Grisaru and associates, 1997).

HELLP Syndrome:

Weinstein (1982) named the combination of hemolysis, elevated liver transaminase levels, thrombocytopenia as HELLP syndrome .These components are used in the distinction between mild and severe preeclampsia.

COAGULATION:

Except for thrombocytopenia other coagulation aberrations consistent with intravascular coagulation are less pronounced in preeclampsia. Unless there is associated placental abruption, plasma fibrinogen levels do not differ remarkably from levels found in normal pregnancy, and fibrin degradation products are elevated only occasionally. Barron and colleagues (1999) stated that it is not necessary to do prothrombin time ,partial thromboplastin time, plasma fibrinogen level routinely in all pregnancy associated hypertensive disorders. Thrombophilias may be associated with early onset preeclampsia. Fibronectin, a glycoprotein associated with endothelial cell basement is elevated in preeclampsia.

FLUID AND ELECROLYTE IMBALANCE:

In preeclampsia women edema is more than normal. The endothelial injury leads to edema, proteinuria leading to decreased oncotic pressure which further causes a shift of fluid from intravascular to extravascular space. There is not much difference in electrolyte concentrations between preeclamptics and controls. This may not be the case if there has been vigorous diuretic therapy, sodium restriction, or administration of free water with sufficient oxytocin to produce

antidiuresis. After an eclamptic convulsion there is decrease in serum pH and serum bicarbonate concentration due to lactic acidosis and compensatory respiratory loss of carbondioxide.

KIDNEY:

During normal pregnancy there is increase in renal blood flow and glomerular filtration rate .With the onset of preeclampsia there is decrease in renal blood flow and glomerular filtration rate. Mildly diminished glomerular filtration may result from a reduced plasma volume. Most of the decrement is probably from increased renal afferent arteriolar resistance that may be elevated up to fivefold. Pathologic changes such as glomerular endotheliosis blocking the filtration barrier, diminished filtration causes serum creatinine values to rise.

Urine sodium concentration is raised in most of the preeclamptic women. Urine osmolality, urine:plasma creatinine ratio, and fractional excretion of sodium are also indicative that a prerenal mechanism is involved. Crystalloid infusion raises left ventricular filling pressure, and although oliguria temporarily improves, rapid infusions may cause clinically evident pulmonary edema. For these preeclamptic women with oliguria intensive intravenous hydration therapy is not indicated, *unless diminished urine output is caused by hemorrhage.*

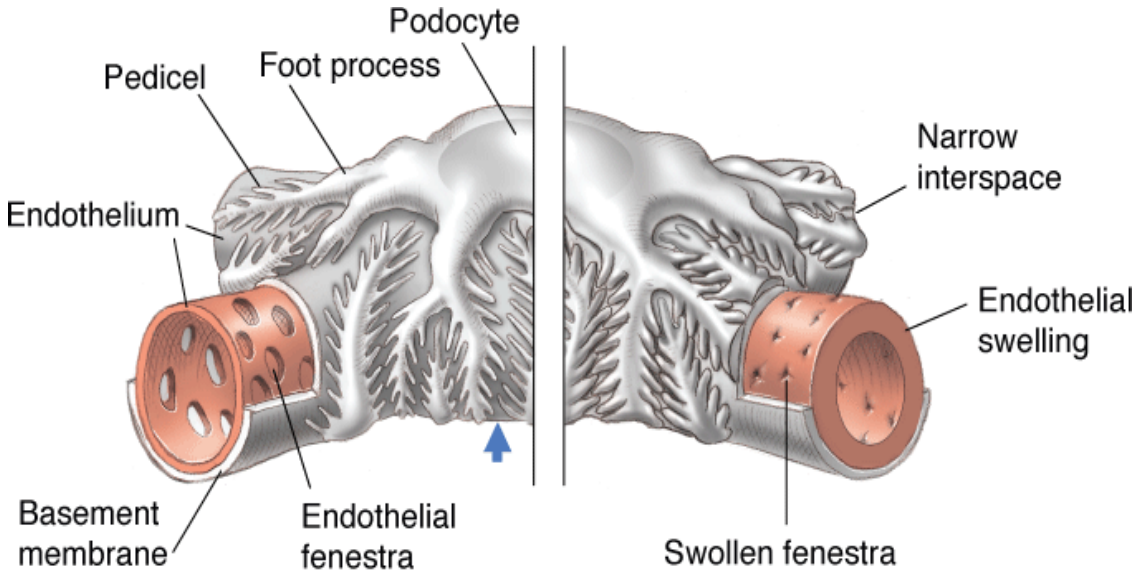
In preeclampsia plasma uricacid increases due to increased tubular reabsorption, increased placental urate production due to oxidative stress.There is also decreased urinary calcium excretion due to tubular reabsorption. Rarely acute tubular necrosis and acute cortical necrosis occurs, which is usually associated with obstetric haemorrhage.

PROTEINURIA:

Approximately 10-15% of women with HELLP syndrome and17% of eclamptic women didn't have proteinuria. Proteinuria is defined as 24 hour urine protein in clean catch midstream sample with >300mg/24 hours or spot urine protein creatinine ratio ≥0.3. A more accurate method involves measurement of albumin excretion. Albumin filtration exceeds that of larger globulins, and with glomerular disease such as preeclampsia, much of the protein in urine is albumin. Patients with nephrotic-range proteinuria has been included by most to be a severe disease.

ANATOMICAL CHANGES :

Hallmark feature is glomerular capillary endotheliosis. Other findings are depicted in the figure below,



LIVER:

Pain in the right upper or mid epigastric region is seen in severe disease. The characteristic lesion is periportal haemorrhage in the liver periphery. Elevation in hepatic transaminase seldom rises above 500 U/L sometimes upto 2000U/L. There is an inverse relationship between platelet levels and serum liver enzymes. Both of them returns to normal usually within 3 days following delivery. The hepatic haemorrhage from infarcted areas extend to form hepatic hematoma which lead to subcapsular hematoma. It can be managed conservatively sometimes needing surgical intervention and liver transplant. Acute fatty liver of pregnancy is one of

the differential diagnosis of preeclampsia (Sibai, 2007). It has also arises late in pregnancy, and often with associated hypertension, elevated serum transaminase and creatinine levels, and thrombocytopenia.

There is increased likelihood of hepatic hematoma and rupture in HELLP syndrome. The subcapsular liver hematoma has 1.6 percent incidence. Other complications are eclampsia—6 percent, placental abruption—10 percent, acute kidney injury—5 percent, and pulmonary edema—10 percent, stroke, coagulopathy, acute respiratory distress syndrome, and sepsis.

BRAIN:

The pathological findings are intracerebral hemorrhage, cortical and subcortical petechial hemorrhage, subcortical edema. The microscopic picture is fibrinoid necrosis of the arterial wall and perivascular microinfarcts and haemorrhages. The pathophysiology of cerebral changes can be explained by two theories,

- in response to severe hypertension there is vasospasm with decreased blood flow ,cerebral ischemia and cytotoxic edema and infarction.

- sudden rise in blood pressure leads to loss of cerebral autoregulatory mechanisms with end capillary endothelial injury with hyper perfusion, with extravasation of plasma and red cells through the tight junction leading to edema.

The preeclampsia syndrome has endothelial activation associated with an interendothelial cell leak that develops at blood pressure levels much lower than those causing vasogenic edema and has a loss of upper-limit autoregulation (Zeeman and colleagues, 2009b). This was termed as *reversible posterior leukoencephalopathy syndrome* by Hinchey and colleagues (1996).Recently it is referred as the *posterior reversible encephalopathy syndrome—PRES* (Narbone and associates, 2006). Eclampsia occurs when cerebral hyperperfusion forces capillary fluid interstitially because of endothelial activation and leads to perivascular edema as occurring in preeclampsia syndrome. Thus eclampsia has similarities to PRES.

Clinical manifestations include

Headache, scotoma, convulsions in eclampsia, blindness and generalised cerebral edema which compress the cerebral ventricles leading to manifestations of transtentorial herniation. The manifestations of cerebral edema varies from lethargy, confusion to coma .The unique

feature of the headache is that it improves after magnesium sulphate infusion. A localised hypodense lesion in CT or a hyperintense T2 image in MRI in parieto-occipital region occurs.

Scotoma, blurred vision, or diplopia are commonly seen in severe preeclampsia and eclampsia. These usually resolve with treatment with magnesium sulfate and or with lowering of blood pressure. Blindness has low occurrence, is usually reversible, and may arise from the visual cortex zone of the occipital lobe, the lateral geniculate nuclei, and retina. In the retina, lesions may include ischemia, infarction, and detachment. Occipital blindness also called as *amaurosis,* usually resolves completely. Retinal blindness is called as *Purtscher retinopathy.* Asymptomatic serous retinal detachment is relatively common and is obvious by examination (Saito and Tano, 1998). Surgical mode of treatment is rarely needed .There is usually good prognosis and vision usually comes back to normal within a week.

TESTS FOR PREDICTION OF PREECLAMPSIA

1. placental perfusion /vascular resistance related tests

Roll over test(28-32 weeks),isometric hand grip test, angiotensin II infusion test, uterine artery Doppler velocimetry (increased resistance).

2. fetal- placental unit endocrine dysfunction

Human chorionic gonadotropin (hCG), alpha-fetoprotein (AFP), estriol, pregnancy-associated protein A (PAPP A), inhibin A, activin A, placental protein 13, corticotropin-releasing hormone .None of these are significant for prediction of preeclampsia.

3. renal dysfunction related tests

- serum uricacid– increase results from decreased uric acid clearance from reduced glomerular filtration, increased tubular reabsorption, reduced secretion.(sensitivity 0 to 55 percent and specificity 77 to 95 percent).

- Microalbuminuria (poor predictive value specificity from 29 -97 %), urinary calcium or kallikrein, micro transferrinuria, *N*-acetyl-β glucosaminidase.

4. endothelial dysfunction and oxidative stress related

- fibronectin (not useful)

- thrombocytopenia and platelet dysfunction

- Increased levels of lipid peroxides coupled with decreased antioxidant activity might predict preeclampsia.

- Hyperhomocysteinemia (not useful)

- angiogenic factors -Serum levels of proangiogenic factors, such as vascular endothelial growth factor (VEGF) and placental growth factor (PlGF), begin to decrease before clinical preeclampsia develops. At the same time, levels of some antiangiogenic factors such as soluble fms-like tyrosine kinase 1 (sFlt-1) and soluble endoglins (sEng) are increased. Because of these findings, measurement of their plasma levels may serve as predictive tests for preeclampsia.

- free fetal DNA – according to Holzgreve and associates (1998) there is increased fetal-maternal cell trafficking in preeclampsia complicating pregnancies. It is hypothesized that free DNA is released by accelerated apoptosis of cytotrophoblasts (DiFederico and colleagues, 1999). Conde-Agudelo and associates (2009) concluded that free fetal DNA quantification is not useful for prediction purposes.

PREVENTION :

Some Methods to Prevent Preeclampsia That Have Been Evaluated in Randomized Trials
Dietary modification—restricted salt diet, calcium supplementation, fish oil supplementation
Cardiovascular drugs—diuretics, antihypertensive drugs[39]
Antioxidants—ascorbic acid (vitamin C), -tocopherol (vitamin E)[40]
Antithrombotic drugs—low-dose aspirin[41], aspirin/dipyridamole, aspirin + heparin, aspirin + ketanserin

None of these are clinically useful[39] .

RELATION BETWEEN PREECLAMP SIA AND HYPOTHYROIDISM VARIOUS LITERATURES.

I. STUDY BY RICHARD J LEVINE ET AL[42],

Analysed the relationship between preeclampsia, hypothyroidism and soluble fms like tyrosine kinase 1.

Soluble fms like tyrosine kinase -1 is an antiangiogenic protein .It inhibits vascular endothelial growth factor and placental growth factor. There is an increase in fms like tyrosine kinase in last two months of

pregnancy. Preeclamptic women are more prone for hypothyroidism in later life. A nested case control study was done in the Calcium for Pre-eclampsia Prevention (CPEP) trial cohort.

