COMPARISON OF OBSTETRIC AND PERINATAL OUTCOME IN PREGNANT WOMEN WITH HYPOTHYROIDISM VERSUS THOSE WITHOUT: A PROSPECTIVE COHORT STUDY.

A DISSERTATION SUBMITTED TO THE TAMIL NADU DR.M.G.R. UNIVERSITY, CHENNAI, IN PARTIAL FULFILMENT OF THE RULES AND REGULATIONS FOR THE DEGREE M.S. (BRANCH II) IN OBSTETRICS AND GYNAECOLOGY, TO BE HELD IN APRIL 2018.

DECLARATION

I hereby declare that this dissertation titled "**Comparison of Obstetric & Perinatal outcomes in pregnant women with hypothyroidism versus those without: a prospective cohort study**" is carried out by me under the guidance and supervision of Dr. Manisha Beck (MD), Professor of Obstetrics and Gynaecology Unit-IV, Christian Medical College, Vellore. This dissertation is submitted in partial fulfilment of the requirements for the degree of M.S in Obstetrics and Gynaecology examination of the Tamil Nadu Dr. M.G.R. Medical University to be held in April 2018.

Vellore Dr. Rama Smita

Date:

CERTIFICATE

This is to certify that "**Comparison of Obstetric & Perinatal outcomes in pregnant women with hypothyroidism versus those without: a prospective cohort study**" is a bonafide work of Dr. Rama Smita that was done under my guidance and supervision towards the partial fulfilment of the requirement for the M.S. (Obstetrics and Gynaecology) examination of the Tamil Nadu Dr M.G.R. Medical University, to be held in April 2018.

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CONTENTS

INTRODUCTION

For every woman in the world it is a joy to be pregnant and successfully carry their baby. A normal functioning thyroid is very essential for this, that helps the woman in their journey through pregnancy. For a healthcare personnel it is the joy of satisfaction in delivering a healthy mother and baby at the end of gestation. But unfortunately, if thyroid dysfunctions especially hypothyroidism are overlooked during early pregnancy, they can have adverse outcomes for both mother and foetus.

In a woman during pregnant state, normally there are different physiological alteration of the pituitary-thyroid axis with an increase iodine metabolism.(1) These alteration lead to increase formation of thyroxine-binding globulin (TBG) and increase in total thyroid- hormone level. There is also increase in the level of thyroid stimulating hormone (TSH).

In pregnancy, there is increased demand for the maternal thyroid hormones by the developing in utero foetus.(2) The early developing foetus, cannot produce its own thyroid hormones till about 12 weeks of gestation life. These maternal thyroid hormones are critically necessary during first trimester period for the essential normal neurodevelopment of the foetus. These requirements in pregnancy lead to increased demand on the maternal thyroid. It can lead to goitrogenic effect on the thyroid gland in some individual and cause thyroid dysfunction i.e. Hypothyroidism.

In the event of such thyroid dysfunction during pregnancy, the earlier the maternal thyroxine level is corrected the better is the outcome for the foetus.

In an individual, sometimes the non-specific changes of hypothyroidism may be overlooked in non-pregnant state, which can lead to under diagnosis during pregnancy. In such cases, the untreated hypothyroidism can have adverse maternal outcome with significant perinatal morbidity. Some studies (3–5) have also shown that lack of thyroid hormone during 1st trimester foetal neuro development is associated with lower intelligence quotient (IQ) and poor cognition of the off springs. Thus, clinical hypothyroidism directly or indirectly, also through its association with other comorbidities affects both mother and foetus.

Therefore, it is necessary that hypothyroidism should be detected early in pregnancy and promptly initiated with treatment. If a pregnant woman is already a known case of hypothyroidism, then proper titration of the thyroid medications in early pregnancy is very important.

Our study was designed to provide an opportunity to evaluate the outcome of pregnancy in hypothyroid mothers and compare it with those without hypothyroidism in the population catered by our Institution. In this study we would also study the various perspectives about the screening for hypothyroidism in pregnancy.

Aim and Objective of the Study:

Aim:

To compare the Obstetric and Perinatal outcomes in pregnant women with hypothyroidism versus those without in a tertiary care hospital in South India.

Objectives of study:

(1) To see if there is any difference in the incidence of adverse maternal and neonatal outcomes in pregnant women with hypothyroidism as compared to those without.

(2) To determine whether routine screening for thyroid disorder should be offered to all pregnant women.

REVIEW OF LITERATURE

Thyroid is one of the important endocrine gland of our body. It secretes triiodothyronine (T3), thyroxine (T4) and a calcium regulating hormone, the calcitonin. These hormones play very important role in maintaining the normal body metabolism and calcium homeostasis for the normal psychosexual development. (6)

Thyroid dysfunction especially hypothyroidism is the most common endocrine disorder in the population of the world. It is also a major concern for women during pregnancy as such thyroid disorder are related with adverse maternal and perinatal outcomes.(7) (8) (9)

The literature describes that most common cause of overt hypothyroidism (OH) in pregnancy is chronic autoimmune thyroiditis. It is also known as **Hashimoto's thyroiditis**. Other causes leading to hypothyroidism are endemic iodine deficiency (in certain geographical areas), and in women who had prior therapy with radioactive iodine.(10,11)

Hyperthyroidism is seen very rarely. It is estimated that in about only 0.1-0.4% of pregnant women have this thyroid dysfunction.(12)

During the pregnancy, a woman's body undergoes different varied physiological and hormonal changes. Such changes lead to the high thyroid hormone demand from the thyroid gland. If the gland is unable to replenish such growing need it can lead to hypothyroidism.

The clinical signs of hypothyroidism may be overlooked in pregnancy as its clinical signs and symptoms can be masked by the hypermetabolic state of pregnancy.(12) (13) In these cases, if hypothyroidism is not detected by the clinician and treated, later it can have adverse effects on both maternal and foetal outcomes.(10) (9)

The current scientific data suggests that mothers with untreated maternal hypothyroidism during the state of pregnancy, are associated with various adverse outcomes. The studies show these mothers can have miscarriages, recurrent pregnancy loss (RPL), abruptio placentae, pre-eclampsia, gestational diabetes (GDM), postpartum haemorrhage (PPH), increase chance of caesarean sections (LSCS), congestive cardiac failure (CCF) and rarely have myopathy in mothers.(10) (14–16)

In some recent studies, they have shown that untreated hypothyroidism in the mother can also affect the perinatal outcome. It could lead to preterm deliveries, intrauterine growth restriction (IUGR), intrauterine foetal death (IUFD), respiratory distress and

high perinatal mortality rates. It is also associated with new-borns who have poor cognitive functions, neurological and developmental impairment.(10,17)

Prevalence in pregnancy:

The prevalence of thyroid disorder in women during pregnancy as quoted in western literature is about 2.5 %. In this about 0.3-0.5% of women have overt hypothyroidism **(OH)** and about 2-2.5% of pregnant women are subclinical hypothyroidism **(SCH).** (10,18) (19)

The prevalence data as per our Indian literature, in comparison showed that the prevalence of hypothyroidism among the pregnant women in India was about 4.8-11%.(2) (20)

A large study by Sahu et al 2010 have reported prevalence rate of hypothyroidism in pregnant women was 6.47%. Out of which 4.58% cases had overt hypothyroidism.(21)

Another study by Ajmani et al, (2014) from India, showed that prevalence rate of hypothyroidism among the Indian pregnant women was 12%. In these hypothyroid women, 3% were overt hypothyroid (OH) & 9% were sub clinical hypothyroid (SCH). The thyroid peroxidase antibodies (TPO) positivity was seen in 50% of the study pregnant women with subclinical hypothyroidism and 7% of the euthyroid pregnant women.(22)

Anatomy of the thyroid gland

The thyroid gland is a brownish-red coloured endocrine gland found in front of the neck over the trachea. It spans across from the level of 5th cervical vertebrae to 1st thoracic vertebrae.

The gland is butterfly in shape and consist of two lateral lobes with pair of superior and inferior poles. The two-lateral thyroid lobes are about 5-6 cm long and are connected by isthmus of height about 12-15 mm. The intervening isthmus overlies traches from second to fourth tracheal rings. (Figure 1) (23,24)

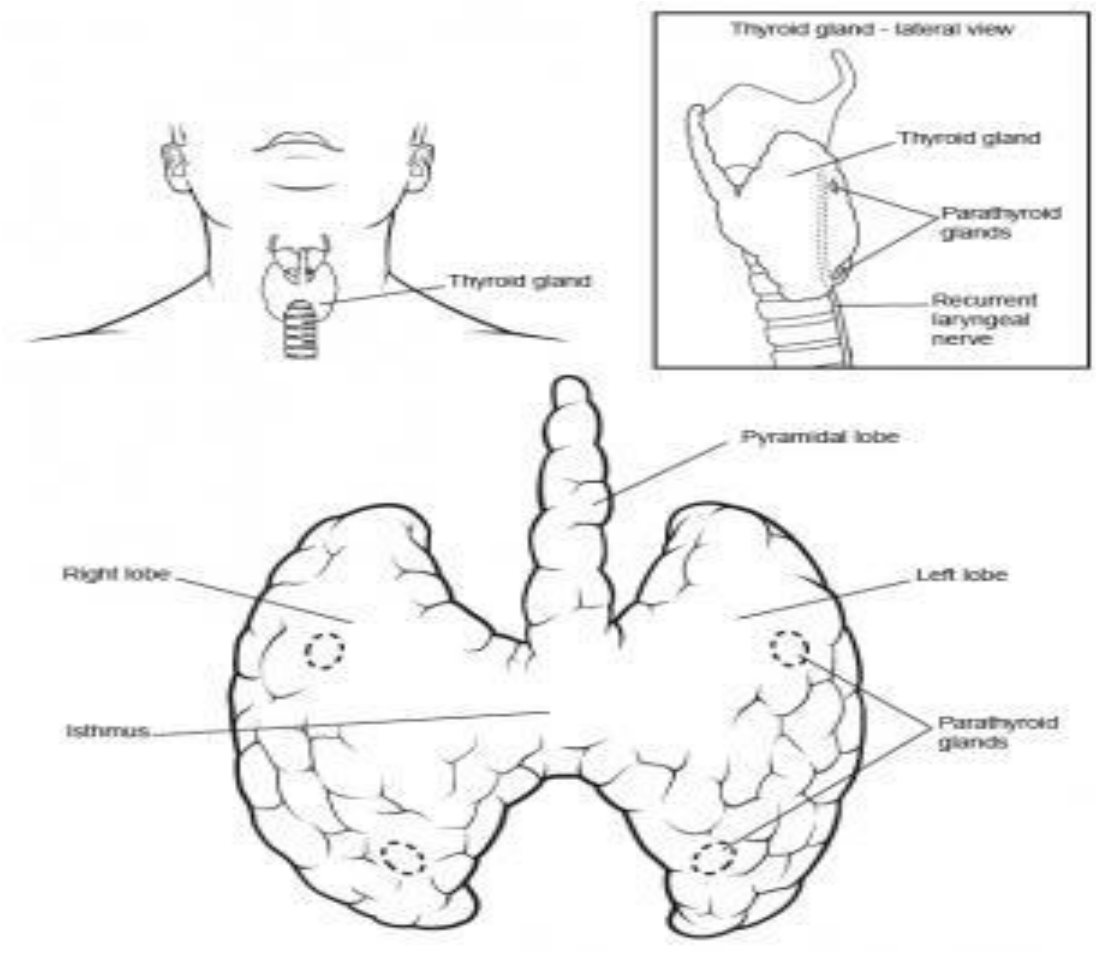


Figure 1 The thyroid gland anatomy

The thyroid gland weighs about average 25-30 g in adults and can be slightly more heavier in women.(25)

The thyroid gland in women usually enlarges in size during menstruation and pregnancy.

The gland has a conical lobe which arises from the isthmus or in some cases from the adjacent lobe (more often from left lobe) toward the hyoid bone. This lobe is called as the Pyramidal lobe. In some individuals, it may be attached by a fibrous or fibromuscular band to the hyoid bone.

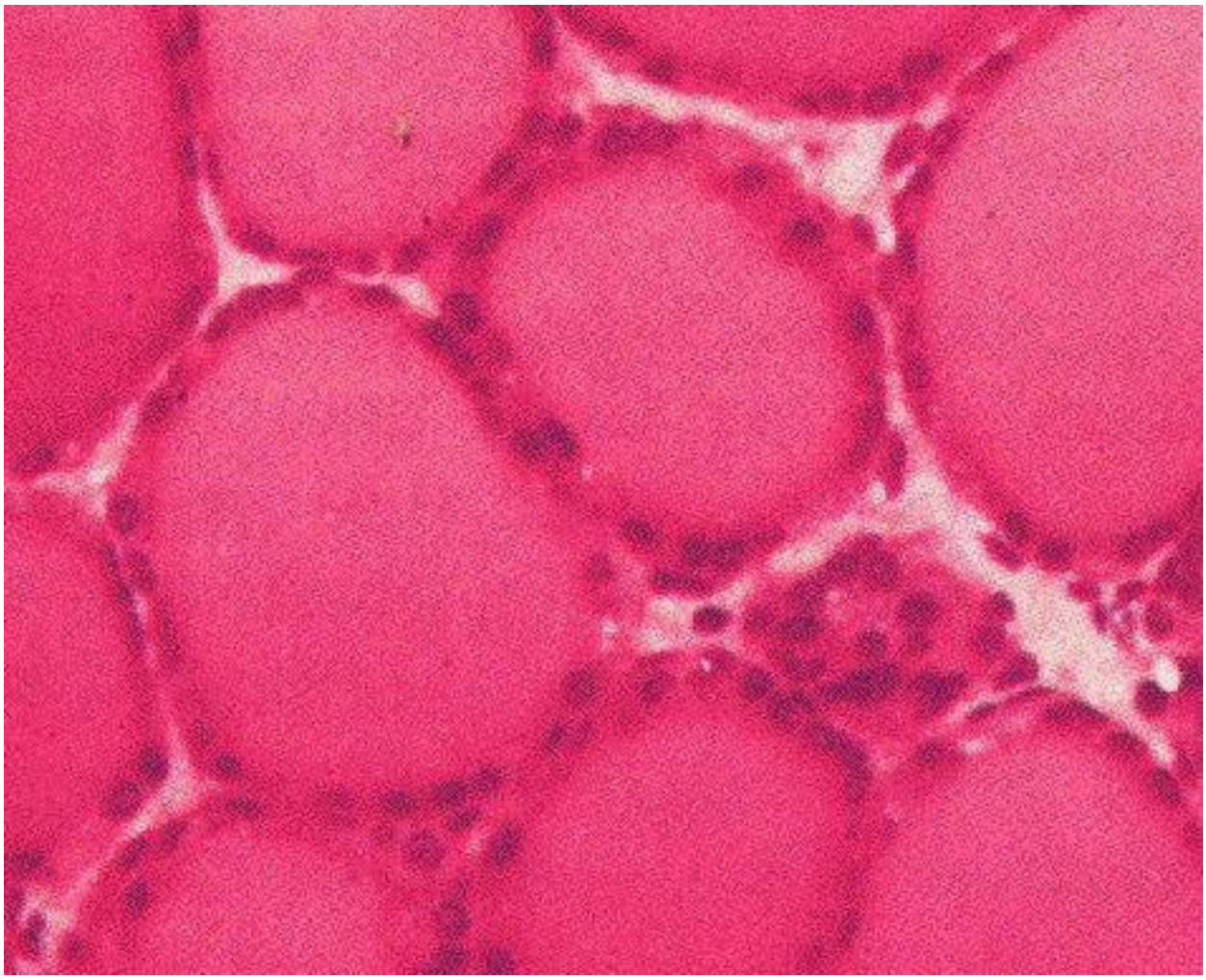
In few instances there may be the remnants of the embryological thyroglossal duct. It can persist either as an accessory nodules or cysts within the thyroid tissue, between the isthmus and foramen caecum of the base of tongue. There are also two pairs of parathyroid glands lying in close proximity of the gland tissue.

Histological structure of thyroid gland

The thyroid gland lies in between the middle layer of deep cervical fascia. It is enclosed by a true inner capsule, which is very thin and closely adheres on the outer gland surface. This capsule sends many extensions within the substance of the gland and forming numerous septae. These septae thus divide the gland into lobes and lobules.

These lobules are made up of many follicles which forms the main structural units of the gland. These thyroid follicles are made up of a layer of simple epithelium which encloses a colloid-filled cavity.

On viewing through microscope this colloid appears homogenous pink in colour on Haematoxylin and Eosin [H&E] stains. (Slide 1)



Slide 1: H&E stain slide of thyroid showing colloid filled normal follicles of gland.

The central colloid which contains the main iodinated glycoprotein, known as iodo thyroglobulin. This glycoprotein is the principal precursor of the later formed active thyroid hormones (T3 &T4)

These follicles may vary in size inside the gland. When the gland is stimulated, these follicular cells turn columnar and lumen gets depleted of colloid.

When the thyroid gland function is suppressed, the follicular cells turn flat with colloid accumulation in the lumen. These follicles are also surrounded by network of dense plexuses made up of fenestrated capillaries, lymphatics and sympathetic nerves.

There are also Parafollicular cells, which lie adjacent to the follicles within the basal lamina.

The epithelial cells of thyroid gland, are made up of two types of cells i.e.(i)

principal cells (or the follicular cells) and (ii) parafollicular cells (or the C cells).

The principal cells have main function is to form colloid (iodothyroglobulin), whereas the parafollicular cells form calcitonin, the main hormone central to the calcium homeostasis in our body.

Biosynthesis of Thyroid hormone in thyroid

The thyroid gland is responsible to produce 2 biologically active hormones, which are Thyroxine (T4) and 3,5,3'-triiodothyronine (T3). The two hormones fundamentally differ in their structure. The Thyroxine hormone has 2 Iodine atoms on its phenyl (outer) ring but T3 hormone has only 1 Iodine atom.

Thyroxine is solely produced by the thyroid gland, whereas T3 is produce not only in thyroid but also in many other peripheral tissues by the process of deiodination of Thyroxine.