There is a greater increase in TSH concentration during pregnancy in pre-eclamptic women than normotensive controls and the increase is proportional with the magnitude of the soluble fms-like tyrosine kinase 1 concentration during pre-eclampsia. Cancer patients treated with VEGF inhibitors have higher risk of hypothyroidism[43-45] .

Compared with the controls preeclampsia had greater BMI . A large number of patients had preterm delivery ($p<0.001$) and small for gestational age infants($P=0.002$). The baseline mean TSH between the two groups didn't differ (p0.14) whereas there is significant difference with the onset of preeclampsia(p0.007).Predelivery to baseline ratio of 95% confidence interval between cases and controls1.64 (1.29 to 2.08). There is a more fall in free T3 than free T4 of predelivery sample compared with the baseline values .

The ratio of predelivery to baseline values is significant in cases than controls[49]. Preeclampsia is subsequently associated with subclinical hypothyroidism in pregnancy and those with a history of preeclampsia are

at increased risk of hypothyroidism in later life and future cardiovascular and renal disease.

Since subclinical hypothyroidism can progress to overt hypothyroidism it implicates that preeclampsia women should be screened for hypothyroidism[46] .

II.MATERNAL THYROID HORMONAL STATUS IN PREECLAMPSIA

Although pregnancy is associated with mild hyperthyroxinemia ,preeclampsia is associated with hypothyroidism which might reflect the severity of preeclampsia. On the contrary preeclampsia develops in 16.7 % of subclinical cases and 43.7 % of overt hypothyroid cases. In preeclampsia the decreased thyroid hormones concentrations is due to reduced plasma protein concentrations [47] and high endothelin levels[48] produced by vascular endothelium after a vascular injury. It was a case control study from July 2001 to December 2002 on 82 preeclampsia patients in third trimester after excluding other medical co morbidities.

According to an Indian study the mean TSH value in preeclampsia is 3.8±0.53mIU/ml whereas in normal group it is 2.3±0.24 mIU/ml[49]. In the present study venous blood taken and analysed for free T3,free T4,TSH by

chemiluminescene assay. According to this study , the preeclampsia patients have higher incidence of biochemical hypothyroidism[47,50-52.].

The non thyroid illness acting as stress factor and reduced plasma albumin concentration causes mild alterations in thyroid function[53,54].The total T3,total T4 decreases. The free T3 titres are significantly related to decreased plasma albumin. The decrease in thyroid hormone with increase in TSH is proportional to high levels of endothelin[54].

The decrease in thyroid binding globulin(TBG) ,T3,T4 can be due to faulty estrogen production by the placenta in preeclampsia[47]. The T3 ,T4 may be normal in the early stage of preeclampsia .Decrease in T3,T4 with increase in TSH can be observed at a later stage of the disease. Rarely this TSH increase may be due to autoimmune thyroiditis since the increase is not too high and they became normal 6 weeks postpartum.

The preeclamptic women with hypothyroidism have small for gestational age infants[47,50,52].Birth weight of infants is inversely proportional to the TSH level [47].By this study they concluded that birth weight of infants has no association with free T3,T4,TSH.The detection of thyroid abnormalities and proper treatment might influence the occurrence and severity of preeclampsia.

III.PREECLAMPSIA LINKED TO REDUCED THYROID FUNCTION

Women with preeclampsia have high TSH due to soluble fms like tyrosine kinase activity. Women with preeclampsia in their prior pregnancy have increased risk of hypothyroidism in subsequent years. Mostly the TSH abnormality is subclinical hypothyroid without clinical manifestations[56].

IV.THYROID HORMONES IN PREGNANCY AND PREECLAMPSIA[57]

In pregnancy there is increased demand of thyroid and there is increased iodine uptake with increased production of thyroid hormones. Estrogen causes a rise in production of TBG and the placenta releases several thyroid stimulatory substances such as hCG. The alpha subunit of hCG has thyrotropic properties[58]. In preeclampsia, due to the placental dysfunction there is decreased estrogen production leading to reduced production of TBG,TT3,TT4 and growth retardation in fetus[59].

The endothelial dysfunction and oxidative stress also play a role[60-62]. The oxidative stress contributing to hyperuricemia in preeclampsia. There is production of superoxide anions leading to reduced production of nitric oxide with vasoconstriction[62,63]. The reduced nitric oxide production in

endothelial cell dysfunction might lead to hypothyroidism in preeclampsia[64,65.]

There is significant negative correlation between TSH levels and birth weight of infants ($P<0.001$) and significant positive correlation between birth weight and serum albumin levels($p<0.001$). There is significant correlation between serum uric acid and birth weight of infants ($p<0.001$) . There is significant negative correlation between TSH and serum albumin levels ($p<0.01$).

The total TT3 were lower in severe preeclampsia compared to normotensive controls. Causes of low TT3 include inability to compensate for fetal demand, increased placental breakdown of hormones, and transfer of maternal T4 to fetus. There is reduced peripheral conversion of T4 to T3 in preeclamptic women with liver and kidney involvement.

Low T3 syndrome is also seen in preeclampsia . There is urinary loss of proteins and protein bound hormones .It is a reflection of inability to compensate for increased fetal demand, transfer of maternal T4 to fetus and increased placental breakdown of thyroid .There are various controversies regarding TT4 levels with some studies reporting lower TT4[47,66] and some reflecting higher TT4 levels[48].

The degree of hypothyroidism might reflect the severity of preeclampsia. Mostly the preeclampsia is associated with biochemical hypothyroidism in contrast to normotensive women [47,66,67,68].

Thyroid hormones are responsible for neurodevelopment of fetus and cause preterm birth in preeclampsia. Identification of thyroid abnormalities and timely intervention with supplementation will help in preventing the occurrence and improving the outcome. Future studies are needed to analyse the association between two.

V.A study by Wilson KL et al,

Analysed the diagnosis of Subclinical Hypothyroidism Early in Pregnancy Is a Risk Factor for the Development of Severe Preeclampsia.

They investigated the association between subclinical thyroid dysfunction and hypertensive disorders in those who presented before 20 weeks of gestation .Subclinical hypothyroidism causes endothelial dysfunction by decreasing nitric oxide production with impaired vascular relaxation .It leads to hypertension, heart failure and atherosclerotic disease.

The incidence of preeclampsia in euthyroid, subclinical hypothyroid and subclinical hyperthyroid are 8.5%,6.2%,10.9%. There is increased risk

of severe preeclampsia in women with subclinical hypothyroidism. (adjusted odds ratio, 1.6; 95% confidence interval, 1.1 to 2.4; P = 0.031).

Subclinical hypothyroidism a common condition of the reproductive age group may be associated with adverse perinatal outcomes[69].Universal screening of thyroid disorders should not be routinely implemented unless proven so. The ATA[70] and the Endocrine society[71] recommends the treatment of women with subclinical hypothyroidism.

MATERIALS & METHODS

MATERIALS AND METHODS

This study was conducted in Govt. R.S.R.M. Lyingin hospital, Royapuram, Chennai from January 2014 to December 2014.

TYPE OF STUDY-

Cross sectional study

SELECTION CRITERIA:

The duration of study was about one year .Women with preeclampsia and normotensive antenatal women who were attending our outpatient and inpatient department after 20 weeks of gestation fulfilling inclusion and exclusion criteria, were counselled for investing thyroid function tests .Informed written consent were obtained from women who were willing for the study. The study was approved by our ethical committee.

In our study 200 preeclampsia patients were compared with 200 normotensive age and gestational age matched controls. Free T3,free T4 and TSH were done for both the groups.

INCLUSION CRITERIA:

- 18 to 35 yrs
- pregnancies complicated by preeclampsia.
- singleton pregnancies.
- patient willing to give consent for the study.

EXCLUSION CRITERIA:

- Other hypertensive disorders of pregnancy (chronic hypertension, gestational hypertension, eclampsia)
- women with known thyroid disorders or on drugs for thyroid disorders.
- multiple pregnancies.
- patient on any drugs known to affect thyroid functions.
- other medical disorders complicating pregnancy .
- patient not willing to give consent

METHOD OF STUDY:

The patients meeting all the inclusion and exclusion criteria were included in the study after obtaining an informed written consent. A total of 200 preeclampsia patients were compared with their age and gestational age matched controls. The study adopted a cross sectional approach.

Five ml of fasting venous blood was obtained from each patient from the cubital vein in (a) preeclamptics once it is diagnosed but before initiation of the treatment and (b) the normotensive controls. All the samples were labelled with different code numbers and sent to the laboratory. The separated sera was stored at -20^{o} C until assayed.

Thyroid function tests (FT3,FT4,TSH)were done for both the groups by chemiluminescence ELISA(CMIA).The results were analysed and its correlation with preeclampsia was done.

STATISTICAL ANALYSIS

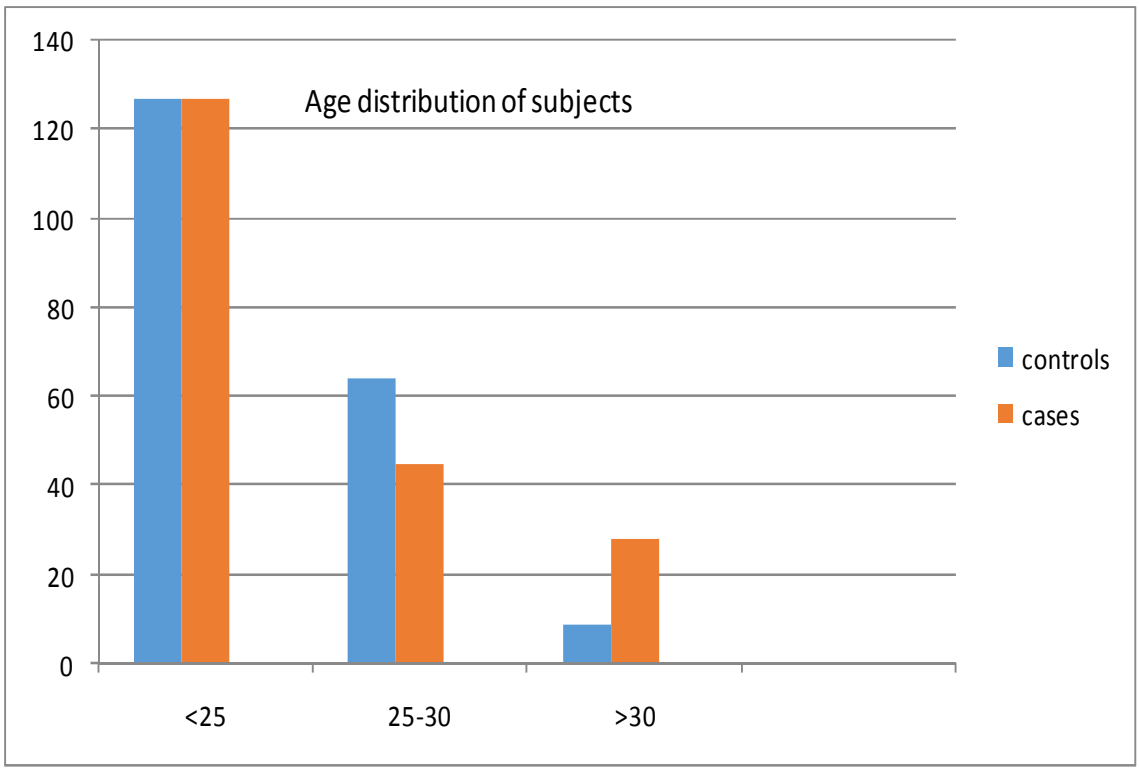
Continuous variables were expressed as mean and standard deviation whereas categorical variables are expressed as frequency and percentage. Descriptive statistics were used to present demographic data. Normality of the data was assessed by Shapiros-Wilk's test. Categorical variables between groups are analysed using Chi square test or Fisher's exact test based on number of observations. Between groups means were compared using independent sample t test. A two sided P value <0.05 was taken as statistically significant.

RESULTS & ANALYSIS

RESULTS.