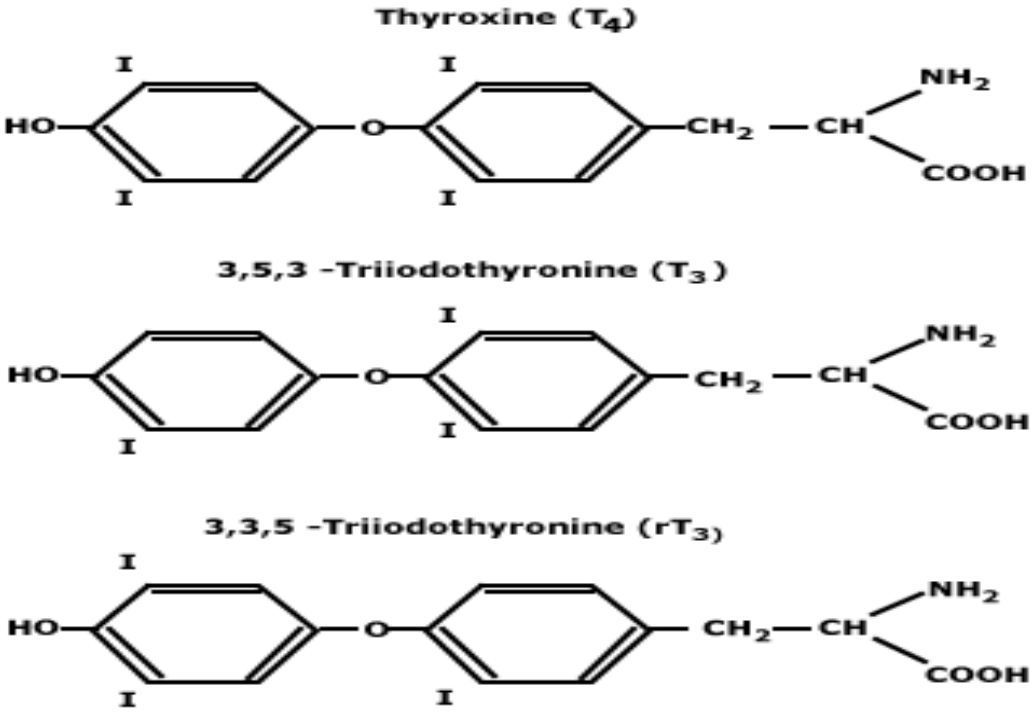


Figure:2 Various thyroid hormone structure.

The thyroid hormone synthesis occurs in the thyroid gland by following steps:(26) (as shown below in Figure 3)

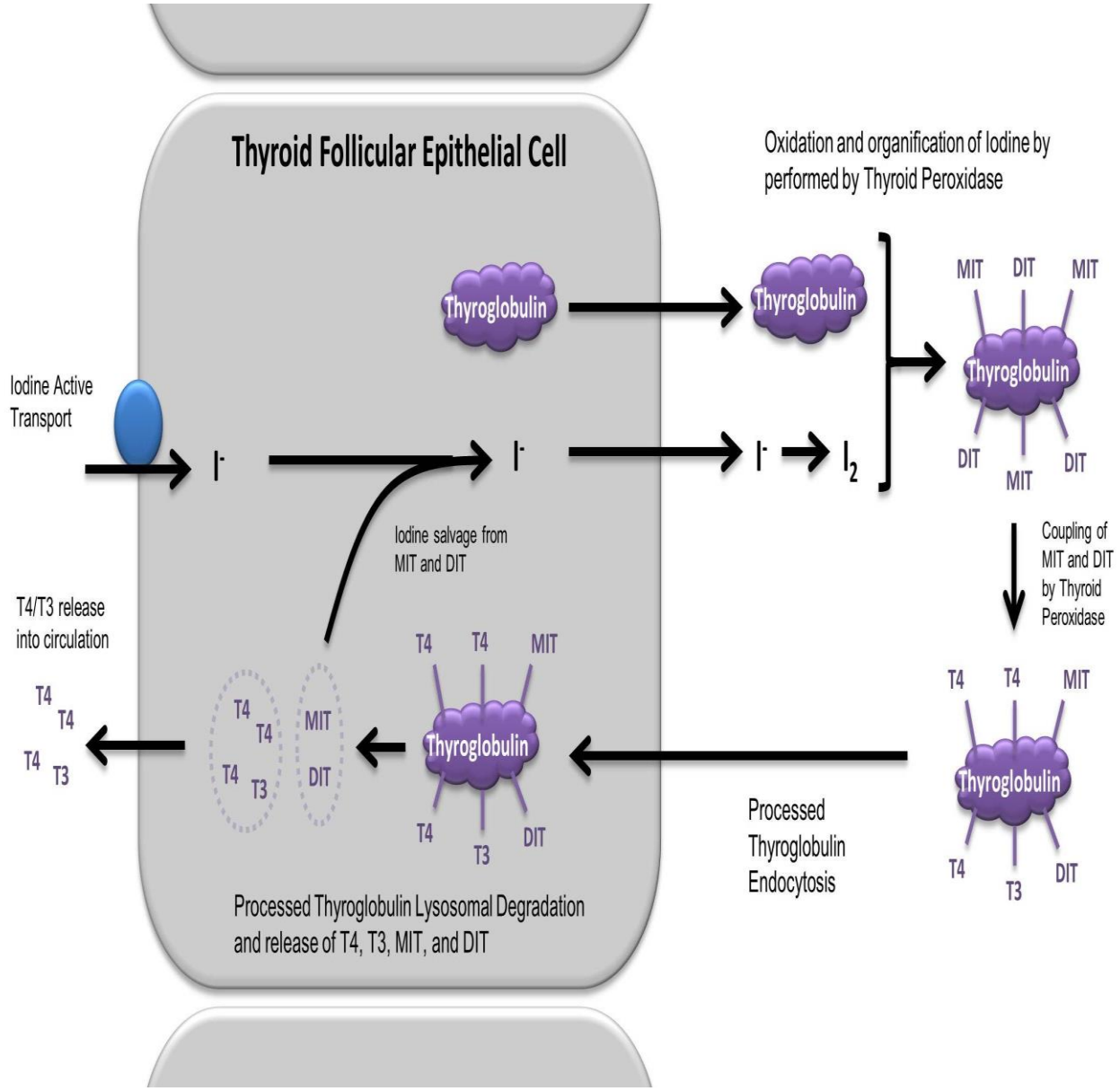


Figure 3: Thyroid Hormone Synthesis Mechanism

(a) Dietary Iodide transport to thyroid:(27)

The iodine present in our diet is absorbed in iodide or the ionic form. It is rapidly absorbed and distributed in the body's extracellular fluid (ECF) compartment. The biosynthesis of thyroid hormones requires large amount of Iodine.

Iodide from the ECF is actively transported into thyroid follicular cells against a chemical & electrical gradient by the sodium iodine transporter. It is a transmembrane protein situated on the membrane of thyroid follicular cells. This process leads to the relative high concentration of Iodide presence in the thyroid gland as compared to the rest of the body.

(b) **Thyroglobulin Synthesis in the follicle:**

Thyroglobulin is a 660 kilodalton (kd) glycoprotein protein made up of many amino acids as tyrosine. It is biosynthesized and then glycosylated in the rough endoplasmic reticulum (RER) of the follicular epithelial cell.

In the follicle cells, tyrosine residues are iodinated and coupled to form T4 and T3 and released into the follicular lumen. The thyroglobulin protein is made up of 6 molecules of mono iodotyrosine (MIT), 4 molecules of di iodotyrosine (DIT), 2 of T4 & 0.2 of T3 per molecule.

(c) **Tyrosyl iodination:**(27)

Thyroid peroxidase is a very important enzyme present in the colloid of the follicular cell lumen. It helps to forms I2 by oxidizing I- ions and then "organifies" the generated I2 by covalently linking it with tyrosine residues of the Thyroglobulin.

This thus leads to formation of either single or double-iodinated forms of tyrosine amino acid known as "Monoiodotyrosine (MIT)" and "Diiodotyrosine (DIT)".

Coupling of iodotyrosyl residues of thyroglobulin

The enzyme thyroid peroxidase helps in coupling of these MIT and DIT residues to generate T4 or T3 hormone within the thyroglobulin. Thyroxine hormone is thus formed by combination of two DIT residues while T3 is formed by combination of one DIT with one MIT residue.

Thyroid peroxidase can combine 2 DIT residues and form T4 more readily, hence thyroid gland is site for primarily producing T4 rather than T3.

This enzyme also helps catalyse iodination of 10% of tyrosine residues of thyroglobulin

(d) **Endocytosis of Peroxidase-processed Thyroglobulin and hormone release**:

The peroxidase-processed thyroglobulin is endocytosed by follicular epithelial cells which fuses with lysosomes to form phagolysosomes. In these phagolysosomes the thyroglobulin is hydrolysed to T4, T3, and its other amino acids.

Whenever the thyroid gland is stimulated these formed thyroid hormones are released into the circulation.

This peroxidase-processed thyroglobulin within the follicle act as reservoir for thyroid hormones. This is the reason why the defects of thyroid hormone often take months to become clinically apparent after gland dysfunction.

(e) **Recycle of iodide:**

The iodotyrosines which are released from the thyroglobulin are deiodinated by the help of iodotyrosine deiodinase enzyme. Thus, most of the iodide is recycled for thyroid hormone synthesis again.

Extrathyroidal T3 hormone production

Approximately 80% of T3 hormone is produced at the extrathyroidal tissue. The process occurs by 5'-deiodination (outer-ring deiodination) of Thyroxine

This reaction takes place with the help of two, plasma membrane and microsomal enzymes i.e. T4-5'-deiodinases (type I) and T4-5'-deiodinases (type II). These enzymes are present in abundant amount in liver and kidney. These organs are the major sources of T3 hormone.

Both enzymes T4-5'-deiodinase (types I and II) are very different in location, biochemical properties and functioning to physiologic stimuli

●Enzyme Type I T4-5'-deiodinase: It is the main deiodinising enzyme present in the liver, kidney, and thyroid. It is also Propylthiouracil(PTU) sensitive. The 35% of the extra thyroidally produced T3 is contributed by this enzyme.

●Enzyme Type II T4-5'-deiodinase: It is the main deiodinising enzyme present in muscle, brain, pituitary, skin, and placenta. It is not inhibited by PTU. The 65% of the extra thyroidally produced T3 is contributed by this enzyme.

The proportion varies in thyroid dysfunction. Type II enzyme contributes higher in hypothyroidism and lower in hyperthyroidism.

Regulation of thyroid hormone: (as shown in Figure 4)

The Thyroid hormone level in body is regulated by 2 pathways as follows:

1) The thyroidal hormone biosynthesis with secretion of thyroxine and triiodothyronine (T3) is regulated by thyroid-stimulating hormone (TSH). The

secretion of TSH is inhibited by high T4 & T3 and stimulated by thyrotropin-releasing hormone (TRH).

2) The extrathyroidal conversion of Thyroxine to T3 is usually regulated by diet, hormonal and disease-related factors.

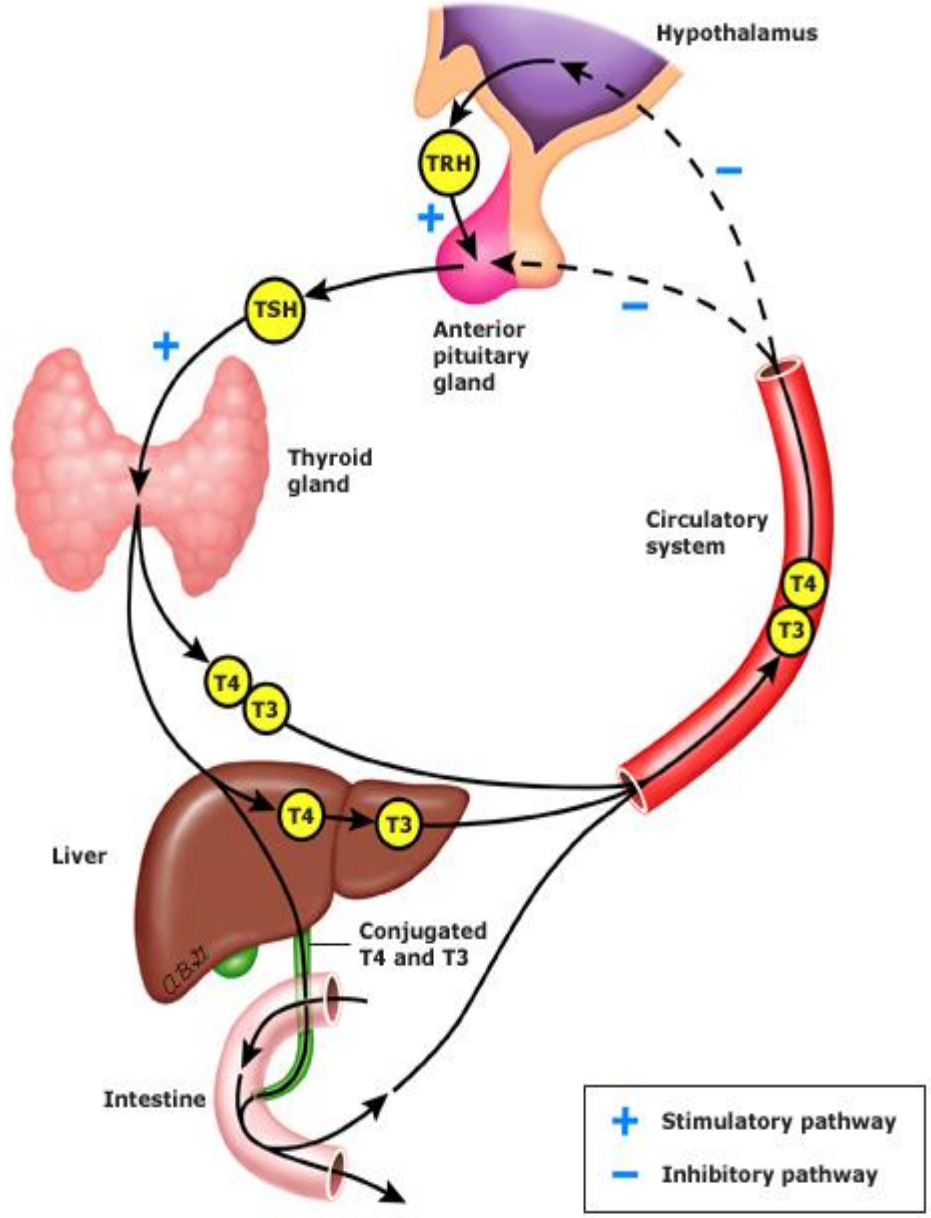


Figure: 4 Regulation of Thyroid hormones

The thyrotropin releasing hormone (TRH) is secreted by Hypothalamus, whose stimulation leads to the secretion of thyroid stimulating hormone (TSH). TRH is a tripeptide made up of pyroglutamyl-histidyl-prolineamide found mainly in the median eminence and paraventricular nuclei of hypothalamus. It is responsible for the TSH secretion via receptor-mediated activation of PCP pathway.

TSH is made and secreted by the thyrotropes present in the anterior pituitary gland. It is secreted in the blood and reaches its target organ i.e. thyroid. In thyroid gland it stimulates the production and secretion of T3 and Thyroxine hormone. TSH biochemically is a glycoprotein made up of alpha and beta subunits containing about 15% carbohydrate.

The normal TSH physiological secretion is pulsatile and its secretion is 50 to 100 percent higher in the late evening than the daytime. The normal TSH secretion rate in individual ranges from 75 to 150 mU/day.

The high level of T3 and Thyroxine causes feedback inhibition of TSH, both directly and indirectly by suppressing the release of hypothalamic TRH. The inhibition of TSH synthesis occurs by inhibition of transcription of TSH subunit genes.

Thyroxine is usually converted to T3 in liver and many other extra thyroid tissues by the action of T4 monodeiodinases.

Some Thyroxine and T3 is conjugated with glucuronide and sulfate in the liver, excreted in the bile and some hydrolyzed in the intestine and excreted. The T4 and T3 in the intestine may be reabsorbed in small amounts. (Figure 4)

Mechanism of action of TSH on the thyroid gland

TSH stimulation is very essential in thyroid hormone biosynthesis and secretion by the thyroid gland. Its stimulation leads to expression of genes in thyroid tissue causing thyroid enlargement.

The specific actions of TSH occurs by binding to its specific trans membrane receptors. This activates the enzyme adenylyl cyclase which increases the cyclic AMP formation in the cell. This process causes the activation of several protein kinases. TSH also stimulates the phospholipase C enzyme activity thus increasing phosphoinositide turnover, increasing intracellular calcium ion concentration and also the protein kinase C activity.

The mechanism how these changes are link to specific steps of hormone formation is not known.

Exogenous agents altering normal TSH secretion

These agents are glucocorticoids, somatostatin infusion and dopamine infusion.

- Glucocorticoids cause decrease in the pulsatile TSH secretion by inhibiting the TRH secretion.
- Somatostatin infusion or its analog Octreotide reduce serum TSH concentrations.

- Dopamine infusion causes a rapid decrease in serum TSH concentrations in the body at doses of 1 mcg/kg per min and more. That is why the serum TSH concentrations of intensive care unit patients on dopamine have low TSH.

  Alternatively.it is also seen that TSH levels rise after the administration of dopamine antagonists such as metoclopramide. These agents act directly on the pituitary gland to cause inhibition. Dopamine is also a physiologically important agent causing inhibition of TSH secretion in body.

The inhibitory effects of these exogenous agents are clinically small.

While high secretion of endogenous dopamine, somatostatin, or glucocorticoid may transiently decrease TSH levels. Their sustained increases in production do not cause reduction of TSH or hypothyroidism.

Physiological changes in thyroid metabolism in pregnancy

In a pregnant woman there is an increased metabolic demand in body and also from the developing foetus. This changes lead to significant changes in the thyroid physiology.(28) (12) It is influenced by two important maternal hormones which are human chorionic gonadotrophin (HCG) and oestrogen hormone. (13,28)

To meet this increase in demand during pregnancy, thyroid gland function increases by 10% but in areas of iodine deficiency its function can double i.e. increase by about 20-40%.(29) This causes thyroid gland to increase in size by 15% during pregnancy which later returns to normal after delivery.(11)

The principal normal variations of thyroid gland function during pregnancy are:(11,12)

(a) **Increase in level of serum thyroxine-binding globulin (TBG)** –

In early phase of first trimester pregnancy the serum TBG levels double i.e. increases to almost two times under the influence of circulating maternal **estrogen** hormone. Increase estrogen causes TBG production and TBG sialylation in the body.

This leads to reduced hepatic clearance of TBG from the blood.

This excess TBG level causes increase of extra thyroidal pool of serum total T4 and T3 level.

So, to maintain the adequate free thyroid hormone circulation, thyroxine (T4) and triiodothyronine (T3) production by the thyroid gland is increased.

Thus, there is an increase in levels of total T4 and T3 by about 50% during the first half of pregnancy and it plateaus at approximately 20 weeks of gestational age.

(b) **Stimulation of thyroid-stimulating hormone [TSH]) receptor by human chorionic gonadotropin (hCG**) –

Human chorionic gonadotropin (hCG) and TSH hormone belong to the same family of glycoprotein hormones and have common alpha subunit with a unique beta subunit. Hence, hCG hormone tend to have a weak TSH like activity in the body. In laboratory study by human thyroid cell culture assay, it was found that about 1 microU of hCG was equivalent to about 0.0013 microU of TSH.

Normally the serum hCG concentrations increase soon after fertilization and peak at 10 to 12 weeks which also causes the increases in total serum T4 and T3 concentrations.

This physiological subclinical hyperthyroidism is transient. Later as the pregnancy progresses the hCG secretion declines and serum free T4 and T3 levels also decline and consequently serum TSH level rises slightly.

Effect of pregnancy on urinary Iodine excretion from body:(13)

In addition to above physiological changes in thyroid gland during pregnancy there is also an increase in urinary iodine excretion. This phenomenon occurs due to high glomerular filtration rate during pregnancy as well as due to increased plasma clearance of iodine. This further leads to decrease in iodine stores of the body. However, in women who have borderline or deficient iodine reserve, there is fall in

circulating thyroid hormone level. This causes hypothyroidism and leading to increase in thyroid stimulating hormone.

Trimester specific TSH reference ranges- (10)

As mentioned, the thyroid gland undergoes important physiological adaptation during pregnancy with changes in size and function. The TSH level during pregnancy is lower as compared to the non-pregnant state. Hence, it is recommended to use population based trimester specific reference ranges as follows.

First trimester TSH range: 0.1 – 2.5mIU/ml

Second trimester TSH range: 0.2 – 3.0mIU/ml

Third trimester TSH range: 0.3 – 3.0mIU/ml.

Definitions:

Primary maternal hypothyroidism is a clinical condition defined as the presence of elevated Thyroid Stimulating Hormone (TSH) levels as per trimester during pregnancy.

Primary hypothyroidism is of two types either Overt Hypothyroidism (OH) or Subclinical Hypothyroidism (SCH) in pregnant women.

Subclinical hypothyroidism is diagnosed clinically in a pregnant women when serum TSH level is between 2.5-10 mIU/L (in first trimester) and between 3-10 mIU/l (in second/third trimester) with normal T4 levels. (10)

Overt hypothyroidism is diagnosed in a pregnant women when the serum TSH level is >2.5millil U/l in first trimester (>3.0 mIU/l in second/third trimester) with raised T4 levels and/or Serum TSH >10mIU/L irrespective of T4 level. (10)

Autoimmune thyroid disease

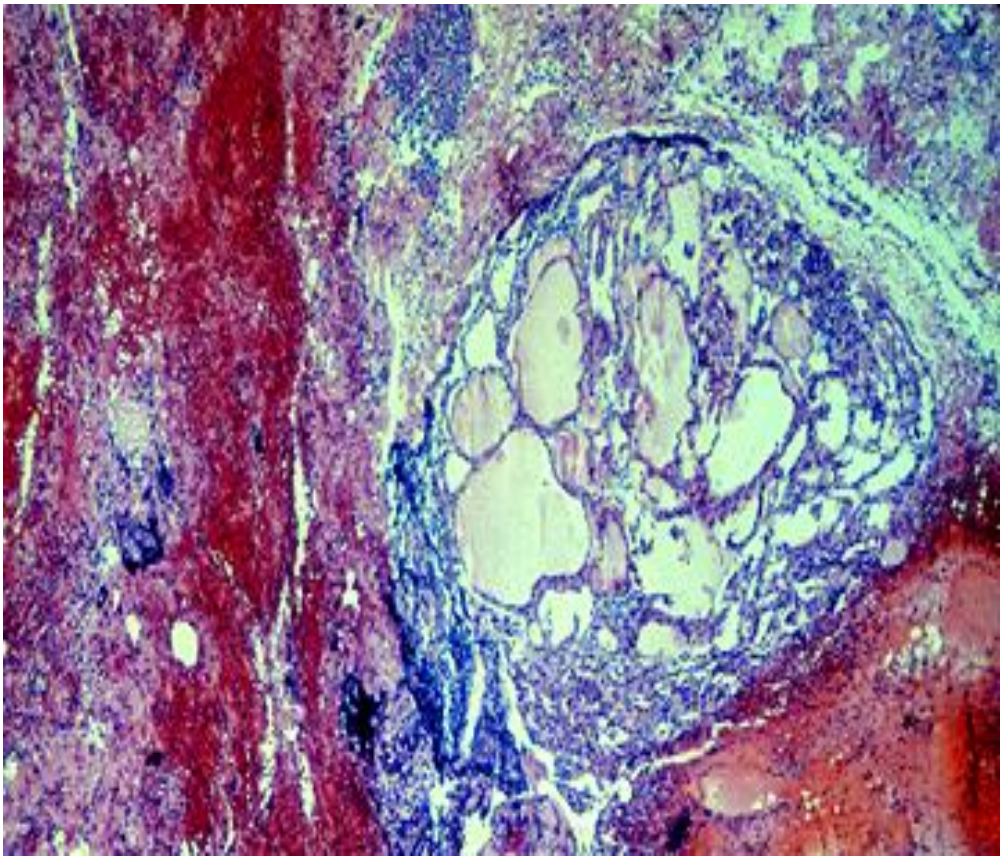
It is chronic autoimmune thyroiditis or also known as Hashimoto's thyroiditis. It was first described by Hashimoto in 1912, in patients with goitre showing intense lymphocytic infiltration of the thyroid gland described as “struma lymphomatosa”

This condition leads to thyroid failure in 10% of a population. It is the most frequent cause of hypothyroidism seen in iodine-sufficient areas of the world.

The condition cause gradual thyroid failure, with or without thyroid enlargement. It occurs more commonly in women with male to female ratio of 1:7. The estimated incidence rate is about 5-20% (30)

It is an autoimmune-mediated condition in which destruction of the gland occurs by apoptosis of the thyroid epithelial cells. The characteristic histopathological feature

consists of dense lymphocytic infiltration, with lymphoid germinal centres, and destruction of thyroid follicles (Slide 2). There are areas of fibrosis and areas of follicular-cell hyperplasia induced by thyroid-stimulating hormone (TSH). There is intrathyroidal lymphocytic infiltration of both T and B lymphocytes types.



Slide 2 Hashimoto thyroiditis

Clinically it is of two types:

- **Atrophic thyroiditis** which results primarily because of the cell-mediated cytotoxicity causing follicular cell apoptosis and complement-dependent antibody-mediated cytotoxicity leading to thyroid damage. The presence of

TSH receptor blocking antibodies results in loss of gland morphological integrity, which may be reversible.

- **Goitrous thyroiditis** resulting by one of three mechanisms (i) lymphocytic and plasma cell infiltration, (ii) production of antibodies that stimulate thyroid enlargement, (iii) excess TSH secretion.

In this condition hypothyroidism is the characteristic functional abnormality but there can be transient hyperthyroidism referred as **Hashitoxicosis**.

The chief pathology consists of presence of various antibodies and antigen-specific T cells directed towards thyroid antigens, leading to chronic autoimmune thyroiditis. The major known thyroid antigens are:

●Thyroglobulin

●Thyroid peroxidase (TPO)

●Thyroid-stimulating hormone (TSH) receptor

Impact of Autoimmune thyroid disease on pregnancy

The study by Thangaratinam et al.2011 (31) had shown that presence of various thyroid autoantibodies in euthyroid pregnant women had significant risk for unexplained subfertility, miscarriage, recurrent miscarriage, preterm birth and maternal postpartum thyroiditis. The detection of thyroid antibodies in early

pregnancy can lead to chance of developing post-partum thyroiditis in 33-50% of cases. (32)

A systematic review and meta-analysis was done restricted to thyroid autoimmunity done on two very small studies. It showed that treatment with levothyroxine lowers the risk for miscarriage and preterm birth. The other effects of treatment on pregnancy complications or subfertility, or the effect were not studied in this review.(31,33)

A recent study showed that pregnant women with thyroid peroxidase antibody have higher depression, anger and postpartum mood disturbances regardless of risk of developing postpartum thyroiditis.(32)

At present there is no guidelines or available treatment for the women diagnosed with thyroid autoantibodies. However, serum TSH should be monitored to identify the development of hypothyroidism later. (34) The Cochrane review found no benefit on outcome of pre-eclampsia when treated with levothyroxine in pregnant women in euthyroid state with TPO positive but there was reduction of miscarriages and pre term. (32)

Thyroid peroxidase (TPO) Antibodies

In pregnant women while doing thyroid function test, TPO antibodies is also done. The assessment of antibody status is important in pregnancy. The study suggest that women with subclinical hypothyroidism and positive anti-thyroid peroxidase (TPO)

antibodies tend to have more risk of adverse pregnancy outcomes at a lower TSH level than in women with negative TPO antibodies. (33)

As per the American Thyroid Association (ATA) systematic review (ATA guidelines on thyroid disease during pregnancy) suggest that the risk of pregnancy-specific complications was apparent more in TPO-positive women with about TSH >2.5 mU/L but was not consistently apparent in TPO-negative women until TSH values exceeded 5 to 10 mU/L.

In another study it was revealed that euthyroid women with high thyroid peroxidase antibody (TPO) concentrations have increased risk of foetal loss, perinatal mortality and have large for gestational age babies.(35)

These pregnant women also have more risk of developing subclinical hypothyroidism in first trimester and thyroiditis in the postpartum period.

But still in euthyroid women with positive TPO antibodies whether to monitor for development of hypothyroidism or to treat with levothyroxine is still a controversy. Though some studies reveal that treatment with levothyroxine may improve the miscarriage rate in such cases further trials are still waited for management guidelines in this regard.

Post-partum thyroiditis

It is a type of destructive thyroiditis caused by an autoimmune process which occurs within a span of one year after delivery. The global prevalence ranges from 1 to 17%.

(36) The mean prevalence in the general population was approximately 7 to 8 % of women.(36)

A higher prevalence rate of about 25% is seen certain women group who have either type 1 diabetes mellitus, had prior history of postpartum thyroiditis and if women has positive anti thyroid peroxidase antibodies (TPO).

It usually presents in the following three ways:

- Transient hyperthyroidism only
- Transient hypothyroidism only
- Transient hyperthyroidism followed by hypothyroidism and then recovery.

Clinical feature

The cause is not known. The affected women present in postpartum period with mildly enlarged, diffuse, nontender thyroid gland and abnormal thyroid function which disappears with recovery. It presents in one to four months post-delivery and lasts two to eight weeks. It has phase of hyperthyroidism followed by hypothyroidism, which lasts from approximately two weeks to six months, and then full recovery.

Pathology:

In the thyroiditis phase, thyroid gland has lymphocytic infiltration with occasional germinal centres with disruption and collapse of thyroid follicles. Usually, fine-needle aspiration biopsies reveal lymphocytes, thyroid follicular cells, and masses of colloid. In phase of recovery it reveals presence of lymphocytic infiltration and some fibrosis but the thyroid follicles are normal.

Treatment of Post-partum thyroiditis

Majority of women require no treatment for either Hyperthyroid or Hypothyroid phase of the illness but their thyroid function should be monitored.

Women who have bothersome symptoms in hyperthyroid phase require treatment with beta adrenergic antagonist drug. Women who are breastfeeding, propranolol is preferred drug of choice.

Women in hypothyroid phase with TSH >10m IU/L require initiation of levothyroxine therapy with monitoring of TSH level.

In 30 % of cases, women don't recover from the hypothyroid phase and require long term levothyroxine therapy. (37)

Isolated maternal hypothyroxinaemia (IMH)

Till present still a proper consensus regarding the definition of IMH is lacking in scientific literature.

As per 2011 American Thyroid Association's clinical guidelines on the management of thyroid disease in pregnancy, it defines IMH as a normal trimester wise maternal TSH with FT4 in the lower 5th or 10^{th} percentile. (38)

The prevalence of IMH in the population as per the study literature ranges from 1.3 to 23.9%. (39,40) This prevalence of IMH is found to be higher among the women residing in countries who still have severe iodine deficiency.

Causes of IMH

The real cause of IMH is still not known clearly. Studies suggest that there are many factors which may be associated with this entity. The various causative factor which can lead to IMH are iodine deficiency (most common), high BMI (obesity), iron deficiency, and imbalance between pro and anti-angiogenic factors.

Impact of Maternal Hypothyroxinaemia

The FASTER Trial, a National Institute of Child Health and Human Development–sponsored study (Oct.1999-Dec.2002) (41) the study documented hypothyroxinaemia in 2.1% in first and 2.3% in the second trimester. This study showed that

hypothyroxinaemia during the **first trimester** was related with preterm labour (adjusted odds ratio 1.62; 95% confidence interval 1.00–2.62) and macrosomia (aOR 1.97; 95% CI 1.37–2.83) and in the **second trimester,** it was associated with gestational diabetes (aOR 1.7; 95% CI 1.02–2.84). The study suggested that the effects of IMH are trimester dependent. In this study the author has not perform conditional analyses and so the results are inconsistent. Hence, the results of this study need to be interpreted with caution.

In a study done by Pop VJ et al. (4) on infants and toddlers born to mothers with low free thyroxine with normal TSH during 12 to 24 weeks of gestation. The study reveals that children born to such mothers had lower mean intelligence, psychomotor or behavioural scores as compared to women with normal thyroid function during gestation.

However, there is still need for further evaluation with a sufficiently powered RCT on the treatment management of IMH early in pregnancy and evaluate whether IMH should be treated or not.

Role of Iodine in Hypothyroidism:

India is a vast country with population living in certain pockets like hilly region and foothills where iodine deficiency is still prevalent. (10) Pregnancy in a woman is a

state of increase thyroid hormone production requiring iodine replenishment for the proper thyroid functioning.

The thyroid gland requires approximately 52 mcg of iodide normally to synthesize adequate amounts of Thyroxine hormone daily.

Dietary iodine is absorbed rapidly as iodide and distributed in the extracellular fluid to reach thyroid gland. Iodine can be obtained by eating foods that naturally contain it as fish, seafood, kelp, some drinking water and vegetables grown in iodine-sufficient soil. Cow's milk is also a source of iodine as it also consumes iodine from its cattle feed.

Pregnant women have higher dietary iodine requirement than non-pregnant women because of increased thyroid hormone production, increased renal iodine excretion, and foetal iodine requirements.

It is found that severe iodine deficiency in mother during pregnancy can result in low thyroid hormone production leading to primary hypothyroidism. This condition can lead to inadequate placental transfer of maternal thyroxine hormone to foetus in utero causing impaired foetal neurologic development. (42)

The World Health Organisation has recommended 250mcg of iodine in diet daily during pregnancy and lactation.

Classification of Iodine status:(43)

A system is derived for classifying the status of iodine deficiency and sufficiency, based on the median urinary iodine concentration in a population.

Iodine sufficiency: defined as a median urinary iodine concentration of:

●100 to 299 mcg/L in children and nonpregnant adults.

●150 to 249 mcg/L in pregnant women.

Iodine deficiency: defined as median urinary iodine concentrations of-

●**Mild** deficiency: 50 to 99 mcg/L

●**Moderate** deficiency: 20 to 49 mcg/L

●**Severe** deficiency: <20 mcg/L

A median average daily iodine intake of 150 mcg corresponds to median urinary iodine concentration of 100 mcg/L.

In addition, the following factors can also exacerbate the effects of iodine deficiency such as coexistent iron deficiency, selenium deficiency, and vitamin A deficiency. The

ingestion of some foods such as cassava or millet containing goitrogenic substances can lead to hypothyroidism.

Thyroid hormone and the foetus:

The developing foetus in utero till about 12 weeks of gestation is not capable of synthesizing its own thyroid hormone and is dependent on the mother for its supply of thyroid hormone. At about 10th to 12th week of gestation is when the foetal TSH starts to appears and the foetal thyroid begins to concentrate iodine and start synthesizing iodothyronines. (11,13)

However, this small amount of hormone synthesis occurs until 18th to 20th week and then gradually the foetal thyroid secretion increases.

As pregnancy reaches term the foetal serum T4, T3, and TSH concentrations differ substantially from the mothers. The foetal thyroid blood profile shows serum TSH levels are higher, serum free T4 concentrations are lower, and serum T3 concentrations are one-half those of the mothers.

After delivery, at birth, the serum TSH level increases rapidly from 50 to 80 mIU/L and then decreases to about 15mIU/L within 48 hours. The serum T3 and T4 concentrations also rapidly rises higher than normal adults.

The amount of maternal thyroid hormones crossing the placenta is not known but evidence suggests that there is significant transfer of maternal thyroid hormones across the placenta. (44,45)

Maternal thyroid hormones are very essential during the growth and development of foetal brain in the first trimester, when the foetus itself has no functional thyroid. Thyroid hormone helps in foetal brain differentiation, synaptogenesis, dendrite growth, axon myelination and neuronal migration. (9) The thyroid hormone works primarily through the TH receptors which are broadly dispersed in foetal brain tissue. (9)

It is also known that maternal TSH-receptor antibodies can cross the placenta and cause either foetal hyperthyroidism or hypothyroidism (46) . Hence, such maternal thyroid disorder adversely affects not only the mother but also the neurodevelopment of the foetus in utero.

Consequences of hypothyroidism on pregnancy:

Dave et al (13) had conducted study which showed risk of miscarriages was significantly higher in pregnant women with hypothyroidism (71.4%) as compared to those without (28%).(17) They also found statistically significant difference in the occurrence of adverse perinatal outcomes in pregnant women with hypothyroidism than those without (46% vs 7.2%).The incidence of hypothyroidism among the

women with recurrent pregnancy loss {RPL} (i.e. up to 12 weeks GA) was 4.1-16.6%.(47)

Other studies have also shown that maternal OH has been associated with increased risk of preterm births, low birth weight (LBW) babies and miscarriages. (15,16)

Sahu M et al. have similarly found in Indian population an increased risk of spontaneous miscarriage, gestational hypertension, anaemia, placental abruption and postpartum haemorrhage (PPH) in pregnant women with untreated hypothyroidism.(21) They also suggest that hypothyroid pregnant women can have increased risk of intrauterine growth restriction(IUGR) , intrauterine demise (IUD) and caesarean deliveries for foetal distress. The miscarriage rate among the subclinical hypothyroid was 12 -21% and in OH was 21%. (21)

The Indian data shows stillbirth rate of 0-16.6% among subclinical hypothyroid while 4.2% for OH. The incidence of pre-eclampsia reported among OH was 16% and 22% for SCH. (10)Similarly it shows the incidence of abruptio placentae is 16% in OH but only 5% among the subclinical hypothyroid. The intrauterine Growth Restriction (IUGR) prevalence rate for OH was 25% while 8% in subclinical hypothyroid and incidence rate were pre-term delivery was 33% among OH and 11% in the subclinical hypothyroid group.(10)

Complications of hypothyroidism in pregnancy

Hypothyroidism if untreated can have adverse effects on the pregnancy outcomes.