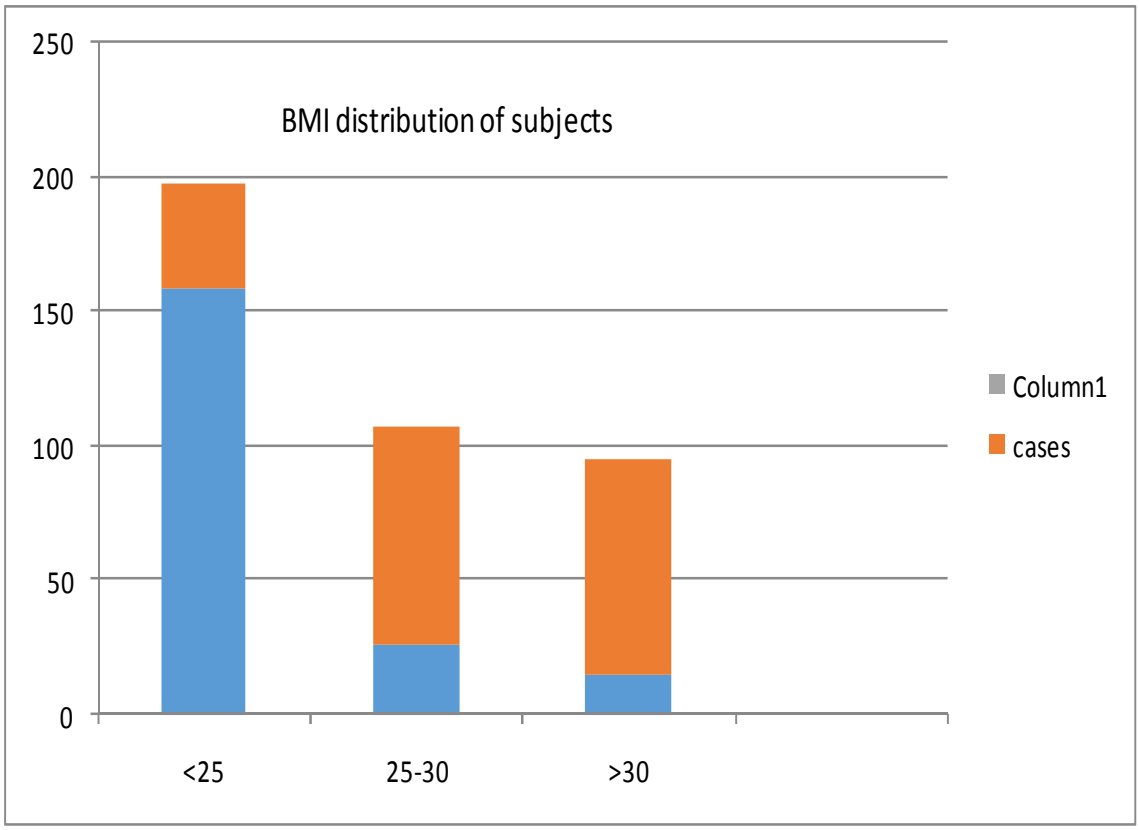
In this study we have compared 200 preeclampsia patients with 200 normotensive controls. The age distribution of subjects is depicted in figure 1.The majority of subjects were in the <25 years age group.

FIGURE 1



The BMI distribution of the subjects are shown in figure 2. As can be seen the majority of control subjects had BMI <25 whereas in cases the BMI was >25 in majority. The relative proportions of each can be seen from figure 2.

FIGURE 2



The baseline characteristics of the study population are tabulated in table 1

TABLE 1

Parameter	controls	patients	P value
Age ±SD	23.99±3.5	24.36±4.25	0.35
Gestational age	34.9±3.4	34.7±2.9	0.66

As seen in table 1 both the groups were equally distributed with respect to age and gestational age (P 0.35 and 0.66 respectively).

The other parameters are given in table 2.

TABLE 2

Variable	group	N	Mean	Std. Deviation	P value
BMI	Patients	200	29.103	4.2763	0.001
	controls	200	23.904	2.9348	
BP	Patients	200	151.14	8.212	<0.0001
	Controls	200	121.47	71.113	

There is a significant difference of BMI and BP between the groups (P is 0.001 for BMI and <0.0001 for BP). The preeclamptic patients were significantly overweight than their normotensive counterparts.

Thyroid function tests were done in both the groups. The tests done were free T3, free T4 and TSH. The results are shown in table 3.

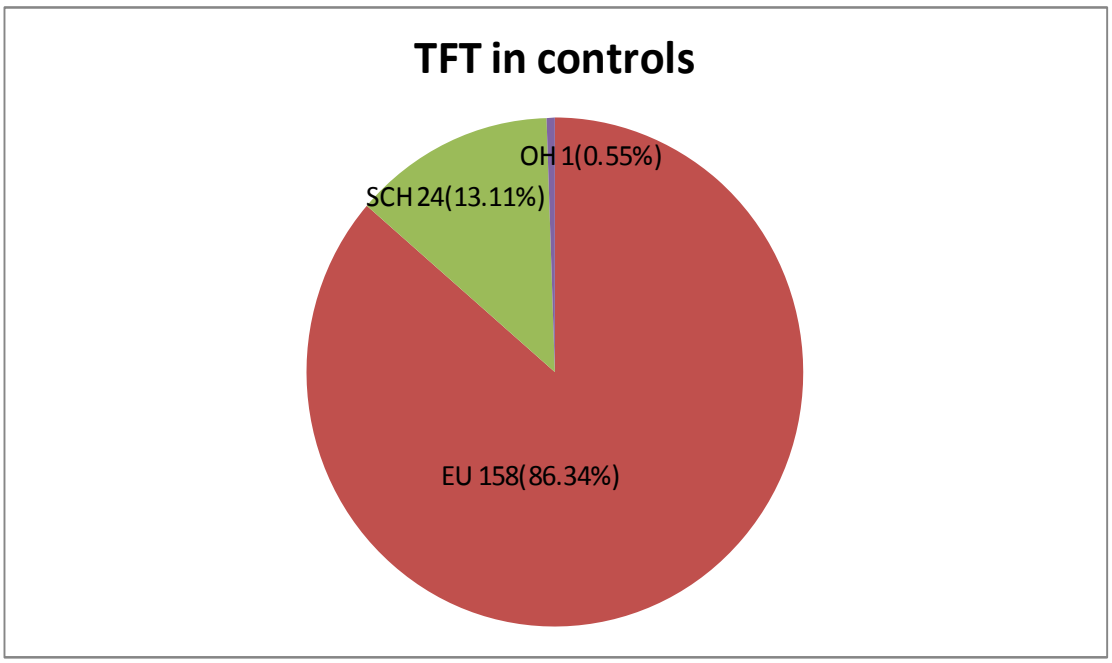
TABLE 3

Variable	Group	N	Mean	SD	P value
FT3	Patients	200	2.213	0.3621	<0.0001
	Controls	200	2.396	0.4452	
FT4	Patients	200	1.240	0.2337	<0.0001
	Controls	200	1.351	0.2454	
TSH	Patients	200	3.1289	2.01076	<0.0001
	Controls	200	2.2479	1.07183	

The mean FT3 and FT4 levels in both the groups were within the normal range. There is also a significant difference in FT3 and FT4 between the groups(P<0.0001),with preeclampsia patients having mean FT3 and FT4 lower than the controls..TSH levels were significantly more for the preeclamptic group (3.12 vs 2.24, P<0.0001).

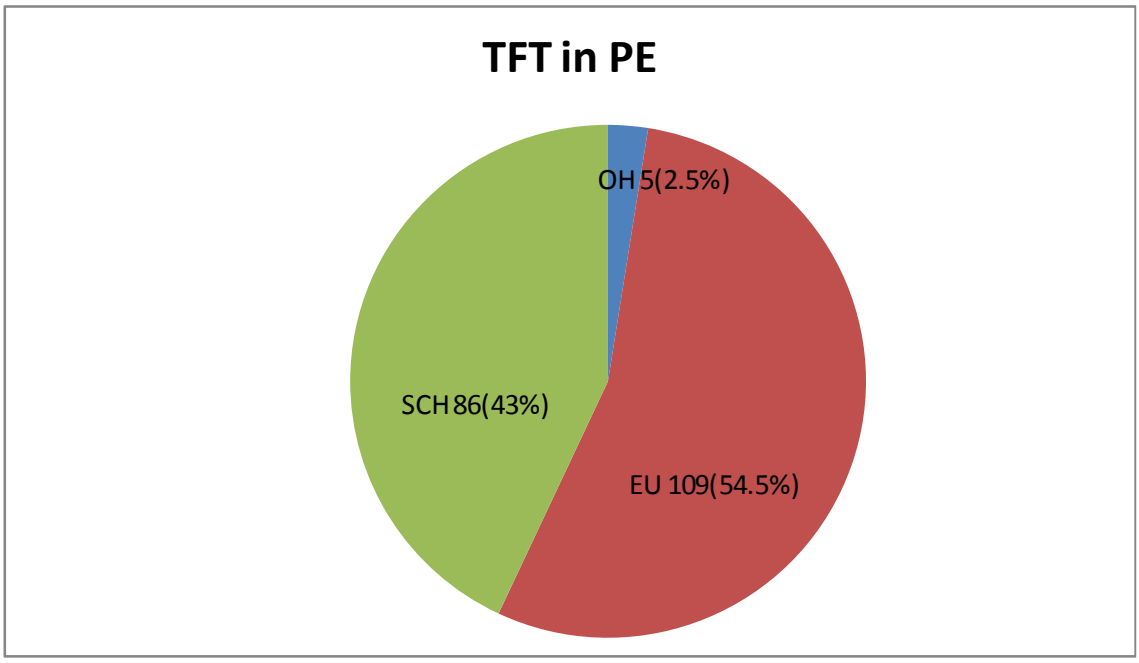
The relative proportions of Euthyroids (EU), subclinical hypothyroids(SCH) and overt hypothyroids(OH) in controls and in PE group are shown in figure 3 and 4 respectively.

FIGURE 3



Among the study population, majority of the subjects belonged to the EU group (86.3% and 54.5% in controls and cases respectively). SCH was more common in the PE group as compared to the controls. The same was true for OH too.

FIGURE 4



The thyroid function tests in severe as compared to mild preeclampsia is given in table 4.

TABLE 4

Variable	Severity	N	Mean	SD	P value
FT3	Severe	41	1.985	0.3046	<0.0001
	mild	159	2.271	0.3532	
FT4	Severe	41	1.104	0.2230	<0.0001
	mild	159	1.275	0.2240	
TSH	Severe	41	5.2949	2.58681	<0.0001
	mild	159	2.5703	1.36632	

There were 41 patients with severe PE as compared to 159 in the mild PE group.As is shown in table 4 , the TFT abnormalities were more common in the severe PE group as compared to the mild PE group.

The TSH was significantly more in severe PE as compared with mild PE(P <0.0001). Both the FT3 and FT4 were numerically less in the severe PE group than the mild PE group with the P value being statistically significant (P<0.0001).

The relation between the onset of preeclampsia and the TFTs is compared and shown in table 5.

TABLE 5

Variable	Onset(weeks)	N	Mean	SD	P value
FT3	≤34 >34	67 133	2.158 2.240	0.3197 0.3799	0.130
FT4	≤34 >34	67 133	1.224 1.249	0.2436 0.2291	0.473
TSH	≤34 >34	67 133	3.1224 3.1321	2.11331 1.96526	0.974

There were 67 patients in the PE group where the disease has occurred before 34 weeks and 133 in the ≥ 34 group. Thyroid function tests were comparable in both the groups(P statistically not significant).

DISCUSSION

DISCUSSION

In this present study we have studied the prevalence of hypothyroidism in preeclamptic patients and the correlation between hypothyroidism and the severity of preeclampsia .We have also analysed the relationship between the onset of preeclampsia and hypothyroidism. The patients were divided into two groups; one group containing 200 preeclamptic patients and the control group of 200 normotensive subjects.

The age distribution of patients included in our study ranged from 18 to 35 years. Majority of them belonged to the less than 25 years age group in both the groups. The mean age of the patients in control and study group was 23.99±3.5 and 24.36±4.25 years respectively, which was comparable (p=0.35).

Both the groups were comparable with respect to their gestational age too(controls-34.9±3.4 weeks and study group 34.7±2.9 weeks) (p=0.66).

In a similar study done by Ashokkumar et al[72], comparing pre-eclamptics with normotensive women, the mean (± SD) age of the study group and the control group was 28.4 ± 6.24 years and 27.5±5.91 years respectively which is quite similar to our subjects.