Depending upon the severity of the biochemical thyroid function test abnormalities, it can be classified to two types:

●Overt hypothyroidism or

●Subclinical hypothyroidism

Overt hypothyroidism

In this type of thyroid dysfunction, the pregnant women present with clinical symptoms of hypothyroidism with blood test showing elevated TSH and decreased free T4. Usually this type is uncommon because of following two factors which contribute to the low number of pregnancy in these women-(48)

1) These hypothyroid women are anovulatory.
2) Overt Hypothyroidism (new or inadequately treated) is associated with increased rate of first trimester spontaneous abortion.

Impact on pregnancy outcome of Overt hypothyroidism:(15)

In continuing pregnancy in overt hypothyroidism, it is associated with various risks of complications as mentioned:

(I) Maternal -

●Preeclampsia and gestational hypertension

●Anaemia

●Placental abruption

●Postpartum haemorrhage

●Preterm delivery, including very preterm delivery (before 32 weeks)

●Increased rate of caesarean section

(II) Foetal:

●Non-reassuring foetal heart rate trace

●Low birth weight (due to preterm delivery for preeclampsia)

●Perinatal morbidity and mortality

●Neuropsychological and cognitive impairment of child

Impact on pregnancy outcome of Subclinical hypothyroidism

In this type of thyroid dysfunction, the pregnant women have no clinical sign/symptoms of hypothyroidism at presentation. It is diagnosed only by impaired thyroid function on blood testing.

In subclinical hypothyroidism the blood examination reveals elevated TSH with a normal free T4. This is more common than overt hypothyroidism and is the most frequently occurring thyroid disorder in pregnancy.(12)

Impact of subclinical hypothyroidism in pregnancy outcome

The studies suggest that the risk of complications during pregnancy is lower in women with subclinical, than with overt hypothyroidism. (49)

However, some studies show that women with subclinical hypothyroidism have increased risk for severe preeclampsia, preterm delivery, placental abruption, and/or pregnancy loss as compared with the euthyroid women.(50)

A study by Liu H et al. (31) suggests that in women who have subclinical hypothyroidism with positive anti-thyroid peroxidase (TPO) antibodies tend to have more risk of adverse pregnancy outcomes occurring at lower TSH value than in hypothyroid women without TPO antibodies.

Impact of subclinical hypothyroidism on children

At this stage, it is uncertain to suggest that children of subclinical hypothyroid mother are at risk for neuropsychological impairment.(3) Though some observational studies suggest that it can be associated with impaired development of cognition in children.

Some expert argue that neurocognitive dysfunction of the children of women with subclinical hypothyroidism could be due to preterm delivery.(49)

Management strategy for hypothyroidism in pregnancy

- **Controversy in screening strategies in pregnancy: Universal vs high risk approach**

Screening is a strategy used to identify the possible presence of an as-yet-undiagnosed disease in individuals without signs or symptoms.

Universal screening involves screening of all individuals in a certain category.

High risk or selective screening is conducted among risk populations only.

There is still ongoing widespread controversy regarding the necessity of universal screening of thyroid function in pregnant women. The clinician need guidelines in an effort to reduce the adverse effect of thyroid dysfunctions and minimize risk of intellectual impairment in the off springs.

Guidelines from the American Association of Clinical Endocrinologists and the American Thyroid Association (2012) do not recommend universal screening but to screen only women at risk. The Society of Maternal Foetal Medicine made similar recommendations in 2012.(51)

Moreover, the American College of Obstetricians and Gynaecologists (ACOG) in 2015 recommended thyroid testing in pregnant women only if they were symptomatic, or had personal history of thyroid disease or other medical disorders such as Diabetes Mellitus.(52)

Cochrane collaboration in 2010 commented that "until more convincing data becomes available, only pregnant women at risk for thyroid disease should be tested".(53)

The Indian Thyroid Society (ITS) recommends screening of TSH levels in all pregnant women at the time of their first visit, ideally during pre-pregnancy evaluation or as soon as pregnancy is confirmed.

Due to the conflicting results in support of universal screening for hypothyroidism in pregnancy, it is not recommended by different societies. However, at present, in view of insufficient data to support universal TSH screening for thyroid dysfunction, the high-risk approach is followed.

As per the latest "*2017 Guidelines of the American Thyroid Association for the Diagnosis and Management of Thyroid Disease during Pregnancy and the Postpartum"* (54) which suggests:

- The role of Universally screening of thyroid dysfunction during pregnancy remains controversial at present.

- There is still insufficient data to recommend for or against universal screening for TSH in early pregnancy.

- Any screening for detecting low FT4 concentrations in pregnant women is not recommended.

-It strongly recommends that all pregnant women be verbally screened at the initial prenatal visit for any history of thyroid dysfunction, and prior or current use of either thyroid hormone or antithyroid medications.

-If pregnant women has any of the following risk factors, then testing for serum TSH is recommended:(54)

1. A history suggestive of hypothyroidism/hyperthyroidism or current symptoms/signs of thyroid dysfunction
2. Previous known thyroid antibody positivity or goitre present.
3. Any prior history of head or neck radiation or thyroid surgery
4. Age >30 years
5. Type 1 diabetes or autoimmune disorders
6. Past history of pregnancy loss, preterm delivery, or infertility
7. Multiple prior pregnancies (≥2)
8. Family history of autoimmune thyroid disease or thyroid dysfunction
9. Morbid obesity (BMI ≥40 kg/m2)

10. Use of amiodarone or lithium, or recent administration of iodinated radiologic contrast

11. Resident of area with moderate to severe iodine insufficiency.

Treatment of hypothyroidism in pregnancy

Levothyroxine is the only drug of choice for treatment of hypothyroidism. It is a category A, FDA approved drug and safe for use in pregnancy. Levothyroxine is easily available for treatment as 'levothyroxine sodium' in the form of oral tablets of different strengths as 25mcgm, 50mcgm, 100mcgm. It is available in moisture proof brown glass bottles and should be stored at room temperature.

The patient is advised to avoid direct exposure of medication to sunlight at all times. The prescribed dose of the medication should be taken empty stomach in the morning regularly. The patient is also advised not to take any food for at least half hour of eating levothyroxine.

If dose is missed on one day it is recommended that patient takes levothyroxine as soon as she remembers it and avoid eating for the next half hour.

If patient forgets to take medicine for one day then patient is advised to take double dose the next morning.

The medication has no contraindications.

In the prescribed recommended dosage levothyroxine has no side effects.

The studies have shown that early treatment of overt hypothyroidism (OH) with levothyroxine helps in reduction of adverse perinatal outcomes and highly recommended. The goal of treatment of oral levothyroxine is to attain trimester specific TSH levels. (10)

The pregnant women who had subclinical hypothyroidism should also be initiated on treatment with levothyroxine and studies have shown that they are less likely to develop eclampsia, pre-eclampsia, miscarriages, placental abnormalities, preterm births and low birth weight babies as compared to the untreated group.

The treatment goal is to attain target TSH in the lower half of the trimester-specific reference range as mentioned. When this is not available, then it is reasonable to target maternal TSH concentrations below 2.5 mU/L.

Justification of our present study

The evidence from present medical literature is currently based on small observational studies or those which are retrospective in nature with very few prospective ones.

Also, the result of studies is conflicting with some showing association with adverse maternal and perinatal outcome whereas others do not.

There is still controversy regarding the Universal or high-risk screening in the pregnant women and we hope to find clinical evidence for our catering population for betterment of our health services.

MATERIALS AND METHODS

Research design: A prospective cohort study was done in Christian Medical College and Hospital Vellore, India.

Study period: November 2016 to August 2017

Study Setting:

Study cohort was selected prospectively from pregnant women attending the antenatal clinic and/or endocrine clinic of Christian Medical College.

The research proposal was presented before the Institutional Review Board of Christian Medical College and Hospital, Vellore and approved prior to the recruitment of cases.

Participants:

Group 1: The pregnant women with known hypothyroidism or those diagnosed to have hypothyroidism at <20 weeks gestation were enrolled into Group 1 if they met the inclusion criteria and willing to participate in the study. These participants were treated for hypothyroidism as per Endocrine protocol.

Group 2: Pregnant women with normal TSH and willing to participate in our study were enrolled.

INCLUSION CRITERIA:

(1) All women with singleton pregnancy who were known hypothyroidism prior to pregnancy.

(2) Diagnosed to have hypothyroidism in less than 20 weeks gestation in current pregnancy.

(3) Agreed to delivery at our hospital.

EXCLUSION CRITERIA:

Pregnancies with **(**1) Multiple gestations

(2) Chronic Hypertension

(3) Pregestational diabetes (DM)

(4) Participants not willing for delivery at our Hospital.

The pregnant women attending antenatal clinic in our hospital were invited for our study. All patients were explained about the nature of our study. Informed consent was obtained from each participant of both the Group 1 and 2.

The women who were previously hypothyroid on treatment or were diagnosed for the first time during pregnancy, if they met the inclusion criteria and were willing to participate in our study were recruited as Group 1.

Of these women, who were previously Hypothyroid prior to pregnancy, in their first visit for ANC check-up, their serum TSH was done. The trimester wise cut off was taken i.e. first trimester TSH <2.5 m IU/L and second trimester TSH <3 m IU/L and their thyroid medication dose was titrated as per the Endocrine protocol.

The women were followed every 4-6 weeks with TSH, till delivery. Target TSH was to maintained its level < 3m IU/L in second and third trimesters.

These women after delivery in post-partum period were advised to start pre-pregnancy dose of Levothyroxine and continue follow up in Endocrinology department with serum TSH and TFT levels after 4-6 weeks.

The women diagnosed as subclinical or overt hypothyroidism during pregnancy were also enrolled in Group 1. The trimester wise cut off for TSH was evaluated and they were started on oral Levothyroxine as per the Endocrine protocol.

These women were also followed with TSH every 4-6 weeks and trimester wise TSH based, thyroxine dose was adjusted according to Endocrine protocol.

The pregnant women, visiting our antenatal clinic with normal thyroid function (confirmed by a blood test) who met the inclusion criteria and were willing to participate in the study were enrolled as Group 2 with prior taking informed consent.

The pregnant women of both the groups were followed as per our study protocol till delivery. In the postpartum period details were recorded. The new born baby details were also obtained from the neonatal charts post-delivery.

Reason for ordering TFTs in pregnancy:

1) Symptomatic
2) Increased BMI (>40 Kg/m2)
3) Recurrent 1st Trimester miscarriages
4) Infertility
5) Family history of Thyroid disorder

6) History of head or neck radiation

7) Coming from area of Iodine deficiency

8)Type 1 Diabetes

Variables:

Subclinical hypothyroidism was diagnosed when serum TSH levels were between 2.5-10 mIU/L (in first trimester) and between 3-10 mIU/l in second trimester with normal T4 levels.

Overt hypothyroidism was diagnosed when serum TSH levels >2.5mIU/l in first trimester (>3.0 mIU/l in second trimester) with raised T4 levels and/or Serum TSH >10mIU/L in any trimester irrespective of T4 level.

Primary Maternal Outcomes: - Hypertensive disorders in Pregnancy.

(a) Gestational Hypertension: Blood pressure 140/90mmHg or more found for the first time after 20 weeks of pregnancy without proteinuria. And returned to normal within 12 weeks post-partum.

(b) Pre-eclampsia was defined as BP >= 140/90 mm Hg with significant urine albumin or urine protein creatinine ratio >0.3.

(c) Eclampsia was defined as presence of generalized tonic clonic convulsions in women with pre-eclampsia in absence of other neurological conditions.

Secondary maternal outcomes:

(a) Spontaneous Miscarriage: Loss of foetus <22week of gestation or foetal weight 500gms after ruling out other causes of miscarriage.

(b) Anaemia: Haemoglobin <11gm/dl in first trimester/ third trimester.

- Haemoglobin <10.5gm/dl in second trimester.

(c) Gestational Diabetes: defined as fasting plasma glucose between 92-126 mg/dl in first trimester or an abnormal glucose tolerance test at 22-26 weeks of pregnancy.

(d) Transverse uterotomy for delivery of the foetus.

Primary Neonatal Outcomes: -

(a) Low Birth weight: defined as birth weight less than 2.5kg.

Secondary Neonatal outcomes: -

(a) Prematurity: defined as birth at less than 37 weeks of gestation.

(b) Intrauterine demise (IUD): defined as death of a foetus in utero at 24 weeks of pregnancy or foetus weighing >500gms.

(c) Intrauterine growth restriction (IUGR): defined as birth weight < 10th centile as per gestation age.

(d) Neonatal Hypothyroidism: defined as increased cord TSH >25 m IU/L. *

*In new born infants if cord TSH is found to be >25m IU/L then thyroid function test (TFT) is done after 72hrs. If found to be abnormal then started on thyroid replacement therapy for lifelong. The infants are followed in Neonatology Department at 3month, 6month and 1 year with regular TFT.

Sample size

The sample size was calculated using n Master 2.0 software. To test the significant difference in gestational hypertension (GHTN) and low birth weight (LBW) among pregnant women with hypothyroidism and in pregnant women with normal thyroid function.

The required number of samples is calculated as below: {reference (7)(28)}

Two Proportion - Hypothesis Testing - Large Proportion - Equal Allocation		
	GHTN	LBW
Proportion in Group 1	0.36	0.56
Proportion in Group 2	0.2	0.2
Estimated risk difference	0.16	0.36
Power (1- beta) %	80	80
Alpha error (%)	5	5
1 or 2 sided	2	2
Required sample size for each arm	122	27

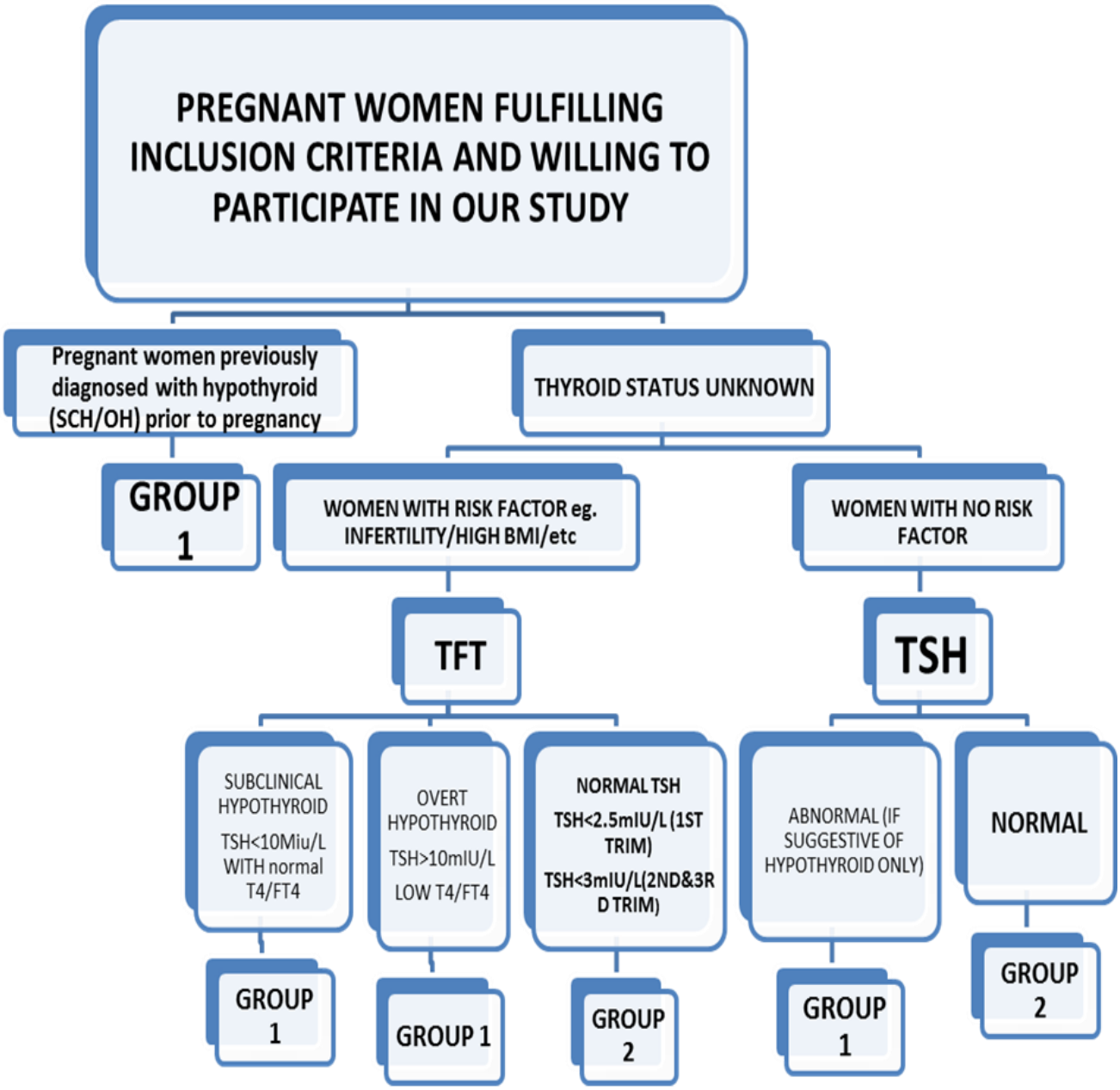
No. of participants in Group 1 = 125

No. of Participants in Group 2 = 125

Total sample size of the study is N=250.

Bias: Bias is not an issue in our study since occurrence of maternal and/or neonatal complications could not be modified in either group.