The mean (± SD) gestational age when TFT was done was 34.3±2.92 weeks in the study group and 35.1 ± 2.86 weeks in the control group which is similar to our present study.

BMI in the present study is significantly more in the study group as compared to the controls (p=0.001).The mean BMI of patients is 29.103±4.2763 and that of the controls is 23.904±2.9348.

The preeclamptic patients were significantly overweight compared to the normotensive controls and it is indeed a known risk factor for preeclampsia[2].

TSH, free T3 ,free T4 were done for both the groups and the results were analysed. The control group in our study had 158 euthyroid subjects (86.34%) ,24 subclinical hypothyroids(13.11%) and one overt hypothyroid(0.55%) .

In the preeclampsia group 109 were euthyroids (54.5%), 86 are subclinical hypothyroids (43%),5 are overt hypothyroids(2.5%). These findings are in accordance with the previous literature stating that preeclamptic women have a higher incidence and prevalence of biochemical hypothyroidism than the normotensive population[47,50,51,52].

The mean free T3 levels in both the groups(2.213 Vs 2.396) were within the normal range ,with the PE group having numerically less FT3 than the controls. The P value is statistically significant (P <0.0001).

The mean free T4 values in our study in preeclampsia Vs controls is (1.240 Vs 1.351) which remains within the normal trimester specific range of FT4. However the PE group had a mean FT4 level which was lower than the controls and the difference was significant statistically (p <0.0001).

The mean TSH value in the preeclamptic group is more than the controls in our study (3.1289 Vs 2.2479)and is significant (P<0.0001).The mean TSH in the preeclamptic group is 3.1289 which is above the cut off for diagnosing SCH during pregnancy in the second and third trimester.

Thus subclinical hypothyroidism is more common in the preeclamptic group in the present study. One reason for this subclinical hypothyroidism could be that the hypothyroidism is in the evolving phase and a larger study with a longer term follow up may be needed to document it.

In a similar study by Ashok Kumar et al [72],the mean FT3 and FT4 were not significantly different in the two groups and the mean TSH

value was significantly higher in the preeclamptic women than that of controls ($P < 0.001$).This is partly comparable to our study where the mean TSH,FT3 and FT4 are significantly different between the groups with the PE group having a high mean TSH and a low mean FT3, FT4.

In an another Indian study the mean TSH titres in the preeclamptic pregnancies has been reported to be 3.8±0.53mIU/ml while in the normal pregnancies it was 2.3±0.24mIU/ ml[73] which again is comparable to the present study.

In a study by Wilson KL et al[74] ,women with subclinical hyperthyroidism had an incidence of hypertensive disorders of 6.2% as compared with 8.5% of euthyroid women and 10.9% of subclinical hypothyroid women. After adjusting ,only women with subclinical hypothyroidism were at increased risk for severe preeclampsia (adjusted odds ratio, 1.6; 95% confidence interval, 1.1 to 2.4; $P = 0.031$) pointing towards a causal role.

In the calcium for preeclampsia prevention cohort, the mean TSH values were increased 2.42 times above baseline in the PE group as compared with a 1.48 times increase in controls[42]. The ratio of the predelivery to baseline TSH ratio of cases to that of the controls was 1.64

(95% confidence interval 1.29 to 2.08) and there is a decrease in free T3 in preeclampsia women than in controls.

Only the predelivery specimens and not the baseline TSH values were significantly higher than in controls .The increase in predelivery TSH values was associated with an increase in the soluble fms like tyrosine kinase and preeclampsia may also predispose to reduced thyroid functions in later years[42].

This study thus suggests PE as a possible risk factor for hypothyroidism and the mechanisms could be one mediated through s-fms like tyrosine kinase. Other mechanism postulated to explain hypothyroidism in PE is placental dysfunction in PE[56] .

In the Nord-Trondelag Health Study, women with history of preeclampsia in their first pregnancy were associated with high concentrations of thyroid stimulating hormone without thyroid peroxidase antibodies, suggesting hypothyroid function in the absence of an autoimmune process. This association was especially strong (5.8, 1.3 to 25.5) if there is history of preeclampsia in more than one pregnancy.

According to Ghalia Ashoor et al**,** measurement of maternal serum TSH can improve the prediction of late-PE provided by a

combination of factors in the maternal history and the measurements of mean arterial pressure and uterine artery pulsatility index[75].

Hypothyroidism may also play a direct role in causing pregnancy hypertension because thyroid hormones act directly on peripheral arterioles to cause dilation (Dernellis and Panaretou, 2002). A study on nonpregnant individuals reported that hypothyroidism is associated with an increase in peripheral resistance due to increased arterial wall thickness (Giannattasio*et al*., 1997) and endothelial dysfunction (Virdis*et al*.,2009). This can be reversed by treatment with thyroid hormones (Giannattasio*et al*., 1997; Dernellis and Panaretou, 2002).

The above mentioned studies point towards an association between PE and hypothyroidism and the association could be either way[42,56,74,75]

On the other hand there a few studies arguing against any relationship between hypothyroidism and preeclampsia. In a study done in Jordon, there was no significant difference in the values of FT3, FT4, TSH between the preeclampsia and the control group[76].

In our present study, the mean TSH is significantly higher in the preeclamptic group and FT3, FT4 significantly low in the preclamptics.

Though a high TSH and a low FT4,FT3 might suggest SCH as a cause for preeclampsia, both the FT3 and FT4 were within the trimester specific range preventing us from drawing any conclusion out of it. Larger studies with a long term follow up would be needed to clarify this.

We have also tried to analyse the relationship between the severity of preeclampsia and hypothyroidism. Out of the 200 preeclamptic patients,41 belonged to the severe and 159 belonged to the mild preeclampsia group.

The TSH was significantly more in the severe preeclampsia group as compared to mild preeclampsia (5.29 Vs 2.57; P <0.0001) . The values of free T4 are (1.104 Vs 1.275) numerically less in severe preeclampsia than mild preeclampsia and they were statistically significant.(P <0.0001). Similarly the values of free T3 are numerically less in the preeclamptics than the controls(1.985 Vs 2.271) with a statistically significant P value (P <0.0001).

These findings strongly suggest an association between the severity of preeclampsia and hypothyroidism.

There are evidences stating that the underlying mechanism for early-PE is impaired trophoblastic invasion of the maternal spiral arteries, reduced placental perfusion and fetal growth restriction (Plasencia*et al*.,

2007; Yu *et al*., 2008; Poon *et al*., 2009),whereas in late-PE the main pathophysiological processes resemble those of the metabolic syndrome with an increase in adipose tissue and impaired glucose and lipid metabolism (Witlin*et al*., 2000; Moldenhauer*et al*., 2003; Vatten*et al*., 2004; D'Anna*et al*., 2006; Egbor*et al*., 2006; Poon *et al*., 2009).

The association between hypothyroidism and late-PE may be mediated by the well described role of thyroid hormones in glucose homeostasis and in the synthesis, metabolism and mobilization of lipids (Duntas, 2002; Pearce, 2004; Chidake l*et al*., 2005).

We have tried to analyse the TFT in early as compared to late onset PE. Out of the 200 preeclamptic patients 67 were early PE and 133 were late PE. The thyroid function tests were comparable between both the groups in our study. So we could not document an association between the onset of preeclampsia and hypothyroidism.

SUMMARY

SUMMARY

The study was conducted in Govt RSRM hospital. The study group was divided into two groups with one containing 200 preclamptic patients and the other containing 200 normotensive women.

TSH, FT3, FT4 are done in both the groups and the results are analysed. The two groups are comparable with respect to their age and gestational age.

There is significant difference of FT3 and FT4 between the groups with the PE group having numerically less FT3 and FT4 than the controls. similarly there is a significant difference of TSH between the groups, with PE ha group having higher TSH than the controls.

The prevalence of SCH in controls is 13.11% in contrast to 43% in preeclamptics and the prevalence of OH in controls Vs preeclamptics is 0.55% Vs 2.5%.

Hypothyroidism is more common and the TSH levels are significantly higher in the severe PE as compared with mild PE, indicating a relation between the severity and hypothyroidism. However our study could not find any significant relation between the onset of PE and hypothyroidism.

The preeclamptic women have higher prevalence of SCH than the normotensive women in this study . So far screening for hypothyroidism is not routinely recommended in pregnancies and it is done on an individual basis.

Since SCH and OH is associated with lots of maternal and perinatal adverse effects, there is a need for early diagnosis of these disorders and hence a routine screening of antenatal women for thyroid function should be made mandatory. The recent trend is also towards treating women with SCH as per the ATA and the endocrine society recommendations.

CONCLUSION

CONCLUSION

To conclude by our study, that the preeclamptics have a higher incidence of hypothyroidism (OH and SCH) in contrast to the normotensive women and there is a correlation between the severity of preeclampsia and hypothyroidism. There is no association between the onset of preeclampsia and hypothyroidism.

The treatment of OH and SCH is mandatory and in future there should be a changing trend towards routine screening of hypothyroidism in contrast to targeted screening, but further larger studies are needed to support this fact.

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ANNEXURES

ABBREVIATION

TFT	-	Thyroid Function Test
FT3	-	Free T3
FT4	-	Free T4
PE	-	Preeclampsia
SCH	-	Subclinical Hypothyroidism
ATA	-	American Thyroid Association
OH	-	Overt Hypothyroisim
EU	-	Euthyroid
TSH	-	Thyroid Stimulating Hormone
TRH	-	Thyroid Releasing Hormone
TT3	-	Total T3
TT4	-	Total T4
VEGF	-	Vascular Endothelial Growth Factor
CPEP	-	Calcium for Pre-eclampsia Prevention
PLGF	-	Placental Growth Factor
sFlt-1	-	solubleFms-like tyrosine kinase 1
AST	-	Aspartate Trans Aminase

ALT	-	Alanine Trans Aminase
LDH	-	Lactate Dehydrogenase
sEng	-	Soluble Endoglins
ELISA	-	Enzyme Linked Immuno Sorbent Assay
CMIA	-	Chemiluminescence ELISA
hCG	-	human Chorionic Gonadotropin
IVF	-	InVitro Fertilization
FASTER trial	-	First And Second Trimester Evaluation Of Risk
AFP	-	Alpha Feto Protein

PROFORMA

1. Name

   Age

   IP/op.no

   Address

   Socioeconomic status

   Education status

2. Menstrual history

   age of menarche

   cycles&flow

   menorrhagia/oligomenorrhoea

   LMP (Last Menstrual Period)

3. obstetric history

   age of marriage

   consanguinity

   gravida/para/live/abortion

   mode of prior delivery/any complications/baby details

   Last Child Birth.

4. Personal history:

   known case of Diabetes /Hypertension / Bronchial Asthma/Tuberculosis / Heart disease/thyroid disease.

   if patient is on any drugs for thyroid disorders or on any other drugs affecting thyroid functions.

5. family h/o
6. general examination including BP/urine albumin
7. breast examination
8. Systemic examination
9. Abdominal examination
10. Per vaginal examination
11. Investigations:

    Urine dipstick protein

    24 hour urinary protein

    Thyroid function test

தகவல் படிவம்

ஸ்டான்லி மருத்துவமனையின் ஆர். எஸ். ஆர். எம். மருத்துவமனையில் மகப்பேறு மற்றும் பெண்கள் நல மருத்துவ துறையில் மேற்கொள்ளப்படும் ஆய்வு தொடர்பான தகவல் படிவம் இது.

இந்த ஆய்வு மரு. நா. உமாதேவி அவர்களால் மற்றும் பிற அனுபவம் வாய்ந்த மருத்துவர்களின் உதவியோடு நடத்தப்படுகிறது.

இந்த ஆய்வு கற்பகாலத்தில் ஏற்படும் உயர் இரத்த அழுத்த நோய்க்கும், தைராய்டு குறைபாட்டிற்கும் உள்ள தொடர்பினை அறிவதற்கு மேற்கொள்ளப்படுகிறது.

இந்த ஆய்வு பெண்கள் தங்கள் சுயவிருப்பத்துடன் பரிசோதனை செய்ய முன்வந்தால் மட்டுமே மேற்கொள்ளப்படும்.

ஒப்புதல் படிவம்

திரு. / திருமதி. .....

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என்ற விலாசத்தில் வசிக்கும் நான், எனக்கு அளிக்கப்பட்ட தகவல் படிவத்தில் உள்ள விவரங்களை படித்தும், கேட்டும் புரிந்து கொண்டேன்.

ஆய்வின் முடிவினை சொந்த அடையாளங்களை வெளியிடாமல் மருத்துவ ஆராய்ச்சிக்காக பயன்படுத்திக் கொள்ள சம்மதிக்கிறேன்.

நாள் : கையொப்பம்

இடம் : பெயர்

CONSENT FORM

STUDY TITLE : **"STUDY ON PREVALENCE OF HYPOTHYROIDISM IN WOMEN WITH PREECLAMPSIA"**

STUDY CENTRE : R.S.R.M. Lying in Hospital, Stanley Medical College, Chennai.

PARTICIPANT NAME : AGE: SEX: I.D.NO.

I confirm that I have understood the purpose of procedure for the above study, I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I understand that the investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties of published, unless as required under the law. I agree not to restrict the use of any results that arise from the study.

I hereby consent to participate in this study of **"STUDY ON PREVALENCE OF HYPOTHYROIDISM IN WOMEN WITH PREECLAMPSIA"**

Place : Signature of Investigator:

Date : Study Investigators Name

Institution : Signature / Thumb Impression of patient

INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Prevalence of Hypothyroidism in women with Preeclampsia

Principal Investigator : Dr. Uma Devi. N

Designation : PG in MS (O&G)

Department : Department of O&G
Government Stanley Medical College,
Chennai-01

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 02.07.2014 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

MASTER CHART

CONTROL GROUP

S.No	Name	Age	IP.No	obstetric code	gestational age (wks)	BMI	BP	urine albumin	fT3	fT4	TSH	Diagnosis
1	Durga	20	10796	primi	36	21.8	110/70	nil	2.2	0.96	3.34	SCH
2	stella	19	10824	primi	32	23.4	120/74	nil	2.7	1.21	4.73	SCH
3	thameema	20	10849	primi	37	23.5	110/76	nil	2.4	1.02	3.24	SCH
4	kavitha	25	10500	G2P1L1	38	24.1	120/72	nil	2.6	1.32	3.61	SCH
5	govindammal	28	10504	G2A1	32	22.3	110/82	nil	2.3	1.3	0.96	E
6	nancy	20	10846	primi	39	23.4	120/80	nil	3	1.3	3.74	SCH
7	jayalakshmi	26	10510	G4P2L2A1	33.2	24.3	110/76	nil	2.3	1.2	3.74	SCH
8	inbalakshmi	29	10842	G3P2L1	39	25.3	120/84	nil	2.5	1.34	3.9	SCH
9	reshmi	33	10830	G2P1L1	31.2	24	120/82	nil	2.5	1.23	2.37	E
10	janaki	24	10887	G3P1L1A1	31.2	24.6	120/86	nil	2.3	1.2	1.19	E
11	nisha	27	10393	G2P1L1	38.2	23.2	124/82	nil	3	1.4	1.4	E
12	bhavani	26	10905	G2P1L1	31.2	24.3	118/82	nil	2.2	1.2	0.98	E
13	vallivital	27	10578	G2P1L1	38.4	23.2	120/84	nil	2.3	1.31	3	E
14	mariyambeevi	19	10567	primi	37.4	24.5	128/76	nil	3	1.53	2.22	E
15	vennila	30	10652	primi	32.2	25.3	128/82	nil	2	1.17	4.47	SCH
16	sudha	20	10638	primi	38.4	24.6	110/82	nil	2.4	1.15	3.52	SCH
17	mariyambeevi	29	11342	primi	33	23.2	120/82	nil	3	1.52	2.22	E
18	lakshmi	23	11232	G2P1L1	33.2	24.3	120/82	nil	2.1	1.2	2.1	E
19	yasmin	21	11531	primi	28.3	23.4	120/84	nil	1.7	1.4	1.4	E
20	lavanya	23	11097	G2P1L0	36.6	24.4	112/72	nil	1.7	1.6	1.53	E
21	ramalakshmi	27	11550	primi	36.4	24.3	118/82	nil	2	1.2	2.2	E
22	syedali fathima	21	11402	primi	33.5	22.4	118/86	nil	1.8	1.53	2.18	E
23	rajeswari	32	11729	primi	37.6	30.7	120/86	nil	3.23	1.17	4.3	SCH
24	muniammal	24	11714	G4P2L1A1	33.4	22.4	112/78	nil	2.5	1.35	2.06	E
25	manimeagalai	28	11678	G3P1L1A1	36.1	22.3	120/72	nil	1.9	0.8	7.38	SCH
26	clara	25	11728	primi	32.1	22.5	118/78	nil	2.08	1.36	2.38	E
27	vasanthi	27	11719	G4P3L2	37.3	23.2	116/74	nil	1.93	1.62	2.77	E
28	mayilvani	21	11720	G2P1L1	30.2	23.8	118/78	nil	2	1	2.3	E
29	gomathy	29	11701	G3P1L1A1	38	25.7	120/86	nil	2.14	1.2	2.55	E
30	almas	20	11727	G2P1L1	28	22.2	112/82	nil	2.24	1.56	2.08	E
31	raadha	21	11737	G2P1L1	34.3	23.3	110/74	nil	2.14	1.49	1.01	E
32	jothi	21	11726	G2P1L1	32	25.2	120/86	nil	2.14	1.2	1.02	E
33	manimegalai	28	11717	G3P2L1	33.2	22.5	112/82	nil	2	1.2	1.4	E
34	meena	20	11696	primi	39.4	23.8	112/82	nil	1.97	0.9	3.21	SCH
35	mumtaz	26	11636	primi	37.2	31.2	110/82	nil	2.4	1.41	3.44	SCH
36	backiyalakshmi	23	11406	G2P1L1	33.2	22.9	116/78	nil	2.2	1.32	2.3	E
37	sajithabanu	25	11627	primi	36	23.2	112/86	nil	1.99	1.49	4	SCH
38	kesha	23	11479	G2P1L1	36	22.5	120/84	nil	2.3	1.1	2.73	E
39	abirami	21	11722	G3P2L1	30	22	114/84	nil	2.73	1.32	3.61	SCH
40	vijayabarathi	21	10012	primi	24	21.3	120/88	nil	2.19	0.82	2.7	E
41	jansi rani	24	11730	G2P1L0	38	22.2	120/78	nil	2.39	1.29	3.43	SCH
42	channammal	22	11905	G2P1L1	31.3	24.3	124/78	nil	2.73	1.1	1.86	E
43	sathyakala	25	11879	G2P1L1	24	21.5	120/80	trace	1.76	1.22	0.77	E
44	bhuvaneswari	26	11839	G2P1L1	38.4	22.1	120/76	nil	2.45	1.06	2.22	E
45	kiruba	32	11838	primi	37	23.3	110/78	nil	0.72	0.54	11.2	OH
46	shobana	23	11832	G3P1L1	32.1	21.6	120/86	nil	2.29	0.9	2.79	E
47	nagalakshmi	30	11413	G2P1L1	35.6	24.5	120/84	nil	1.9	1.3	0.66	E
48	karima	22	11887	primi	30.2	26.3	118/74	nil	2.52	0.89	2.51	E
49	suganthi	23	11881	G2P1L1	32	25.3	120/82	nil	2.38	0.99	3.71	SCH
50	malar	25	11890	primi	39.3	32.4	120/82	nil	2.22	1.12	3.26	SCH
51	gowri	30	11892	G2P1L1	37.6	24.7	124/82	nil	2	1.42	2.17	E
52	dhanalakshmi	28	119939	G2P1L0	32	31.6	118/82	nil	2	1.02	1.54	E
53	meena	24	11933	G3P1L1A1	37.5	31.4	120/74	nil	2.19	1.23	3.82	SCH
54	shaidha	23	11927	G2P1L1	37	23	118/86	nil	2.02	1.53	0.71	E

55	bhavani	29	11938	primi	29.1	29.6	120/82	nil	1.8	1.62	2.31	E
56	rihana	25	11961	G2P1L1	38.6	32.8	118/78	nil	1.69	1.08	3.27	SCH
57	aparna	23	11945	primi	30.2	31.2	120/82	nil	2.46	1.21	3.71	SCH
58	sarala	30	11934	G3P1L1	36	20.2	114/82	nil	1.9	1.02	1.11	E
59	vimala	20	12010	primi	28.3	23.2	118/74	nil	1.8	2.51	2.51	E
60	karima	23	12004	G2P1L1	32.3	26.8	120/82	nil	2.52	1.6	3.75	SCH
61	mamatha	20	11967	G4P2L0A1	37	23.3	110/80	nil	2.84	1.27	1.5	E
62	bhuvaneswari	21	12303	G3A2	33	31.4	110/70	nil	2.91	1.42	0.96	E
63	chitra	20	11018	primi	39	31.7	120/82	nil	2.1	1.4	0.32	E
64	lavanya	22	9590	primi	37	28.8	110/82	nil	1.9	1.6	2.12	E
65	mahalakshmi	21	9513	G2P1L1	32.2	23.2	120/82	nil	2.1	0.9	2.13	E
66	chandrika	24	9517	primi	37.2	23.5	124/82	nil	2.2	1.2	2.22	E
67	suganya	21	9612	G3P1L1A1	33.2	31.1	118/82	nil	2.32	1.29	1.75	E
68	rukzhana	20	9599	primi	37.3	32	120/84	nil	2.15	1.2	1.52	E
69	bhuvaneswari	30	9582	G2P1L1	36.1	27	120/84	nil	2.1	1.34	1.43	E
70	nirmala	25	9584	primi	31	28	118/84	nil	2.2	1.4	2.79	E
71	kalpana	24	9612	G2P1L1	36	27	120/72	trace	2.14	1.38	1.11	E
72	naventaj	22	9613	primi	38	23	118/82	nil	1.9	1.6	2.12	E
73	sasikala	22	9598	G2P1L1	36.3	32	110/72	nil	2.3	0.9	2.6	E
74	saranya	23	9602	G3P2L1	32.2	33	114/74	nil	3.2	1.4	2.98	E
75	ambiga	27	9673	G2P1L1	37	24.3	118/82	nil	3.1	1.3	2.92	E
76	selvamary	31	9684	primi	33.2	32	120/82	nil	2.5	1.2	2.44	E
77	jayalakshmi	28	9872	primi	36	31.6	118/82	nil	3.2	1.6	2.97	E
78	suganya	21	9869	primi	31.2	27.8	120/82	nil	2.2	1.46	1.9	E
79	vasanthi	26	9564	G3P1L1A1	38	26.4	120/72	nil	2	1.62	1.06	E
80	nandhini	21	9985	primi	33.2	23	118/74	nil	1.9	1.4	2.96	E
81	kavitha	22	9948	G2A1	37	27.5	120/82	nil	2.3	1.72	1.97	E
82	jancy	26	9971	primi	36.2	27.7	120/72	nil	2.4	1	2.97	E
83	thirumala	25	10000	G2P1L1	32.2	27.9	110/72	nil	2.2	0.9	3.67	SCH
84	usharani	28	9859	G3P2L2	36	25.5	110/82	nil	2.1	1.1	1.28	E
85	sangeetha	24	9914	primi	33.2	22.6	110/72	nil	2.1	1.62	2.6	E
86	sasikala	22	9598	G2P1L1	36.4	28.2	112/82	nil	2.3	1.25	0.73	E
87	sudharani	23	10090	G2P1L0	31.2	28.5	120/82	nil	2.4	1.12	2.57	E
88	navatha	22	9920	G3P1L1A1	28	25.2	118/82	nil	2	1.58	2.18	E
89	ameena	21	10151	G2P1L1	36.3	27	116/76	nil	2.2	1.23	2.16	E
90	mohanapriya	23	10138	G2A1	34	24.4	120/82	nil	1.92	1.73	1.85	E
91	anandhi	23	10143	G2P1L1	36.5	23.4	110/82	nil	2.12	1.45	3.84	SCH
92	anukirthiga	22	10187	primi	36.4	21.8	116/84	nil	2.23	1.62	1.91	E
93	nabeesa	23	10349	primi	24	22	120/82	nil	2.04	1.3	2.13	E
94	abirami	21	11722	G3P2L1	28	21	110/72	trace	1.9	1.47	1.5	E
95	sevanthi	21	12295	primi	36.3	25.8	120/82	nil	2.6	1.17	1.6	E
96	sarala	30	12287	G3P2L1	30.1	20.2	112/82	nil	2.5	1.32	2.78	E
97	vani	30	11934	G3P1L1A1	38.4	28.9	118/84	nil	2.6	1.17	2.1	E
98	alimabanu	23	12392	G2P1L0A0	33.4	23.1	120/82	nil	3.23	1.23	2.3	E
99	noorjahan	20	12127	primi	35	23.3	110/82	nil	3	1.31	2.1	E
100	kalaiarasi	25	12432	G3P1L1A1	33.2	24.3	110/72	nil	3.2	1.12	2.4	E
101	radhika	28	5636	G3P2L2	36.1	23	110/72	nil	1.9	1.72	2.13	E
102	mehta	20	5647	G2P1L1	30.1	21.5	120/72	nil	2.1	1.4	1.2	E
103	Shivani	22	5640	primi	38	24.3	110/72	nil	2.2	1.5	1.6	E
104	surekha	24	5350	G2P1L1	37.2	23.5	124/82	nil	2.4	1.57	1.3	E
105	ammulu	20	5633	G2P1L1	28.9	21.4	124/82	nil	3.1	1.82	2.1	E
106	mohana	25	5638	G2P1L1	39	23.3	122/80	nil	2.4	1.23	2.3	E
107	rekha	20	5600	primi	30.2	21.5	116/78	nil	2.1	1.67	1.8	E
108	devi	24	5651	G2P1L1	36.3	23.2	114/78	nil	2.7	1.3	1.7	E
109	jothy	35	5293	primi	29	22.2	110/82	nil	2.6	1.32	1.6	E
110	vadivukarasi	30	5168	G2P1L1	32.3	23.1	112/82	nil	3.2	1.49	1.43	E
111	sangeetha	22	5658	G2P1L1	37.4	21.2	1120/70	nil	1.9	1.12	2.21	E