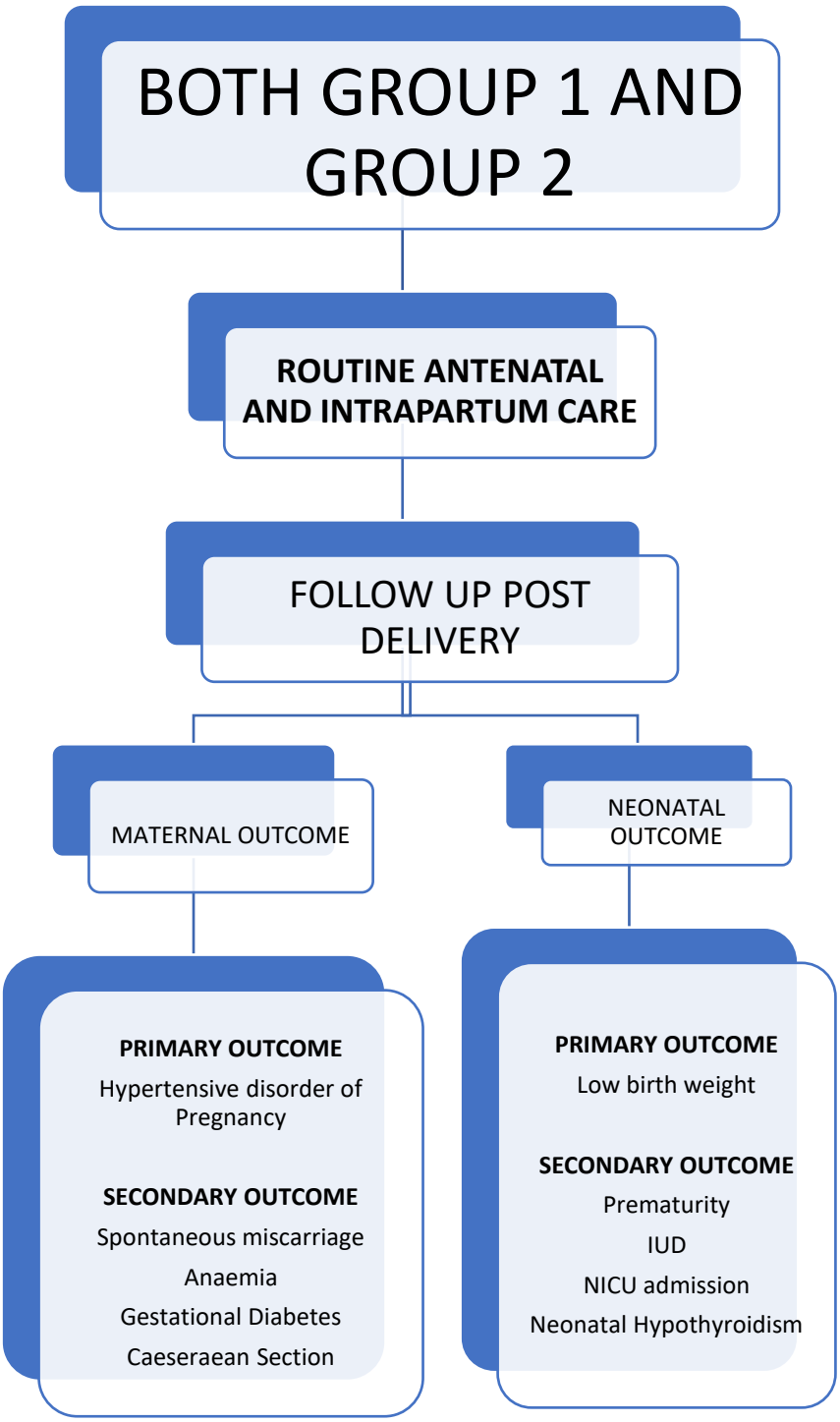
Diagrammatic Algorithm of the study



SUBCLINICAL HYPOTHYROIDISM TSH >2.5-3 <10 with normal T4

OVERT HYPOTHYROIDISM TSH>10 m IU/L or >2.5-3 and <10 with abnormal T4

(Note: If TSH is abnormal in women with no risk factor then complete TFT is done prior recruiting her to Group 1.)



BIOCHEMICAL ANALYSIS OF SAMPLES IN BRIEF:

The blood for estimation of serum TSH was collected from the patient at booking along with routine antenatal bloods and was sent to the biochemistry lab.

The TSH3-UL assay is performed by fully automated Advia Centaur XP analyser (**Siemens Healthcare Diagnostics**, Munich, Germany) in our laboratory.

Advil Centaur XP uses the direct chemiluminescent principle.

Advia Centaur XP assay uses dimethyl form of acridinium ester as the chemiluminescent label.

The Advia Centaur XP "TSH3-UL assay" is type of two-site sandwich immunoassay which uses direct chemiluminometric technology. In this process, the first antibody is the Lite reagent. This is a monoclonal mouse anti-TSH antibody labelled with acridinium ester. The second antibody, in the Solid Phase, is a polyclonal sheep anti-TSH antibody. It is covalently coupled to the paramagnetic particles.

The Advia Centaur TSH assay is very efficient and sensitive. It can measure the hormone TSH concentration up to 150 mIU/L with a minimum detectable concentration (analytical sensitivity) of 0.004 mIU/L.

The reference range is from 0.3 to 4.5 mIU/L

STATISTICAL ANALYSIS

The data entry was done by using Epidata software and analysed using MS Excel and SPSS package. The baseline characteristics and foetal and neonatal outcomes of both the study groups were analysed using descriptive statistics and frequencies.

For normally distributed continuous variables mean, SD and range were used and for non-normal continuous variables, median and IQR were used for variable description. The continuous variables were represented using histogram plot, while categorical variables were presented in simple bar charts, cluster bar charts and pie charts.

The foetal and neonatal outcomes in group 1 and group 2 of categorical variable types were compared using either by Chi-square test or Fisher's exact test. The continuous variables were compared using Independent samples t test or Mann-Whitney U test based on the normality assumption. The normality was decided based on histogram plot. p value <0.05 was considered as statistically significant.

STATISTICAL FORMULA USED

$$H_o : P_1 = P_2; \qquad H_a : P_1 \neq P_2$$

$$n = \frac{\left\{ Z_{1-\frac{\alpha}{2}} \sqrt{2\,\bar{P}\,(1-\bar{P})} + Z_{1-\beta} \sqrt{P_1\,(1-P_1) + P_2\,(1-P_2)} \right\}^2}{(P_1 - P_2)^2}$$

Where,

$$\bar{P} = \frac{P_1 + P_2}{2}$$

P_1 : Proportion in the first group

P_2 : Proportion in the second group

α : Significance level

$1-\beta$: Power

RESULTS

In our study, a total of 250 pregnant women fulfilling the inclusion criteria were recruited who were willing to participate from the antenatal clinic of our hospital. And they were divided into Group 1 and Group 2.

Group 1 = 127 (n)

Pregnant women who were known cases of hypothyroidism prior to pregnancy or diagnosed for the first time during pregnancy.

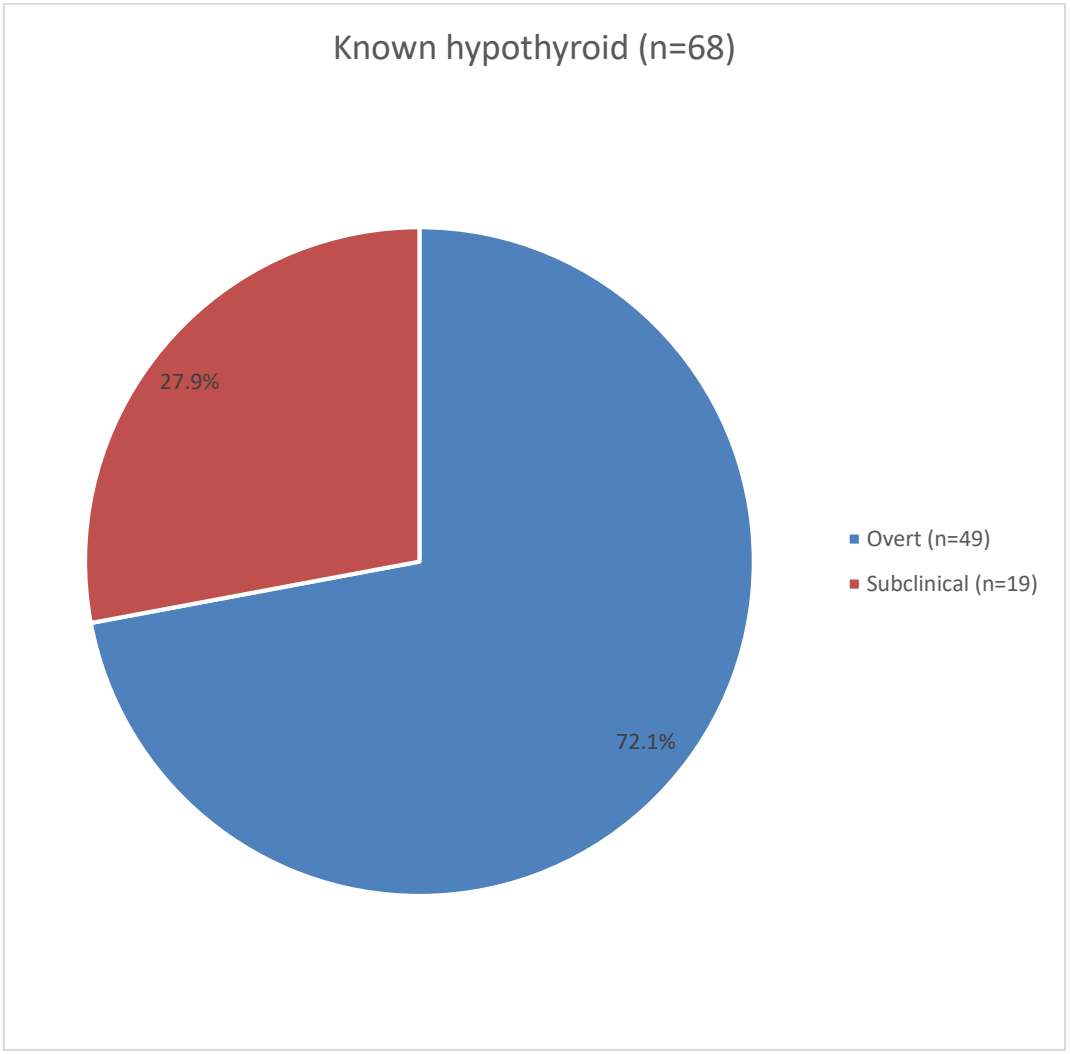
Group 2 = 123 (N)

Pregnant women with normal thyroid function tests at booking.

In Group 1, out of 127 women, 68 (53.5%) were known cases of hypothyroidism prior to pregnancy and were on medication. Among the known case of hypothyroidism n= 68, 49 women had overt and 19 women had subclinical hypothyroidism respectively (Fig.1).

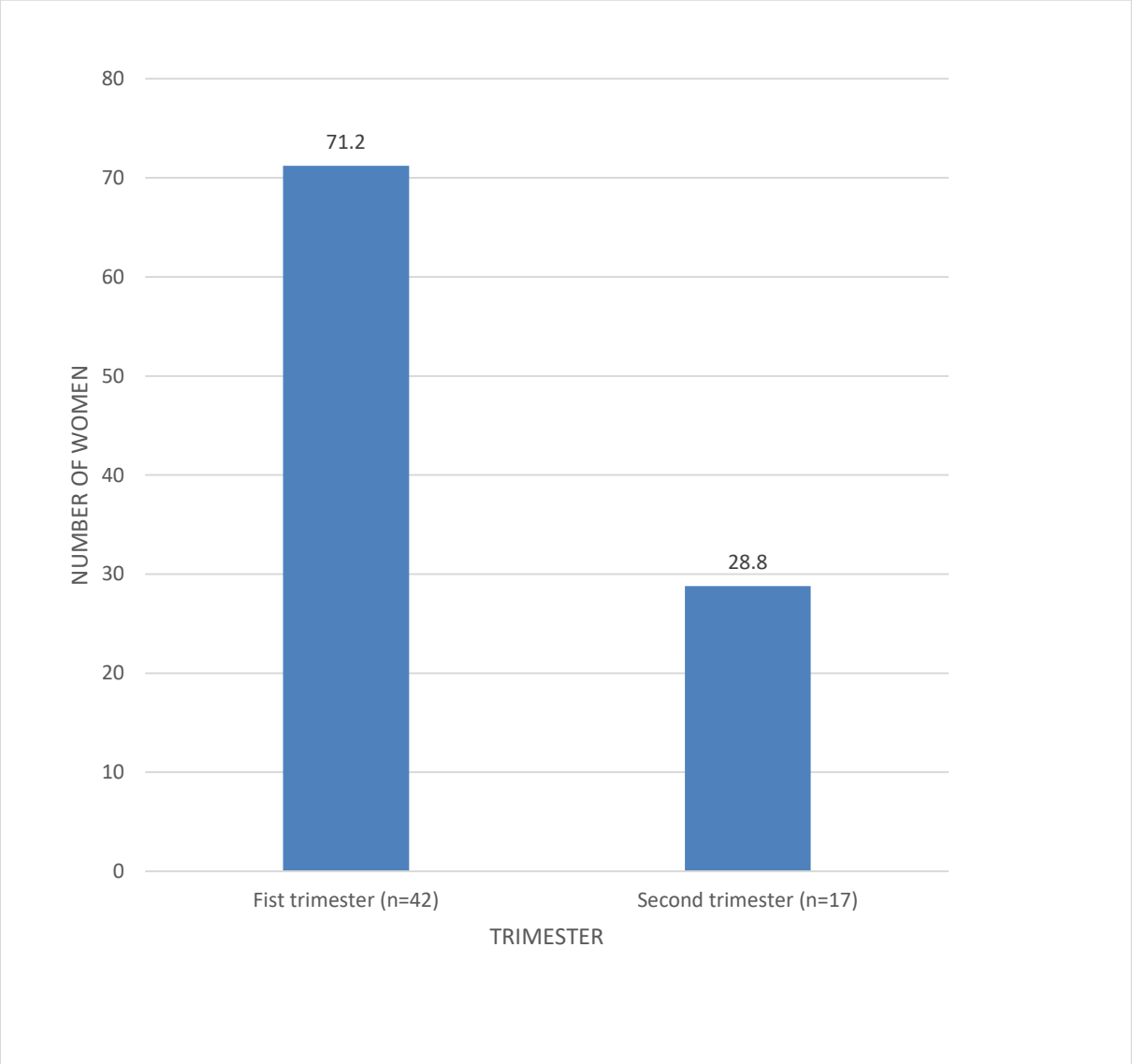
In Group 1, the rest 59 women (46.4%) out of 127 were diagnosed to have hypothyroidism during pregnancy. Out of these 71.2% (n=42) were diagnosed in first trimester and 28.8% (n=17) were diagnosed in second trimester for hypothyroidism. (Fig.2)

Figure 1: Percentage of subclinical and overt disease among known cases of hypothyroidism in Group 1



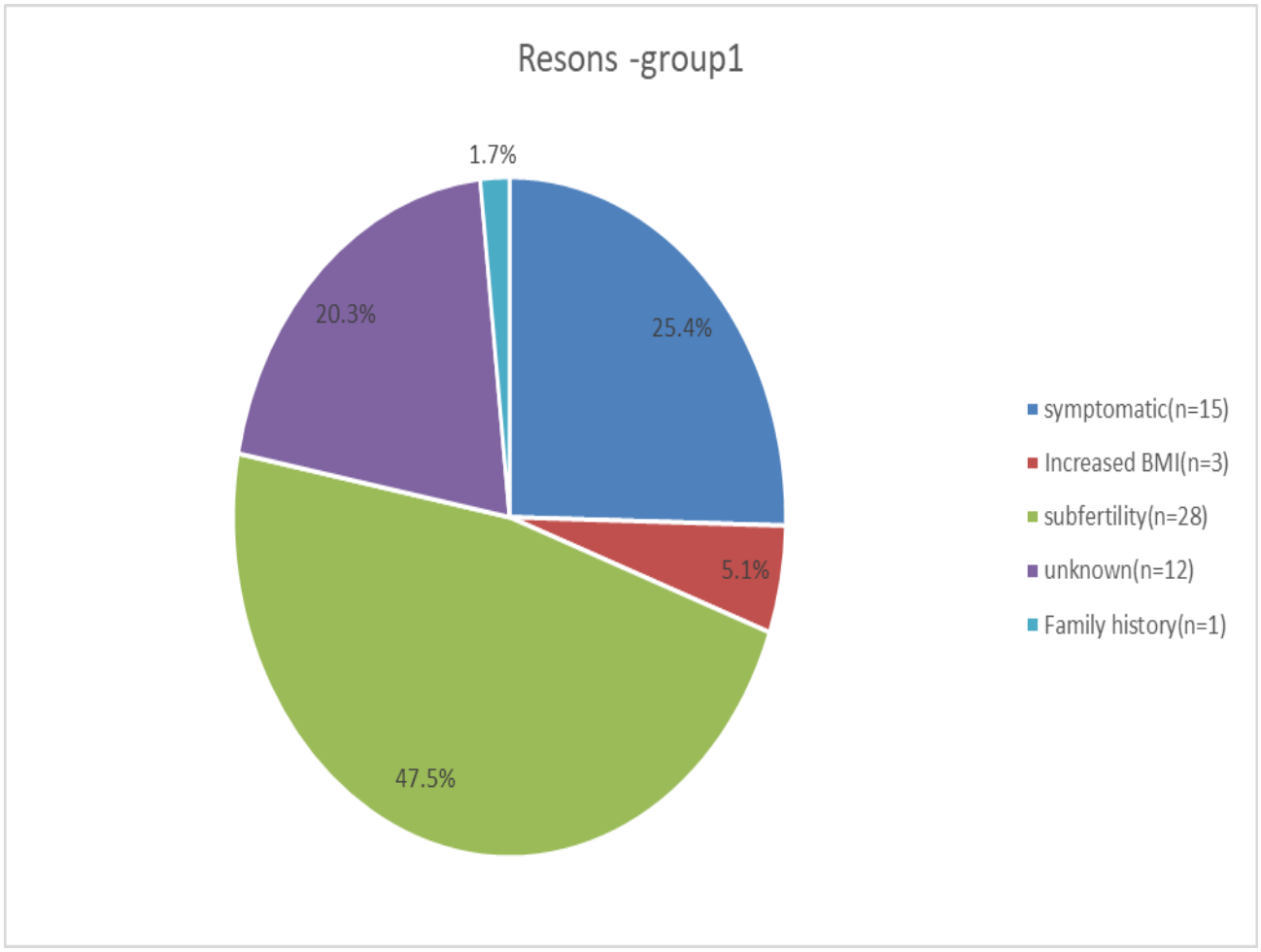
There were no cases of overt hypothyroidism among those diagnosed during pregnancy.