112	fathima	23	5543	G2P1L!	38.4	20.1	104/72	trace	2.3	1.17	1.76	E
113	devi	22	5612	primi	32.1	21.3	110/72	nil	1.9	1.72	2.24	E
114	pooja	25	5644	G2P1L1	36.2	20.4	108/74	nil	3	1.67	2.34	E
115	kalaiarasi	19	5681	primi	33.2	22.1	106/82	nil	2.5	1.48	2.12	E
116	jayanthi	26	5631	G2P1L1	37	22.4	110/80	nil	3.24	1.38	2.45	E
117	mariyamma	31	5684	G3P2L1	32	21.3	110/70	nil	1.94	1.02	2.12	E
118	kokila	27	5639	primi	37.4	24.2	120/82	nil	2.23	1.22	2.12	E
119	kathijabee	24	5398	G3P1L1A1	31	23.1	110/82	nil	1.98	1.02	2.32	E
120	kuppulakshmi	23	5599	G2P1L1	28	22.1	120/78	nil	2.24	1.34	2.43	E
121	yogalakshmi	22	5646	primi	38.4	24.2	110/82	nil	2.16	1.62	2.52	E
122	reenarani	22	5575	primi	38	21.4	120/78	nil	2.05	1.4	1.24	E
123	lalithakumari	24	5584	G2P1L1	28	24.3	110/82	nil	2.45	1.43	1.09	E
124	mumtaz	28	5689	G2P1L1	37.4	22.3	120/82	nil	3.2	1.03	1.24	E
125	ammu	25	5624	G2P1L1	31	23.1	110/78	nil	3.4	1.7	1.45	E
126	kavitha	21	5654	G2P1L1	29	24.1	120/78	nil	2.56	1.21	1.23	E
127	sarbunisha	28	5679	G2P1L1	39	22.1	124/86	nil	2.42	1.32	1.32	E
128	yasodha	28	5675	G2P1L1	36.2	23.1	120/78	nil	2.34	1.42	1.07	E
129	megala	25	5623	primi	30.2	24.1	114/78	nil	3.23	1.42	1.18	E
130	bavani	21	5694	primi	36.4	23.1	118/82	nil	1.97	1.23	2.12	E
131	thasleen	24	5601	primi	37	24.2	120/78	nil	1.83	1.54	2.32	E
132	jaya	39	5691	G4P2L2A1	36.4	22.4	114/76	nil	2.23	1.32	2.23	E
133	indumathy	26	5682	G3P1L1A1	32.1	21.5	120/82	nil	2.43	1.62	2.12	E
134	saraswathy	28	5660	G3P1L1A1	38	24.3	116/76	nil	3.23	1.23	2.32	E
135	muniammal	21	5704	primi	33.4	21	120/84	nil	3.12	1.42	2.34	E
136	premalatha	28	5695	primi	37.4	23.2	118/78	nil	2.08	1.24	2.31	E
137	vachala	25	5673	G2P1L1	31.2	20.2	120/72	nil	2.14	1.32	2.13	E
138	jothilakshmi	18	5715	primi	30.2	23.2	118/72	nil	2.24	1.2	2.22	E
139	mahurnisha	20	5718	primi	38	24.3	120/78	nil	3.21	1.4	1.78	E
140	subamangalam	25	5170	G4A3	31	24.3	120/82	nil	1.93	1.24	1.79	E
141	shabana	23	5700	primi	34	23.4	112/78	nil	2.24	1.34	1.9	E
142	ramya	29	5687	G2P1L1	36.2	20.2	114/86	nil	2.22	1.63	2.09	E
143	suganthi	21	5734	primi	35.5	24.5	112/78	nil	2.41	1.72	2.56	E
144	subha	22	5711	primi	34.2	23.2	114/82	nil	2.22	1.23	1.92	E
145	kavitha	21	5736	primi	38.4	21.3	116/82	nil	3.21	0.97	1.86	E
146	anjali	29	5771	G2P1L1	37.4	24.2	120/72	nil	1.98	1.24	1.78	E
147	subetha	28	5739	G2P1L1	38.3	24.3	120/72	nil	2.21	1.45	1.67	E
148	nageswari	22	5770	G2P1L1	37.4	23.3	122/78	nil	3.34	1.62	1.69	E
149	sivapoongodi	32	5725	G2P1L1	36.5	22.3	120/82	nil	2.43	1.3	2.14	E
150	srimetha	19	5762	primi	37.3	21.2	114/78	nil	2.67	1.67	2.02	E
151	asha	25	5780	G2P1L0	38.3	20.2	110/72	nil	2.3	1.2	2.52	E
152	lalitha	28	5733	G4P1L1A2	37.5	21.2	112/82	nil	2.1	1.4	1.18	E
153	ashina	21	5776	primi	38.5	22.2	112/78	nil	2.34	1.5	2.12	E
154	ebsiba	22	5751	primi	37.4	20.3	114/82	nil	2.31	1.8	2.13	E
155	karpagam	24	5761	G2P1L1	38.4	20.3	120/82	nil	2.21	1.34	1.54	E
156	krishnaveni	23	5732	G3P1L1A1	37.4	20.4	114/82	nil	2.54	1.65	1.34	E
157	maheswari	23	5611	primi	39	21.2	108/78	nil	2.65	1.45	1.67	E
158	priya	32	5588	G2P1L1	37.4	22.3	110/78	nil	2.56	1.34	1.84	E
159	baby	23	5697	primi	37.5	22.2	112/84	nil	2.47	1.67	1.9	E
160	kamatchi	23	5613	G2P1L1	38.5	21.2	108/78	nil	2.86	1.34	2.2	E
161	mohanasundari	24	5538	G2P1L1	39	20.8	110/72	nil	2.3	1.24	2.98	E
162	jabinabanu	24	5775	G2P1L1	36	23.4	114/76	nil	3.12	1.45	2.12	E
163	gayatri	20	5553	primi	36.3	28	112/82	nil	2.34	1.34	2.43	E
164	ponnarasi	23	5749	primi	36.5	24.2	114/78	nil	3.12	1.24	2.76	E
165	revathy	22	5744	primi	36.6	24.4	114/78	nil	2.12	1.54	2.63	E
166	manimegalai	21	5755	G2P1L1	37	23.4	116/76	nil	2.13	0.92	2.43	E
167	hemalatha	19	5533	primi	37.2	21.3	120/78	nil	2.45	1.46	2.99	E
168	nagaammal	23	5779	G2P1L1	37.4	23.5	122/82	nil	2.57	1.35	2.13	E

169	gowri	23	5759	G2P1L1	38	24.3	112/82	trace	2.59	1.46	2.33	E
170	mumtaz	26	5586	G2P1L1	38.3	23.3	120/84	nil	3.21	1.63	2.12	E
171	kalaimathi	26	5786	G2P1L1	38.2	25.3	122/82	nil	2.89	1.72	2.32	E
172	rekha	24	5735	primi	37.3	21.4	122/82	nil	3.24	1.27	1.96	E
173	shanthi	20	5773	primi	29.1	22.5	118/78	nil	3.6	1.64	1.45	E
174	jayapriya	22	5790	G2P1L1	37.6	24.3	120/72	nil	2.89	1.62	1.43	E
175	bharathy	22	5491	G2P1L1	30.2	20.1	114/76	nil	3.24	1.73	2.13	E
176	loganayaki	24	5791	G2P1L1	38.4	20.4	122/82	nil	2.83	1.63	2.45	E
177	gomathy	24	5466	primi	38.3	21.3	120/72	nil	2.3	1.54	2.3	E
178	thenmani	30	5796	G3P1L1A1	33.4	22.3	112/78	nil	2.42	1.52	2.1	E
179	mala	24	5800	G3P2L2	37.3	21.3	114/78	nil	2.15	1.43	1.09	E
180	dhivya	20	5798	G3A2	28.4	24.3	116/82	nil	2.15	1.27	1.98	E
181	jansy	25	5501	G3P1L1A1	30.3	24.5	120/72	nil	3.23	1.43	1.19	E
182	nageswari	23	5802	primi	33.4	24.3	122/74	nil	2.15	1.64	1.8	E
183	mohana	19	5748	primi	38.3	21.3	112/78	nil	1.98	1.56	1.67	E
184	geetha	21	5722	primi	38.5	23.2	120/72	nil	2.03	1.34	2.2	E
185	selvi	22	5811	primi	32	24.3	118/72	nil	2.45	1.21	2.1	E
186	sasikala	21	5370	G2A1	37.2	22.3	120/72	nil	2.54	1.11	2.13	E
187	padmini	24	5813	primi	31.3	21	114/78	nil	3.5	1.23	2.13	E
188	rajeswari	20	5635	primi	37.2	22.1	120/72	nil	2.05	1.24	2.34	E
189	vaishnavi	21	5810	G3A2	36.3	21.4	112/78	nil	2.49	1.22	2.3	E
190	eswari	25	5816	primi	37.3	22.3	120/72	nil	3.23	1.23	2.11	E
191	sandhya	18	5803	primi	38	21.2	112/78	nil	2.21	1.42	2.23	E
192	priya	23	5605	G2P1L1	37.3	23.4	120/72	nil	2.56	1.23	1.23	E
193	lakshmi	23	5821	G3P1L1A1	36.3	23.5	110/72	nil	2.79	1.54	2.67	E
194	gowthami	20	5826	G2P1L1	36.2	22.1	120/78	nil	2.76	1.74	2.13	E
195	teresa	29	5822	G2P1L1	37.2	21.5	120/72	nil	2.4	1.3	2.2	E
196	jayasree	22	5814	primi	37.3	22.7	110/82	nil	1.96	1.73	2.34	E
197	malathy	22	5772	primi	35.3	21.4	112/78	nil	3.23	1.23	2.31	E
198	kasthuri	20	5827	primi	36.4	21	114/78	nil	1.73	1.54	1.21	E
199	asha	22	5531	G2P1L0	37.2	20.3	120/78	nil	2.1	1.26	2.43	E
200	renuka	22	5852	G2P1L1	38.2	21.3	120/74	nil	2.32	2.13	2.34	E

STUDY GROUP (PREECLAMPSIA)

S. NO	Name	Age	IP.NO	obstetric code	Gestational age(wks)	BMI	BP	urine albumin	24 hr urinary protein	severity	FT3	FT4	TSH	Diagnosis
1	lakshmi	30	10582	G3P1L1A1	24	32.1	146/98	1+	340		2.4	1.3	3.27	SCH
2	selvi	23	10686	Primi	34.3	34.2	160/112	3+	5021	severe	1.8	0.9	5.6	SCH
3	kalpana	25	10875	Primi	36.5	24.4	144/98	1+	305		2.1	1.11	2.98	E
4	pramila	22	11659	G2P1L1	36	32.4	150/94	trace	220		2.4	1.02	3.15	SCH
5	chitra	22	11787	Primi	36.4	24.8	148/94	trace	234		2.38	0.9	3.14	SCH
6	sharmila	21	11882	Primi	32	32.3	164/116	1+	453	severe	1.01	0.4	11.4	OH
7	hemavathy	20	12034	Primi	32	26.3	148/98	1+	425		2.1	0.98	4.2	SCH
8	shymala	23	11834	Primi	38	34.4	150/92	1+	357		1.2	0.3	11.3	OH
9	ammu	23	11978	Primi	34.3	34.2	144/94	1+	320		2.73	0.99	1.31	E
10	hasinabegum	34	12142	Primi	32	28.3	146/94	trace	212		2.13	0.8	2.12	E
11	karimabegum'	32	11562	G2P1L1	34.2	35.4	154/94	trace	120		1.94	1.03	1.81	E
12	rekha	25	11452	Primi	36.6	26.4	162/114	2+	3500	severe	1.9	1.26	2.89	E
13	deepa	22	12523	Primi	36	24.3	142/98	1+	142		2.19	1.31	1.9	E
14	hemavathy	22	10240	G3P1L1A1	29.2	34.3	150/92	trace	153		2.2	1.4	2.34	E
15	ameena	20	12344	Primi	38.5	23.3	164/114	2+	2134		2.3	1.21	4.3	SCH
16	mumtaz	27	12184	G3P1L1A1	35	34.3	162/120	3+	5045	severe	2.19	0.9	1.2	E
17	deepa	22	12331	Primi	32	26	146/94	trace	154		2.34	1.04	2.14	E
18	nagammal	21	10867	Primi	31.1	28	164/118	2+	2451	severe	2.13	1.3	5.48	SCH
19	meena	20	10485	Primi	32	30.5	154/92	trace	120		2	1.42	1.13	E
20	porkodi	20	12388	Primi	36.3	32.1	166/114	2+	2472	severe	2.07	1.06	6.23	SCH
21	benazirbegum	24	12462	Primi	38.3	26.4	144/96	trace	142		2.31	1	2.23	E
22	saranya	25	12464	G2P1L1	33	32.3	146/94	1+	352		1.97	1.4	3.53	SCH
23	kalyani	27	12465	G2P1L1	33	33.8	152/92	1+	341		2.2	1.31	2.3	E
24	devi	21	14254	Primi	35	24.2	146/94	trace	127		2.9	1.08	3.78	SCH
25	thulasi	23	12558	G2A1	38.1	35.4	170/112	2+	2200	severe	2.07	1.1	2.42	E
26	devika	32	12557	Primi	32.4	30.1	156/98	2+	2311		2.35	1	3.43	SCH
27	venkatta	20	5625	Primi	37.2	22.1	152/92	nil	98		2.13	1.2	1.23	E
28	latha	32	4949	G2P1L1	36	23.1	154/96	nil	74		2.34	1.43	1.5	E
29	sandhya	21	5808	G4P3L1	37.3	31.1	162/112	1+	564	severe	2.1	1.14	5.24	SCH
30	jayanthi	23	5867	Primi	33.2	34.2	158/92	trace	123		1.83	1.23	1.54	E
31	chellammal	22	5910	Primi	37.1	32.1	164/114	1+	763	severe	1.82	1.01	6.24	SCH
32	meena	22	5870	Primi	36.2	35.4	154/94	nil	102		2.13	1.37	1.2	E
33	rahmathnisha	22	4635	G4P1L1A2	29	34.2	170/124	3+	5421	severe	0.98	0.45	13.2	OH
34	backiyalakshmi	19	5892	Primi	36.2	34.4	154/98	nil	124		2.24	1.45	3.12	SCH
35	noorjahan	21	5958	Primi	32.1	24.1	142/100	1+	312		2.11	1.09	2.4	E
36	ummukulthum	21	5959	Primi	36.1	21.2	154/96	3+	5210		2.34	1.42	2.1	E
37	devi	22	5938	Primi	29.3	35.3	152/92	nil	120		2.09	0.98	1.8	E
38	saranya	24	6033	G3P2L1	35.4	34.3	142/96	nil	112		2.05	1.42	1.65	E
39	sakira	30	6032	G3P2L2	34.2	32.1	152/94	nil	78		2.34	1.45	3.53	SCH
40	roshini	20	6034	Primi	37.3	35.4	146/100	1+	212		2.11	1.17	2.34	E
41	sarala	18	5831	Primi	32.3	22.1	144/98	trace	290		1.96	1.45	2.14	E
42	nandhini	20	5705	Primi	39	31.2	142/94	1+	145		1.2	0.5	11.6	OH
43	kavibharathi	20	6050	Primi	37.1	30.1	142/96	1+	315		2.13	1.28	3.12	SCH
44	sangeetha	30	6081	G2P1L1	29.2	21.56	152/94	trace	124		2.03	1.38	1.82	E
45	ezhilarasi	25	6114	G2P1L1	35.2	34.2	154/98	3+	5201		2.05	1.41	3.21	SCH
46	hemasundari	32	5961	G4A3	33.4	31.2	162/116	2+	2512	severe	1.98	0.96	1.9	E
47	hameedasultana	38	6135	Primi	37.4	20.93	144/92	2+	2422		2.12	1.18	3.23	SCH
48	sathya	23	6185	Primi	37.6	36.4	152/92	trace	120		2.32	1.43	2.1	E
49	nagammal	25	5950	G2P1L1	31.2	34.2	142/98	1+	121		2.32	1.54	2.01	E
50	rathi	32	6227	G2P1L1	35.3	34.2	150/100	1+	352		2.01	1.09	3.21	SCH
51	menaka	30	6196	G2P1L1	36.5	27.2	160/112	1+	768	severe	1.92	1.12	4.62	SCH
52	dhivya	24	6354	Primi	37.2	25.6	172/114	2+	2462	severe	2.1	1.21	5.25	SCH
53	hannis	23	6414	Primi	33	32.1	150/92	nil	94		2.12	1.21	1.43	E
54	shalini	25	6396	Primi	36.1	32.1	154/98	3+	5620	severe	1.2	0.55	12.3	OH
55	suganya	22	6436	Primi	32	23.2	146/96	nil	67		2.1	1.32	2.01	E
56	chitra	21	5483	G2P1L1	36.1	24.3	142/94	trace	163		2.23	0.98	3.78	SCH
57	megala	25	6346	G2P1L1	35.2	34.2	162/112	1+	624	severe	2.1	1.05	2.16	E
58	karthiga	31	6413	Primi	33.2	26.4	164/100	2+	2722	severe	2.11	1.09	5.6	SCH
59	seetha	25	6573	Primi	37.2	24.3	148/98	1+	341		2.13	1.31	2.1	E
60	gomathi	24	6551	Primi	32.5	27.1	148/98	1+	323		2.12	1.02	4.34	SCH

61	kanchana	36	6633	Primi	38.6	34.1	168/98	2+	3564	severe	2.32	1.28	1.7	E
62	menaka	21	6687	Primi	32.4	28	152/92	nil	78		2.03	1.28	1.49	E
63	sumathy	21	6698	Primi	30	28.3	162/110	3+	5467	severe	2.04	1.02	4.58	SCH
64	bazirunisha	22	6741	Primi	37.2	32.2	162/122	3+	5246	severe	2.17	1.05	5.72	SCH
65	karpagam	34	6762	Primi	38.1	28.3	154/98	nil	56		2.21	1.31	2.1	E
66	dilasthbanu	22	6809	Primi	33	26.3	164/116	2+	2521	severe	2.21	1.33	1.8	E
67	vijayalakshmi	20	6860	Primi	39	23.1	154/90	nil	123		2.14	1.16	1.34	E
68	srilekha	23	6819	Primi	37.2	23.2	150/94	1+	382		2.43	1.32	3.23	SCH
69	shenbagm	23	6857	Primi	32.1	26.2	144/90	1+	321		2.01	1.45	2.1	E
70	chandra	24	6919	Primi	34.2	26.1	144/98	1+	120		2.18	1.51	2.12	E
71	kumutha	19	6927	Primi	37.3	28.5	168/122	2+	3422	severe	2.21	1.21	2.13	E
72	gomathy	18	6854	Primi	36.2	26.3	148/94	trace	132		2.32	1.53	1.7	E
73	kalaiselvi	25	6962	Primi	35.6	27.3	164/122	2+	2622	severe	2.21	1.31	1.8	E
74	suganthi	20	6936	Primi	33	26.1	164/110	1+	453	severe	2.14	1.2	6.24	SCH
75	bama	32	7000	G2A1	38.2	29.2	154/116	1+	356		2.45	1.41	1.87	E
76	tamilarasi	24	7019	Primi	32.1	25.1	152/90	1+	320		2.15	1.43	1.67	E
77	nirmala	34	7043	G2P1L1	36.3	34.2	148/92	1+	324		1.92	1.1	3.23	SCH
78	teresa	31	7063	Primi	31.2	28.6	146/92	2+	2500		2.12	1.03	3.12	SCH
79	sakithabanu	18	7118	Primi	29.2	27	170/124	3+	5421	severe	1.87	0.98	4.54	SCH
80	aruna	23	7009	Primi	38.2	24.2	148/94	trace	221		1.97	1.28	2.1	E
81	nandhini	21	7102	Primi	39	29.7	152/82	trace	120		2.1	1.4	1.98	E
82	radha	32	7089	G2P1L1	37.2	28.4	166/112	1+	420	severe	1.81	1.08	6.43	SCH
83	nandhini	22	7155	Primi	29	31.4	162/112	1+	763	severe	1.96	1.16	5.48	SCH
84	devi	21	7202	Primi	36.2	35.2	164/122	2+	2482	severe	1.87	0.97	6.32	SCH
85	varalakshmi	20	7171	Primi	29.4	28.4	150/102	trace	125		2.08	1.2	3.23	SCH
86	aruna	19	7158	Primi	32	28.1	140/92	1+	241		1.98	1.45	1.45	E
87	hemalatha	21	7233	Primi	37.3	34.2	152/98	trace	140		2.01	1.32	3.32	SCH
88	lakshmi	23	7228	G2P1L1	38.2	27.3	172/112	1+	864	severe	1.91	0.98	4.83	SCH
89	anitha	20	7243	Primi	31.3	26.4	140/92	nil	89		2.22	1.19	2.1	E
90	prasanna	20	7326	Primi	38.2	26.7	144/96	nil	68		2.43	1.51	1.7	E
91	selvi	35	7314	Primi	35.6	28.5	140/90	1+	321		2.43	1.12	3.65	SCH
92	savithri	24	7315	Primi	31.2	26.3	142/92	trace	102		2.1	1.43	2.1	E
93	brindha	25	7308	G2P1L1	37.4	28.3	142/90	1+	112		2.16	1.6	1.97	E
94	bhuvaneswari	25	7364	Primi	29	23.1	154/114	1+	387		2.19	1.21	4.22	SCH
95	prema	24	7156	Primi	37.4	24.2	150/92	1+	321		2.06	1.31	2.1	E
96	jayanthi	23	7375	G2P1L0	36.6	24.1	160/92	trace	123		2.1	1.2	3.25	SCH
97	savithri	20	7424	Primi	36.3	32	162/110	2+	2542	severe	1.97	1.21	5.28	SCH
98	abirami	20	7400	Primi	35.4	29.3	152/96	1+	424		2.08	0.99	3.43	SCH
99	keerthika	20	7379	Primi	35.2	28.3	140/92	1+	102		2.41	1.21	1.7	E
100	vaktha	24	7494	Primi	35.2	34.5	144/98	nil	87		2.27	1.42	1.35	E
101	mary	24	7441	G2A1	34.6	26.5	164/124	1+	652	severe	2.02	1.23	6.24	SCH
102	nirmala	29	7517	G2A1	36.2	27.3	142/98	1+	314		2.41	1.3	2.12	E
103	nagamleeswari	22	7674	Primi	32	31.2	164/120	1+	564	severe	2.12	1.25	5.62	SCH
104	jansirani	22	7721	Primi	35.5	28	146/94	1+	342		2.15	1.5	3.23	SCH
105	backiyalakshmi	23	7723	G2P1L1	37.2	28.3	150/96	2+	2421		2.82	1.3	1.4	E
106	roobini	26	7486	G2P1L1	37.4	27.4	154/98	2+	2452		3.21	1.5	4.12	SCH
107	anitha	31	7822	Primi	32.3	28.3	144/92	1+	423		2.41	1.23	1.23	E
108	veni	29	8022	G2P1L1	38.3	27.1	154/100	nil	87		3.12	1.35	3.2	SCH
109	ranjini	21	8109	Primi	33.3	26.3	146/98	1+	321		2.43	1.65	1.3	E
110	dhanalakshmi	29	7945	G3P2L2	34.2	29.3	160/114	1+	673	severe	2.1	1.23	5.24	SCH
111	nuzrathbegum	19	8006	Primi	31.2	26.3	140/98	1+	342		3.12	1.4	1.63	E
112	banupriya	22	8151	Primi	36.1	28.3	144/92	1+	352		2.12	1.6	3.1	SCH
113	shabana	21	8312	G5P2L2A2	29	29	148/94	1+	423		2.3	1.2	2.13	E
114	sermakani	34	8258	G3P2L1	37.3	27.3	144/92	1+	421		1.98	1.2	3.76	SCH
115	swapna	22	8327	G3A2	38.2	26.2	142/98	1+	420		3.01	1.3	1.5	E
116	sasikala	22	8380	G3P2L2	38.1	28.3	144/98	2+	2602		2.1	1.2	3.21	SCH
117	dilsath	22	8379	G3P2L1	39	29.3	150/102	1+	321		3.4	1.5	2.21	E
118	sangeetha	27	8337	Primi	35.3	27.3	152/96	1+	312		3.12	1.43	3.12	SCH
119	jakulin	32	8495	G3A2	37.1	26.4	144/98	1+	102		3.5	1.52	2.14	E
120	riswana	21	8437	Primi	32.1	26.3	142/90	1+	122		2.34	1.3	3.5	SCH
121	geetha	32	3579	G3P2L2	31.2	29.3	146/92	1+	321		2.52	1.02	3.56	SCH
122	devi	22	8528	Primi	34.6	27.4	150/92	1+	364		1.96	1.52	3.62	SCH
123	pravthy	20	8616	Primi	29.3	26.3	142/98	1+	384		2.51	1.03	2.1	E