Figure.2 Percentage of Cases of Hypothyroidism diagnosed during pregnancy in first trimester and second trimester in Group 1



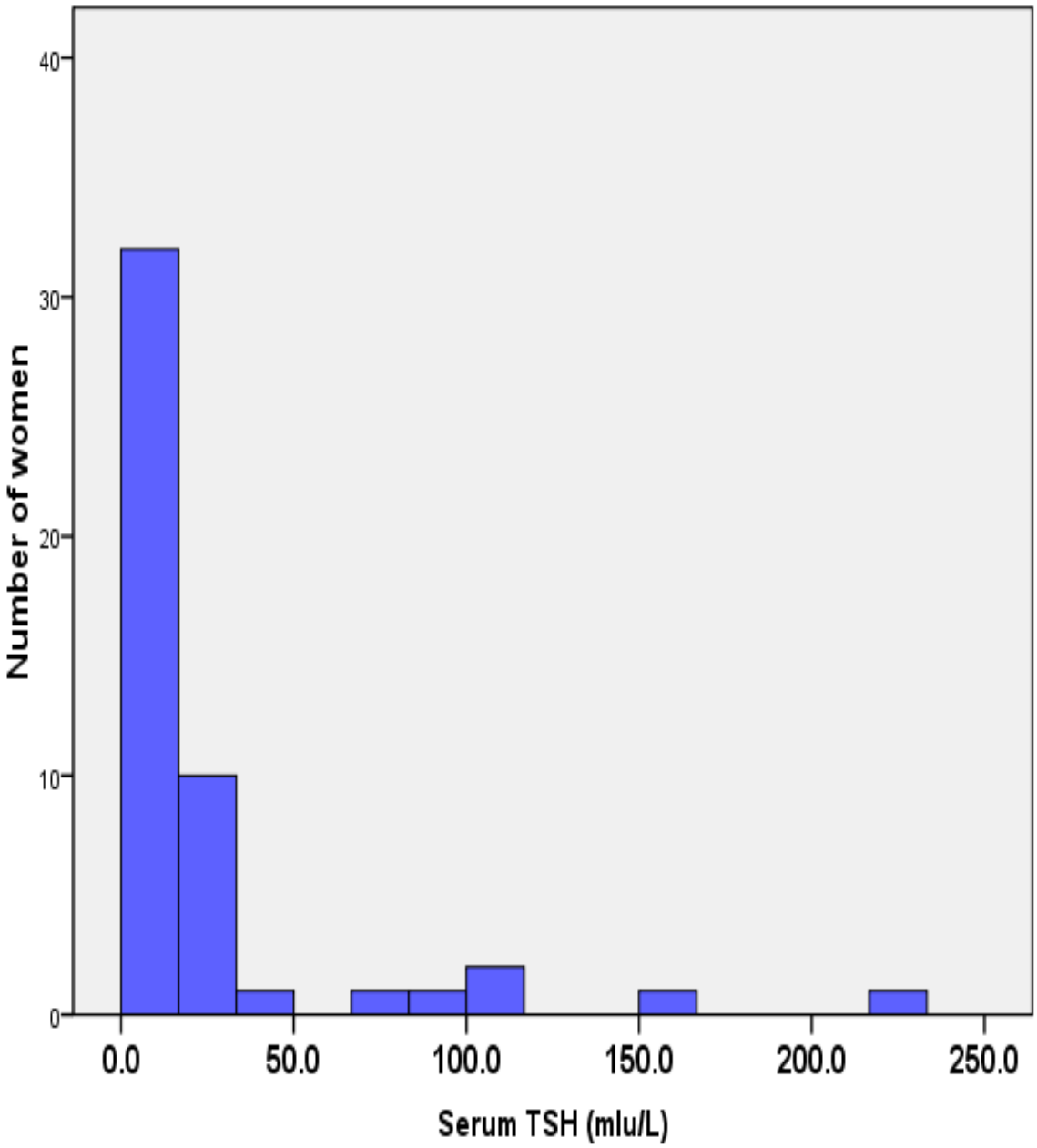
The median gestational age at diagnosis was 12 weeks. (IQR 10-16)

Figure 3: Reasons for ordering Thyroid function tests in pregnant women



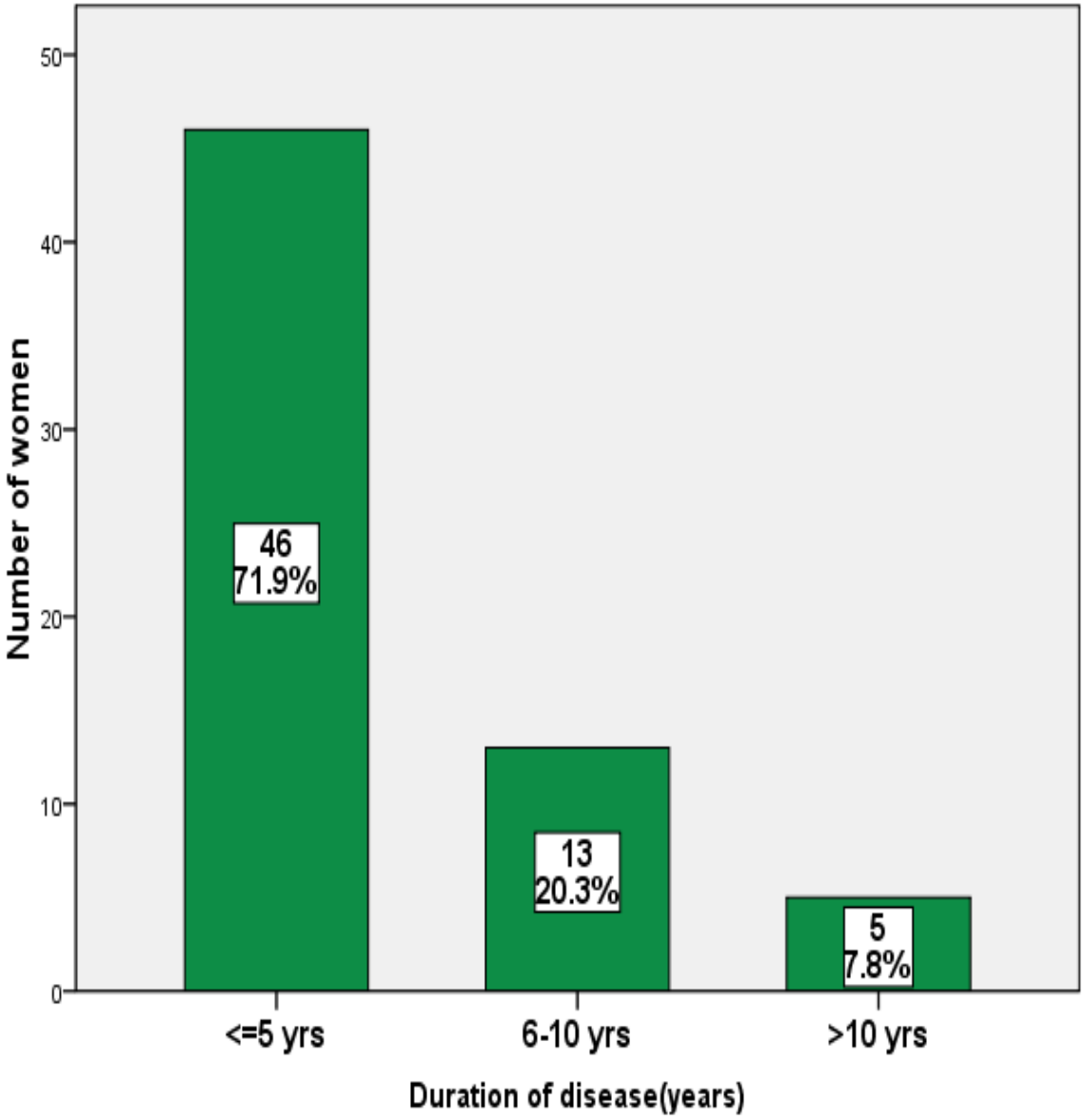
The most common indication for ordering TFTs during pregnancy was history of subfertility (n= 28) followed by presence of symptoms (n=15) and in 12 cases were picked up during routine investigations in pregnancy outside (Fig 3).

Figure 4. Values of serum TSH (mIu/L) in Group 1



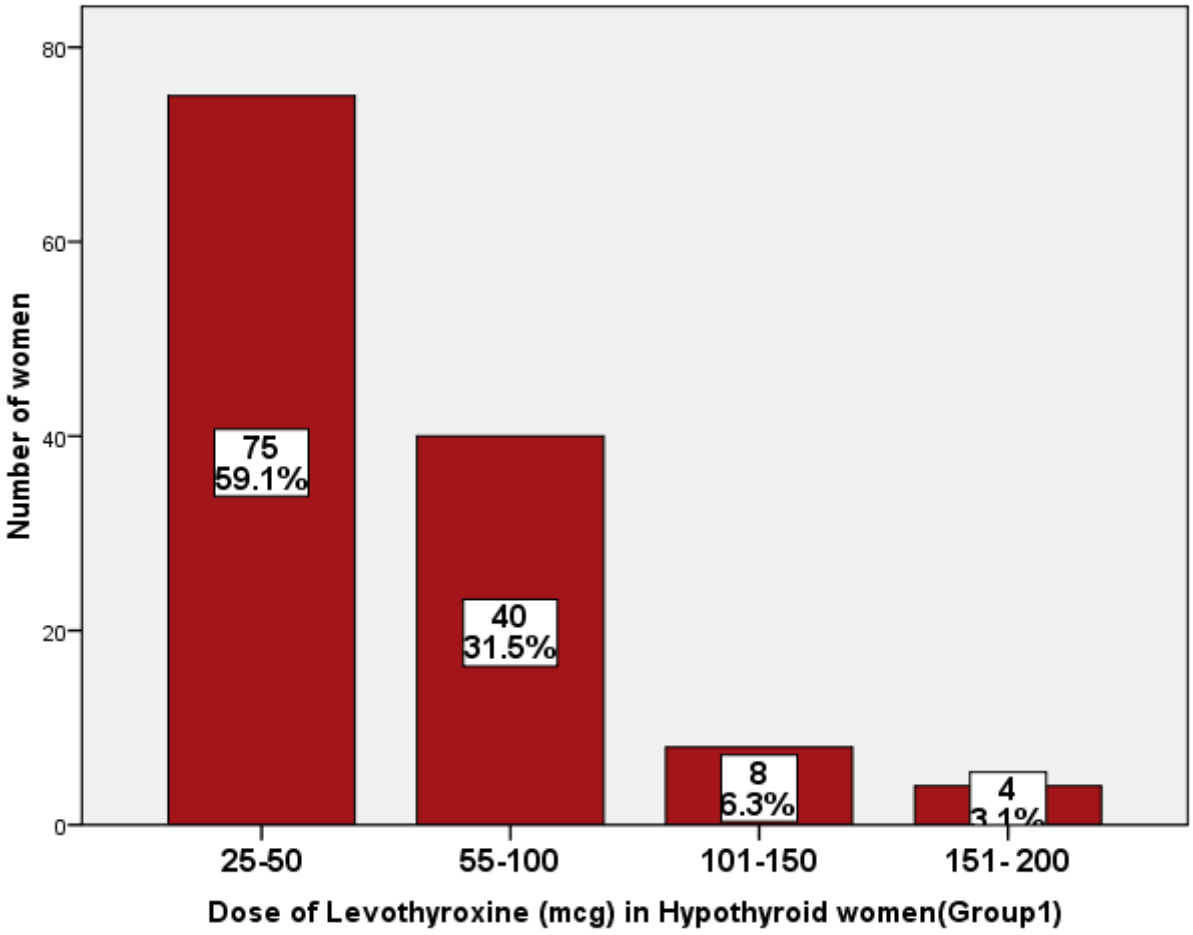
Median TSH value was found to be 5.6 mIu/L with inter quartile range (IQR) being 4.5-7.6

Figure 5 Duration of disease in years in known hypothyroid cases in Group 1



Median duration of disease was 3 years with IQR (2-6 years). In four cases, duration of disease was not known by the patients

Figure 6: Dose of Thyroxine in pregnant women with hypothyroidism in Group 1



Median dose of Levothyroxine in pregnant women with prior known hypothyroidism was 75 micrograms with IQR (50-100) in comparison to that in women diagnosed to have hypothyroidism during pregnancy where the dose was 50 micrograms with IQR (25-50). Among the women who were on medications, 43 (33.8%) required alteration (increased) in dose of medication. There were none in whom the dose required to be reduced.

MATERNAL CHARACTERISTICS

Table 1 Maternal Characteristics

Maternal characteristics	Group1 n=127	Group2 N=123	p value
Age (years) Mean+/-SD	27.5 +/-4.1	25.1 +/-4.4	<0.001*
BMI (Kg/m2); Mean +/-SD	28.1+/-4.4	25.1+/-4.1	<0.001**`
Parity			
Primigravida (n, %)	64 (51.2 %)	68 (54.4 %)	0.789 (both)
Multigravida (n, %)	61 (48.8 %)	57 (45.6 %)	
Bad obstetric H/O (N, %)	n=37(29.1%)	N=26 (21.1%)	0.145
Previous abortion	17(45.9%)	9(7.2%)	0.369
Previous Neonatal death	3(8.1%)	3(11.5%)	0.983
Previous abortion and Neonatal	17(45.9%)	14(53.8%)	0.537

The maternal characteristics were similar in both groups in terms of age, Parity, and bad obstetric H/O (Table1).

Although there was statistically significant difference in the ages of pregnant women in both the groups with older women being found more in group 1, this could be explained by the large sample size we had and this difference may not be clinically significant.

Heavier women were found in group1 as compared to group 2, and this difference was statistically significant (p value <0.001). This could be explained by the fact that hypothyroidism occurs more in heavier women.

In Group 1 had 37 pregnant women (29.1%) had previous bad obstetrics history, of which 45.9 % (n=17) had abortion,8.1 %(n=3) had neonatal death and 45.9% (n=17) had history of previous abortion and neonatal death. In Group 2 ,26 pregnant women (21.1 %) had previous bad obstetrics history, of which 07.2% (N=9) had abortion,11.5% (N=3) had previous neonatal death and 53.8% (N=14) had both previous abortion and neonatal death. Among both Groups previous abortion was clinically high in Group 1 (45.9%) as compared with Group 2 (7.2%) but was not statically significant. (Table 1) and (Fig.10).

Among 250 pregnant women, three had comorbid medical condition of Bronchial Asthma and all of these were from Group 2.

Figure 7: Distribution of ages of pregnant women in both groups

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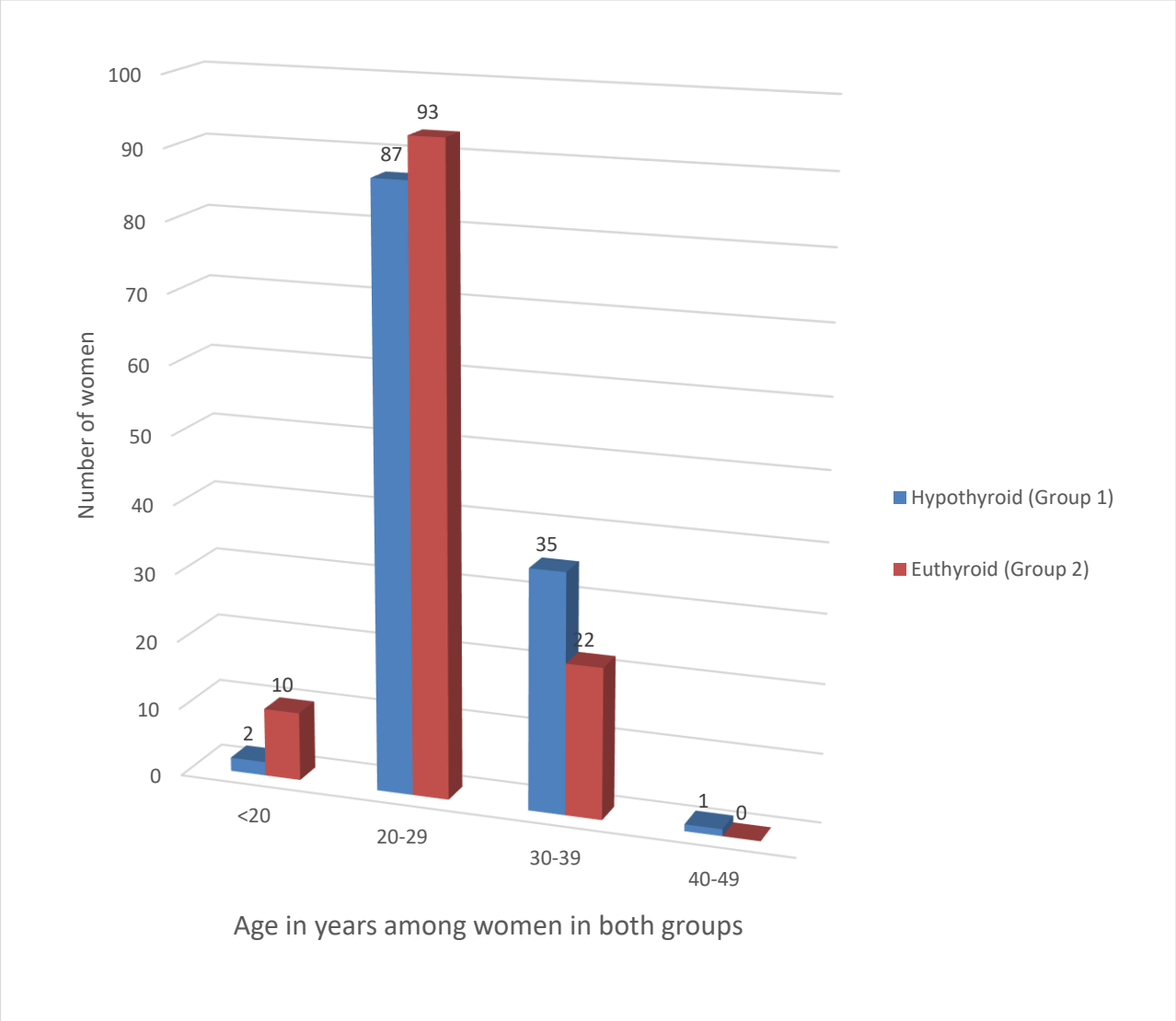
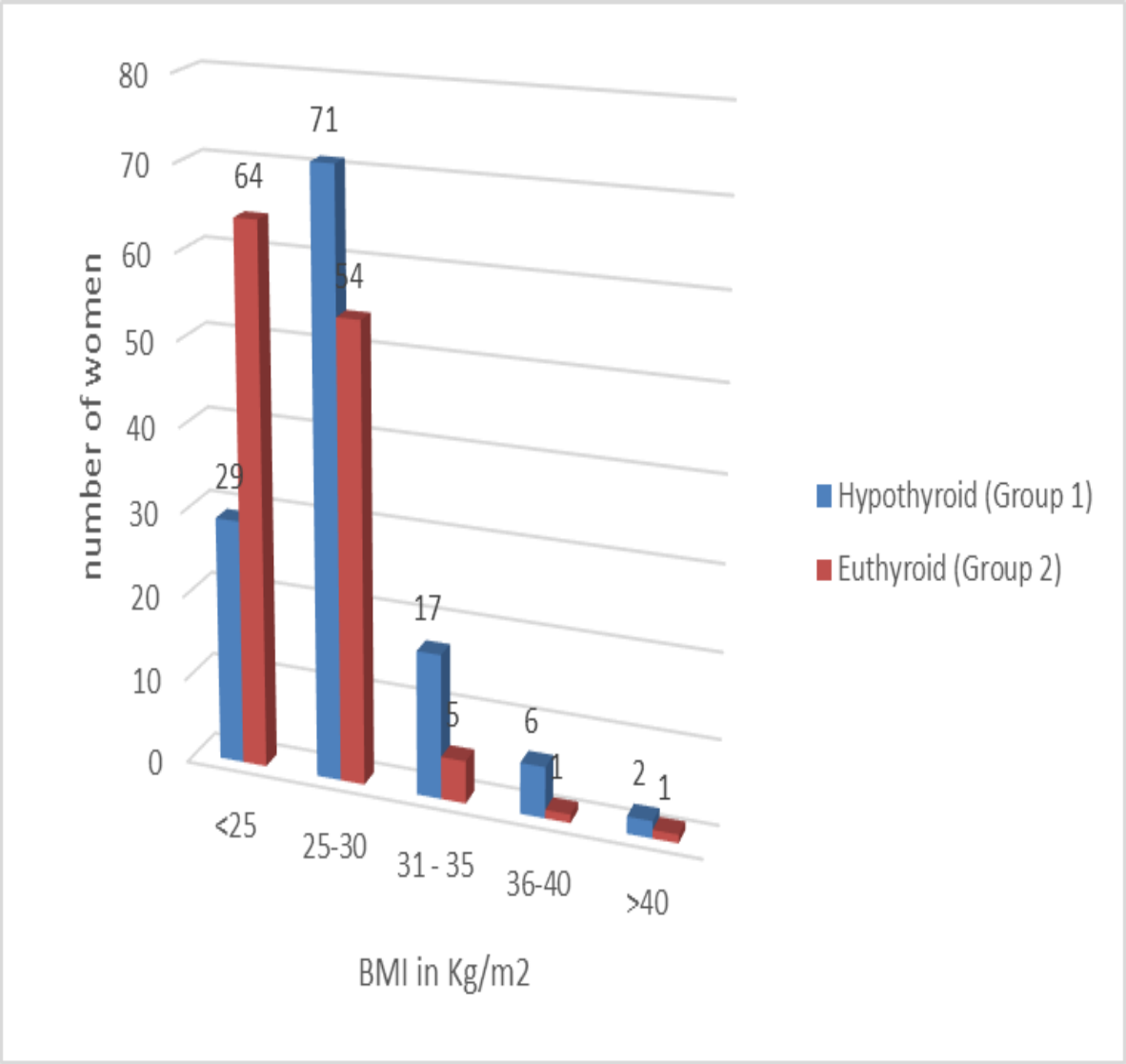