124	deepa	25	8557	G2P1L0	37	27	144/96	trace	213		2.61	1.1	1.73	E
125	usha	22	8795	Primi	35.3	26	152/98	trace	267		2.67	1.7	4.2	SCH
126	mythili	21	8803	Primi	37.4	27.2	144/94	1+	332		2.34	1.2	1.82	E
127	chitra	22	8849	Primi	29.4	27.3	164/110	1+	624	severe	2.01	1.32	5.41	SCH
128	durgadevi	26	8894	Primi	38.2	28.3	150/94	nil	102		2.31	1.22	3.21	SCH
129	jeevitha	29	9035	Primi	38.1	27.3	142/94	1+	129		1.92	1.2	1.23	E
130	karimunisha	23	9042	Primi	37.2	26.3	148/96	trace	142		1.82	1.43	3.12	SCH
131	velankanni	27	9036	Primi	38.1	28.3	140/96	1+	105		2.13	1.24	3.6	SCH
132	lavanya	21	9153	Primi	29.2	29.3	152/92	trace	183		2.4	1.5	1.25	E
133	menaka	23	9288	G2P1L1	37.1	28.3	142/92	1+	142		2.15	1.6	1.4	E
134	jothilakshmi	20	9018	Primi	32.2	28.1	150/92	1+	104		2.07	1.04	3.17	SCH
135	fathima	24	9317	G3P1L1A1	36.4	27.2	154/94	trace	182		2.15	1.52	3.25	SCH
136	aarthi	27	8875	G3P2L1	37.2	26.4	150/98	2+	2824		2.23	1.72	4.21	SCH
137	priya	23	9486	Primi	29.5	28.4	142/94	trace	210		2.41	1.2	5.97	SCH
138	sakilabanu	27	9493	Primi	38.4	27.3	154/98	2+	2802		1.98	1.2	2.1	E
139	thajunisha	26	9267	G2P1L1	36.3	28.5	144/92	nil	125		2.22	1.03	1.2	E
140	premalatha	31	9505	Primi	34.6	32.1	164/110	1+	464	severe	1.9	1.31	6.74	SCH
141	lakshmi	31	9520	G3P2L1	35.6	34.3	164/114	1+	673	severe	2.08	1.24	7.63	SCH
142	priyanka	20	9630	Primi	35.3	28.3	142/92	1+	263		2.71	1.21	3.4	SCH
143	nagadarshini	32	8155	G2P1L2	36.1	35.3	144/92	1+	272		2.03	1.52	1.8	E
144	radhika	23	9656	Primi	29.3	29.3	154/104	2+	2602		3.04	1.04	4.1	SCH
145	radhudevi	32	9650	Primi	38.2	36.3	152/96	1+	321		1.72	1.09	1.4	E
146	uma	20	9676	Primi	31.3	27.4	150/94	1+	352		2.43	0.92	1.2	E
147	gayatri	27	9605	Primi	37.2	38.5	166/114	2+	2724	severe	2.14	1.05	6.24	SCH
148	fathima	24	9881	Primi	36.3	27.3	144/92	trace	283		2.1	0.98	3.87	SCH
149	manjula	19	9694	Primi	30.3	36.3	148/98	1+	252		2.01	1.2	2.89	E
150	prasanna	24	9931	Primi	38.2	23.4	144/94	1+	283		1.97	1.42	2.1	E
151	ramya	22	9839	G2A1	31.2	25.2	142/94	1+	234		1.82	0.94	2.13	E
152	meena	30	10140	G3P1L1A1	36.2	31.2	142/98	1+	263		2.3	1.32	2.12	E
153	mamtha	26	8941	Primi	32.3	33.4	146/98	1+	302		2.53	1.42	1.63	E
154	sumithra	22	10036	G2P1L1	34.2	32.1	146/94	trace	289		2.14	1.03	3.42	SCH
155	archana	31	10132	G2P1L1	37.4	34.2	142/98	1+	329		2.04	1.04	2.12	E
156	hemavathy	23	9621	Primi	38.4	36.2	152/92	1+	342		2.41	1.21	3.12	SCH
157	shabana	22	10204	Primi	29.7	31.2	146/92	1+	283		2.03	1.31	2.12	E
158	venmathy	32	8937	Primi	34.2	37.2	152/98	trace	293		2.83	1.05	2.12	E
159	gowri	34	10307	Primi	35.4	21.4	152/98	1+	342		1.87	0.95	4.52	SCH
160	subashini	21	10240	G3P1L1A1	39	22	152/92	trace	282		3.21	0.94	1.42	E
161	stella	21	10380	Primi	34.5	32.2	156/98	trace	243		2.32	1.23	1.62	E
162	gnanaselvi	33	10385	G2P1L1	36.2	26.4	148/92	1+	342		2.1	0.98	4.3	SCH
163	manjula	21	10416	G2P1L1	35.6	24.1	142/94	1+	271		2.63	1.52	2.12	E
164	vanitha	20	10389	Primi	30.1	31.2	152/96	1+	361		1.92	1.34	3.14	SCH
165	nancy	20	10413	G3A2	37.2	32.1	146/92	trace	213		1.95	1.52	1.23	E
166	anbina	30	10412	G3P1L1A1	29.6	36.3	148/100	1+	321		1.98	1.62	3.21	SCH
167	geetha	25	10422	Primi	38.2	23.1	142/98	trace	126		2.03	1.05	1.23	E
168	durgadevi	23	10407	Primi	36.3	32.4	142/98	1+	128		2.08	1.36	1.42	E
169	gracy	30	10142	Primi	36.2	24.2	152/98	1+	320		2.18	1.52	3.42	SCH
170	ranjini	21	10362	Primi	30.3	34.5	170/110	2+	3524	severe	2.06	1.15	4.01	SCH
171	puspha	31	10400	G2P1L1	38.2	33.4	152/94	trace	218		2.42	1.29	1.5	E
172	yuvarani	21	10395	G2P1L1	28.6	23.1	152/94	1+	214		2.21	1.62	2.12	E
173	veena	27	10399	Primi	37.1	35.4	148/100	1+	283		2.43	1.29	2.41	E
174	nadhiya	26	10405	Primi	32.2	21.4	142/100	1+	246		2.73	1.02	3.56	SCH
175	kanchana	26	10372	G3P2L2	36.3	36.4	154/98	trace	282		1.96	1.62	2.12	E
176	anitha	22	10280	G3P1L1A1	34.6	33.2	152/98	1+	302		2.03	0.98	1.82	E
177	narmadha	23	10365	G2P1L1	31.5	21.5	142/100	1+	320		2.06	1.62	1.23	E
178	jarina	24	10278	Primi	34.5	32.2	164/112	2+	2523	severe	2.51	1.42	4.92	SCH
179	sridevi	23	10387	Primi	33.1	21.3	148/98	1+	324		2.42	1.25	3.42	SCH
180	murugeswari	22	10355	Primi	36.2	32.3	152/92	trace	242		2.02	1.62	3.26	SCH
181	prabavathy	26	10316	Primi	35.2	24.2	142/98	1+	320		2.43	1.32	3.41	SCH
182	anish	21	10339	Primi	36.3	34.2	152/98	trace	212		2.83	1.52	2.43	E
183	rasheeda	26	10269	Primi	32.5	32.3	142/100	1+	242		213	1.26	1.43	E
184	muthu	27	10338	Primi	28.4	34.3	162/114	2+	3569	severe	1.92	1.32	4.82	SCH
185	navamani	24	10220	G3P2L1	38.4	24.3	142/98	1+	242		2.05	1.3	2.1	E
186	nayaki	24	9268	Primi	37.2	33.4	170/112	2+	2622	severe	1.95	1.21	6.02	SCH

187	chitra	22	10369	Primi	34.6	23.4	152/98	trace	273		2.41	1.62	2.23	E
188	sudha	23	10090	G2P1L1	31.5	25.3	142/98	1+	362		2.15	1.52	2.11	E
189	anandhi	23	10143	G2P1L1	36.4	24.2	152/94	1+	284		2.41	1.26	1.42	E
190	navtha	22	9920	G3P1L1A1	37.2	33.2	164/120	3+	5208	severe	2.41	1.26	5.62	SCH
191	vijayakumari	24	10288	Primi	29.3	34.3	142/96	2+	2622		2.31	1.24	1.42	E
192	kokila	25	10282	Primi	36.3	23.1	152/102	1+	382		2.51	1.52	1.32	E
193	sudha	24	10318	Primi	37.4	35.4	142/94	trace	240		2.51	1.25	2.1	E
194	gowri	25	10306	Primi	38.4	32.2	142/96	1+	1424		2.16	1.52	1.42	E
195	nishanthi	23	10300	Primi	38.4	35.6	144/94	1+	320		3.04	1.38	2.9	E
196	saidhani	25	10229	Primi	36.2	24.3	152/94	trace	128		2.4	1.19	2.33	E
197	priyanka	20	1034	Primi	35.2	24.4	148/102	trace	268		3.21	1.26	1.02	E
198	karpagam	20	10188	Primi	34.2	35.4	152/94	trace	204		1.94	1.52	1.94	E
199	saridha	28	10284	G3A2	33.5	23.4	152/92	1+	242		2.52	1.62	2.14	E
200	yasodha	20	10723	Primi	37.3	37.4	142/92	1+	273		2.31	1.23	2.22	E