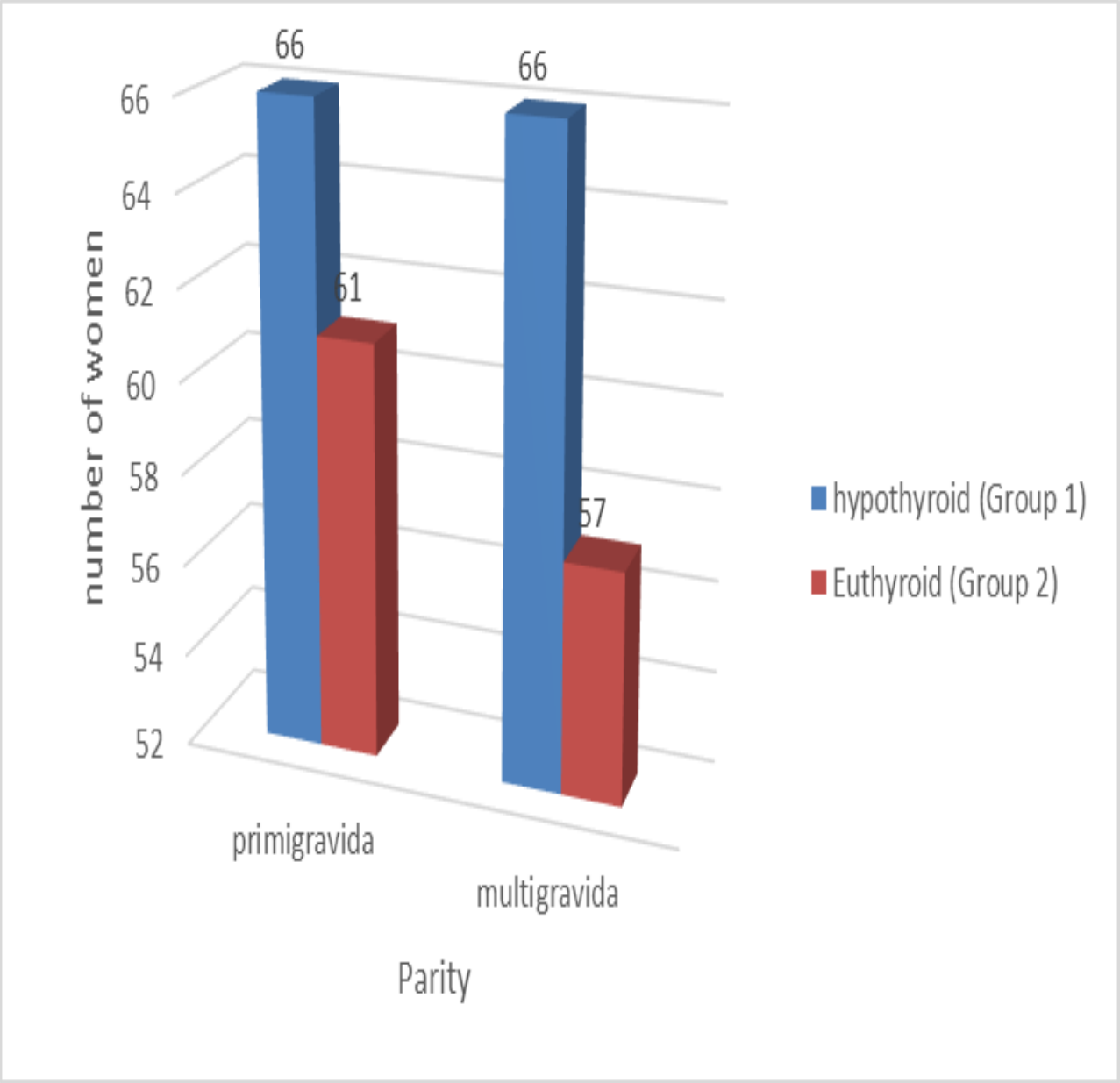
Majority of pregnant women recruited in both groups were in age group 20-29 years. (Fig.7)

Figure 8. Distribution of BMI of pregnant women in both the groups.



Majority of pregnant women in both groups were under BMI 26-30 kg/m2. More women in Group 1 had higher BMI than group 2 (p value <0.001). This is because women with hypothyroidism are known to be obese.

Figure 9. Parity of women in both groups.



Majority of pregnant women in both Group 1 and Group 2 were primigravida as compared with multigravida.

Figure 10: Occurrence of bad obstetric history in both groups.

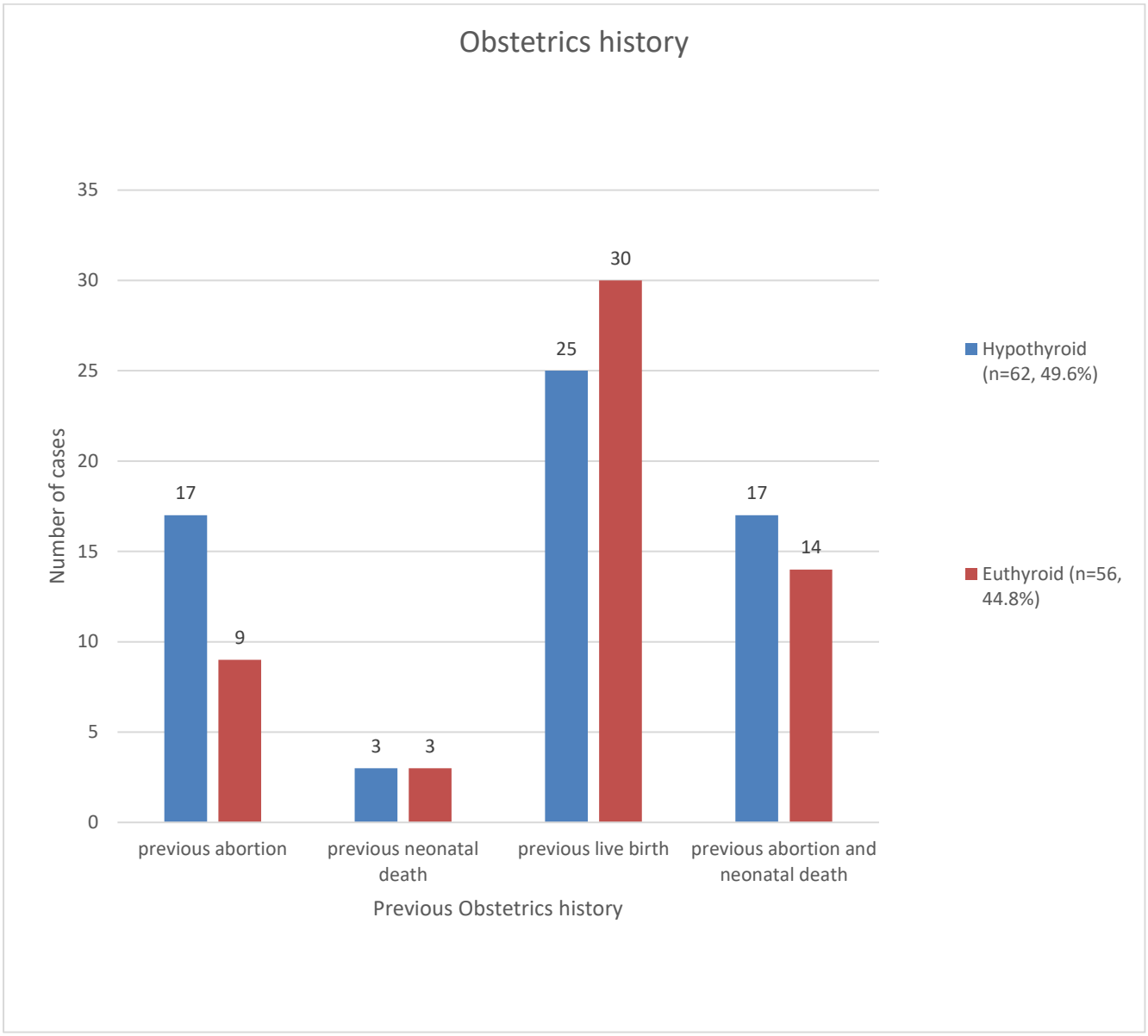


TABLE 2: MATERNAL OUTCOMES IN BOTH GROUPS

OUTCOMES	GROUP 1(n=127)	GROUP 2 (N=123)	p VALUE
Hypertensive disorders	19(15%)	4(3.3%)	0.001*
Anaemia	33(26%)	21(17.1%)	0.087
Preterm Labour	7 (5.5%)	11 (8.8%)	0.294
Gestational diabetes	36 (28.3%)	24 (19.5%)	0.102
LSCS	45 (35.4%)	39 (31.7%)	0.533

*significant p value

Occurrence of hypertensive disorders was significantly more in Group 1as compared to Group 2 with p value (.001**).** Although, anaemia and gestational diabetes were seen more often in Group 1, the difference between the two groups was not statically significant. The incidence of LSCS was similar in both the groups (Table 2). There were no cases of miscarriage (threatened/spontaneous) or postpartum haemorrhage in either group.

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TABLE 3: NEONATAL OUTCOMES IN BOTH GROUPS

NEONATAL OUTCOMES	**GROUP 1** (n=127)	**GROUP 2** (N=123)	**p*VALUE**
Gestational age at delivery <37 weeks (preterm birth)	12 (9.4%)	11 (8.9%)	0.890
Birth weight at delivery<2.5 kg (LBW)	19 (15.0%)	16 (13.0%)	0.656
Cord TSH >25 mIU/dl	4 (3.1%)	0	0.122
Foetal Growth Restriction	17 (13.4%)	18 (14.6%)	0.776
Neonatal Jaundice	1 (0.8%)	6 (4.9%)	0.063
NICU Admission	26 (20.4%)	12 (9.7%)	0.018
Neonatal death	1 (0.8%)	0	>0.999

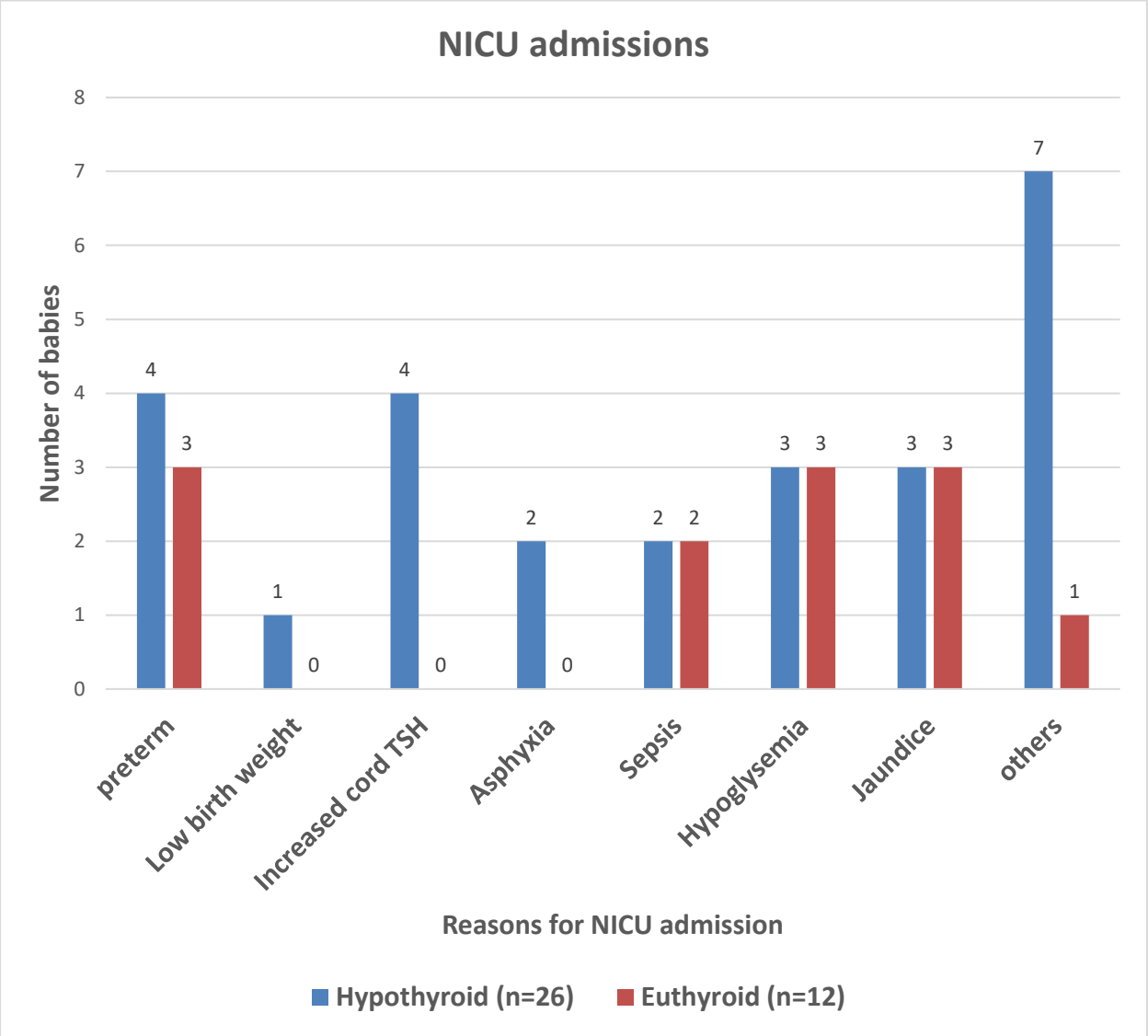
Although there were significantly more number of cases of hypertensive disorders in Group1 in comparison to group2, the effects of this was not reflected in the neonatal outcomes since there was no difference in the incidence of foetal growth restriction

and Low birth weight between the two groups (Table 3) This can be explained by the fact that; most cases of hypertension were mild to moderate gestational hypertension. There was no significant difference in the occurrence of severe gestational hypertension or pre-eclampsia in both the groups.

More babies from Group 1 had NICU admission than Group 2 (p value 0.018). Reasons were admission to NICU have been depicted in Figure 11.

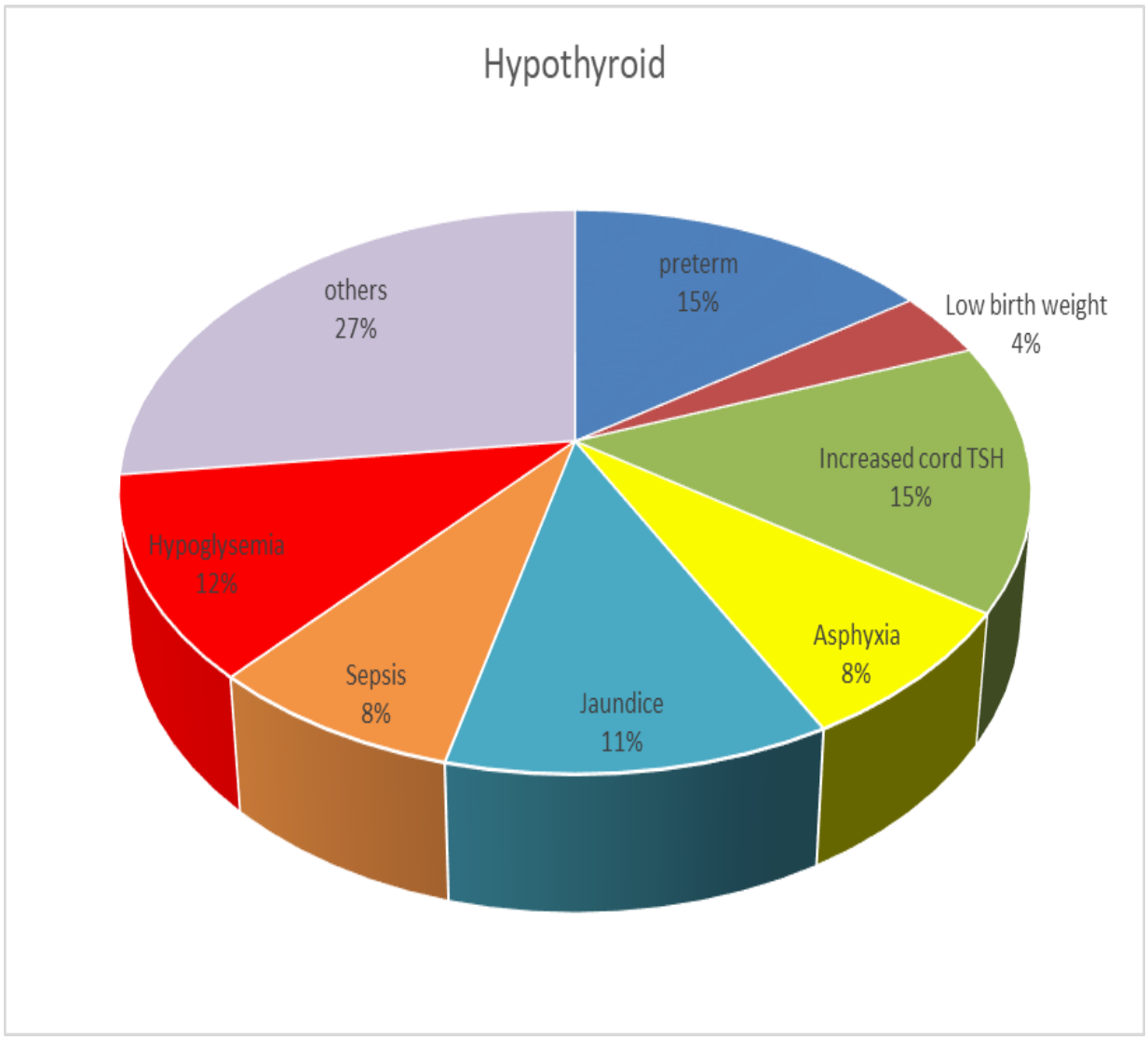
Out of 250 babies,4 had elevated cord TSH. All four of them belonged to group1. One baby from Group1 had early neonatal death due to severe asphyxia. The parturient had rupture uterus following labour induction and baby was severely depressed at birth with cord pH 6.

Figure 11. Causes of NICU admission



NICU admission were significant in Group 1.

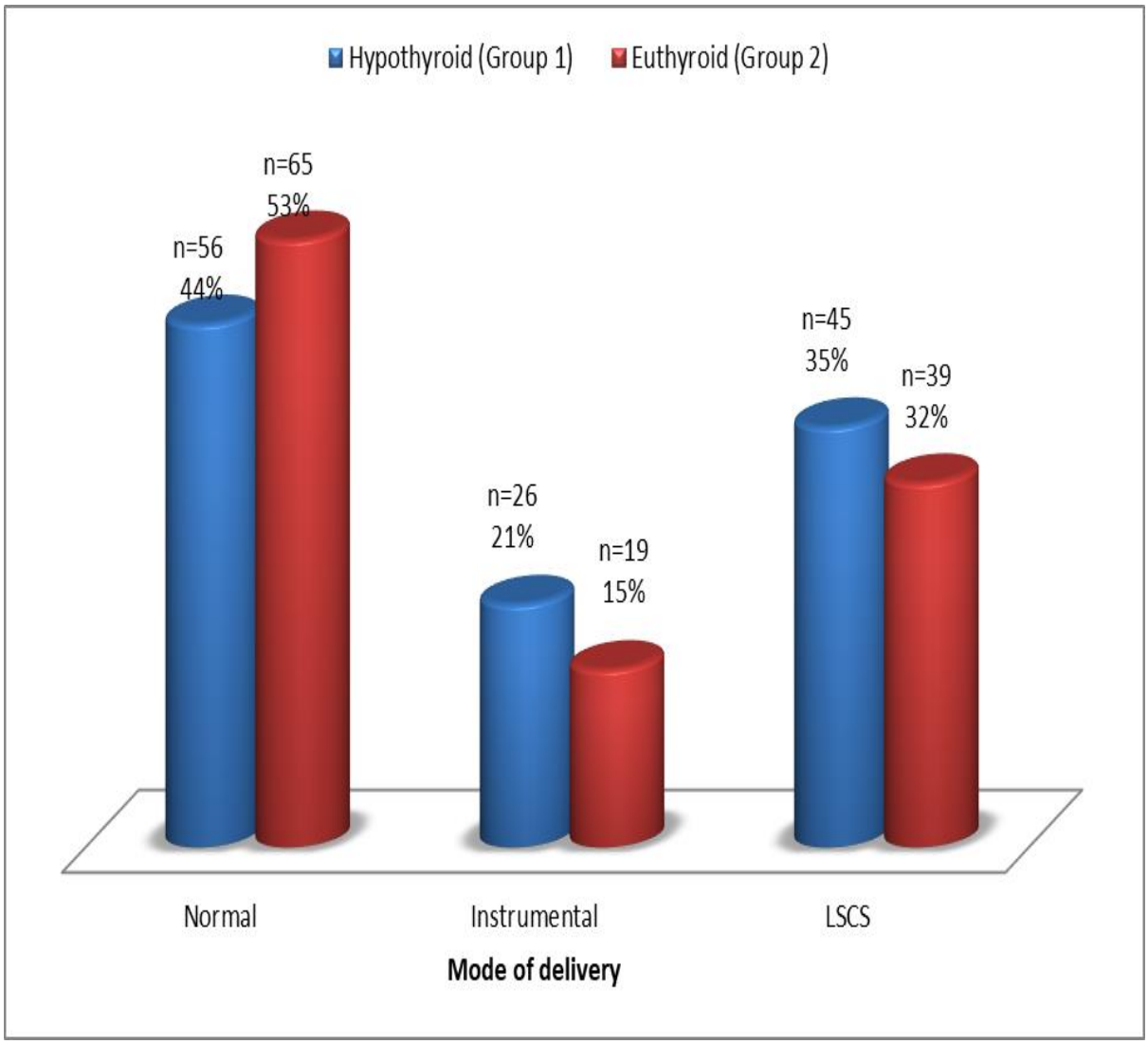
Figure 12: REASONS FOR NICU ADMISSION IN GROUP 1.



Most common cause for admission to NICU in Group1 babies has been classified as others. (27%, n=7) (Figure 12). Out of 7, three babies had bilateral hydronephrosis; two had transient tachypnoea of the new born; one baby had patent ductus arteriosus and one was diagnosed to have microcephaly.

More Babies born to women with overt hypothyroidism required NICU admission for sepsis / jaundice/, hypoglycaemia. However, statistical significance could not be calculated due to small numbers.

Figure:13 MODE OF DELIVERY IN BOTH GROUPS



The mode of delivery of pregnant women in both the groups were comparable. There was no statistical difference in the LSCS rates of women between both the groups. (p value 0.533) (Table 1)

TABLE 4: COMPARISON OF MATERNAL AND NEONATAL OUTCOMES AMONG KNOW CASE OF HYPOTHYROID WOMEN WITH OVERT AND SUBCLINICAL HYPOTHYROIDISM IN GROUP 1.

Maternal outcome	Overt hypothyroidism (n=49)	Subclinical Hypothyroidism (n=19)	p. value
Hypertensive disorders	3 (6.1%)	3 (15.8%)	0.338
Anaemia	13 (26.5%)	5 (26.3%)	0.986
Preterm labour	3 (6.1%)	3 (15.8%)	0.338
GDM	20 (40.8%)	3 (15.8%)	0.050
LSCS	16 (32.7%)	9 (47.4%)	0.259
Neonatal outcome			
Low birth weight	5 (10.2%)	4 (21.1%)	0.253
NICU admission	9 (18.4%)	5 (26.3%)	0.512
Cord TSH >25(mIu/L)	1 (2.0%)	2 (10.5%)	0.187

On doing subgroup analysis of women in group1, (overt versus subclinical hypothyroidism) there were no statistically significant differences for either adverse maternal or neonatal outcomes between the types of hypothyroidism.(Table 4).

Total 4 neonates had cord TSH >25 m IU/L . Out of these , one baby was born to mother who was newly diagnosed to have hypothyroidism during pregnancy.

DISCUSSION

Thyroid dysfunction is the most common endocrine disorder in pregnancy as per ACOG practice Bulletin 2001 (55). This prospective cohort study helped us to study the maternal and perinatal outcomes in women with thyroid dysfunction.

There have been several studies, evaluating maternal and neonatal outcomes in women with hypothyroidism.(7,13,15,20)

Few studies have compared the outcomes between hypothyroid and euthyroid women (28,56) Others have compared outcomes between pregnant women with overt and subclinical hypothyroidism (7,9,15). Few have compared outcomes between women with subclinical hypothyroidism and euthyroid.(49). Some of these studies are retrospective in nature(9) whereas others are prospective in nature(7,56). Few had both retrospective and prospective components (28)

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Ours is a prospective cohort study which compared outcomes of pregnant women with and without hypothyroidism. We recruited women with known hypothyroidism and those diagnosed for the very first time during pregnancy in Group1 and compared their outcomes with euthyroid women (Group2). Other studies which compared hypothyroid and euthyroid women, recruited hypothyroid women diagnosed for first time during pregnancy (7,56)

In Group1, 68 women had known thyroid dysfunction prior to pregnancy and 59(46.4%) were diagnosed for the first time during pregnancy. Out of these, 72 % were overt and the rest were subclinical(Fig1). There were no overt cases amongst those diagnosed for the first time.

Amongst those in whom hypothyroidism was diagnosed for the first time during pregnancy, the median age of diagnosis.

was 12 weeks. Others have also reported similar findings (57)

Most common reason for ordering TFTs in pregnancy was history of subfertility (47.5%) followed by presence of symptoms (25.4%).

Twelve cases were picked up incidentally during routine antenatal bloods. (Fig 3). These blood tests were done elsewhere since we do not offer universal screening for thyroid dysfunction in pregnancy.

Median Serum TSH levels in our cohort was 5.6 mIU/L with IQR being 4.5-7.6. (Figure 4) Median dose of levothyroxine(LT4) was 75 mcg in women with known hypothyroidism as compared to 50 mcg in those diagnosed during pregnancy. Women who were diagnosed to have subclinical hypothyroidism elsewhere and were on thyroid medications, were continued on these drugs. Women who were diagnosed here during pregnancy, were started on thyroid medications as per Endocrine protocol.

Nearly 34% women required an increase in the dosage of their medications. (Figure 6) Abalovich et al studied the evolution of disease during the pregnancy, reported that nearly 70 % of their cohort required increased dose of medication during pregnancy. (15)

The maternal characteristics were comparable in terms of age, parity and previous obstetric history.(Table 1) Although there was statistically significant difference in the age of participants in both the groups (p value <0.001, Table 1), mean age being higher in group 1, this could be explained due to large sample size and may not be clinically significant.(58)

History of miscarriages comparable in both hypothyroid and euthyroid groups.

The difference in BMI in both groups was statistically significant (p value <0.001, Table 1). This can be explained by the fact that hypothyroidism is known to effect heavier women and vice versa (women are obese due to decreased metabolic rate as found in hypothyroidism).

Significantly greater number of women in Group1 had hypertensive disorders of pregnancy. (15% vs 3.3 %, p value <0.001, Table 2). Nirmala et al found 3.8 times higher risk of developing gestational hypertension in women with hypothyroidism (28).

Others have also found increased incidence of gestational hypertension in women with hypothyroidism(7,21,56). Most of these studies have reported increased incidence with overt hypothyroidism over euthyroidism(21,56,59). This is in contrast to some studies where authors have found difference between euthyroid and both overt as well as subclinical hypothyroidism (7).

We did not find statistically significant difference in maternal anaemia between the two groups (26% versus 17.1%, p value 0.08, Table2). This is consistent with findings in the study by Kishore et al.(7). This is contrast to findings reported by other authors, who have reported higher incidence of anaemia in overt hypothyroid pregnancies (56).

Relationship between anaemia and thyroid dysfunction is not very clear. It has been hypothesized that iron deficiency might cause impairment of heme dependant thyroid peroxidase, which limits synthesis of thyroid hormones and can lead to hypothyroidism. This can be corrected by correcting the maternal anaemia (60).

The incidence of preterm birth in both the groups were similar (5.5% vs 8.85, p value 0.294, Table 2) This is in contrast to studies done by other researchers, who found an increased incidence of preterm birth and low birth weight secondary to prematurity. (7,59) Some have found this increased incidence even with subclinical hypothyroidism (7,49)

In a systematic review done by Sheehan et al (61), there was a small but highly statistically significant increase in risk of preterm labour in both hypothyroidism and hyperthyroidism. The major limitation of this review, however, was the studies included in it did not differentiate between spontaneous and iatrogenic preterm births

The possible biological explanation for this is not very clear. It has been proposed that myometrium possesses thyroid hormone receptor, the presence of TSH and thyrotropin releasing hormone receptors have been demonstrated in the primate uterus (62). TSH is also known to bind to HCG receptors which are present in the myometrium (63).

Our study did not find increased risk of gestational diabetes in women with hypothyroidism (p value 0.102, Table 2). Karakosta et al found four-fold increase in incidence of gestational diabetes in women with elevated TSH and presence of thyroid antibodies (64).

The LSCS rate in both the groups were similar (35.4% vs 31.7%, p value 0.533, Table 2). This is in contrast to findings in other studies.(7,21,28,65). These studies have found foetal distress as the cause for increased LSCS rate in hypothyroid group (21,21,56).

Others have reported increased incidence of abruption with untreated hypothyroidism. (7,49) We did not encounter abruptio placentae in either group

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We did not have any cases of miscarriage in either group. This is in contrast to findings reported by others.(20,28,56). Abalovich et al found that overt as well as subclinical hypothyroidism was associated with miscarriages if left untreated. Incidence of miscarriage decreased if thyroid replacement was adequate (15).

Overall, the incidence of maternal complications in our study was low in comparison to others (7,21,28,56) This could be explained by the fact that majority of the pregnant women in Group1 were subclinical hypothyroid (19% among previously known cases and 100% in newly diagnosed cases)

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Casey et al. (49) comparing outcomes in subclinical hypothyroidism versus euthyroidism, found hypothyroid women were 3 times more likely to have placental abruption (relative risk 3.0, 95% CI 1.1-8.2). Preterm birth was almost two-fold higher in women with subclinical hypothyroidism.

Our low incidence of complications could be explained by optimal treatment and strict follow up of hypothyroidism in pregnancy. 34% women in group1 required increased dosage of levothyroxine and none required reduction in the dosage of medications. This has been substantiated by Abalovich et al who stated that evolution of

pregnancies conceived under hypothyroidism did not depend upon severity of disease but adequacy of therapy.(15)

We did not notice any increased adverse maternal outcome while comparing overt versus subclinical hypothyroidism. (Table 4). Sahu et al, comparing overt versus subclinical hypothyroidism found that incidence of gestational hypertension, gestational diabetes, foetal growth restriction. intrauterine demise and neonatal complications were significantly higher in the overt group (65)

Among the neonatal outcomes, we did not find significant difference in the incidence of preterm birth, delivery <37 weeks, and low birth weight babies (birth weight <2.5 kgs) between both the groups (p values 0.890 and 0.656, Table 4) Leung et al had found increased incidence of low birth weight babies secondary to premature delivery or gestational hypertension in both overt and subclinical hypothyroidism(49).

Others have found increased incidence of low birth weight in untreated hypothyroidism (56)

Although we reported higher incidence of hypertensive disorders in Group1 in comparison to Group2, this did not reflect in our neonatal outcomes, as the incidence of low birth weight and foetal growth restriction were comparable in the two groups (p value 0.656 and 0.776, Table 3). This is in contrast to findings by others (7,56,65).

Our findings can be explained by the fact that most women in group 1 had mild to moderate gestational hypertension. The incidence of severe gestational hypertension and pre-eclampsia were similar in both groups (4.7% versus 2.3 %, p value)

Significantly more number of babies from Group1 required NICU admission (20.4% versus 9.7%, p value 0.018). This is in spite of the fact that incidence of low birth weight and foetal growth restriction was similar in both groups. (Table 3)

On analysing, reasons for NICU admission in babies born to hypothyroid mothers, we found that majority (27%) of babies went to NICU for causes, such as presence of congenital anomaly, and patent ductus arteriosus which are unlikely related to maternal hypothyroid status. (Fig 11) Other causes for neonatal admission like sepsis/hypoglycaemia and neonatal jaundice were comparable in both groups. (Fig 11)

One neonate had severe asphyxia and died after 6 hours of life. The parturient had ruptured uterus following induction of labour and underwent emergency laparotomy for the same. (Fig 11)

Rajalakshmi et al found increased incidence of NICU admissions in babies born to mothers with untreated hypothyroidism. They had also found increased occurrence of foetal growth restriction and low birth weight in these babies (56)

We found 4 cases (3.1%) of neonatal hypothyroidism in Group 1 and none in Group 2 (p value 0.122, Table 3). Similar findings have been reported by others.(56)

CONCLUSIONS

In conclusion, we found that evolution of disease during pregnancy does not depend upon the degree of hypothyroidism, whether overt or subclinical, but on the therapy received.

Timely initiation of therapy and close treatment monitoring to titrate the dose of individual thyroid medications is of paramount importance in prevention of obstetric and neonatal complications.

LIMITATIONS

The limitations of our study are, we did not test for thyroid antibodies due to financial constraints. Thyroid peroxidase antibodies have been identified as risk factors for obstetric complications such as miscarriage, preterm birth, gestational diabetes (34,64)

We did not do long term follow up of the neonates born to hypothyroid mothers. A number of observational studies have found that children born to mothers with untreated hypothyroidism during pregnancy have lower IQ scores when compared with those born to euthyroid women (3,66).

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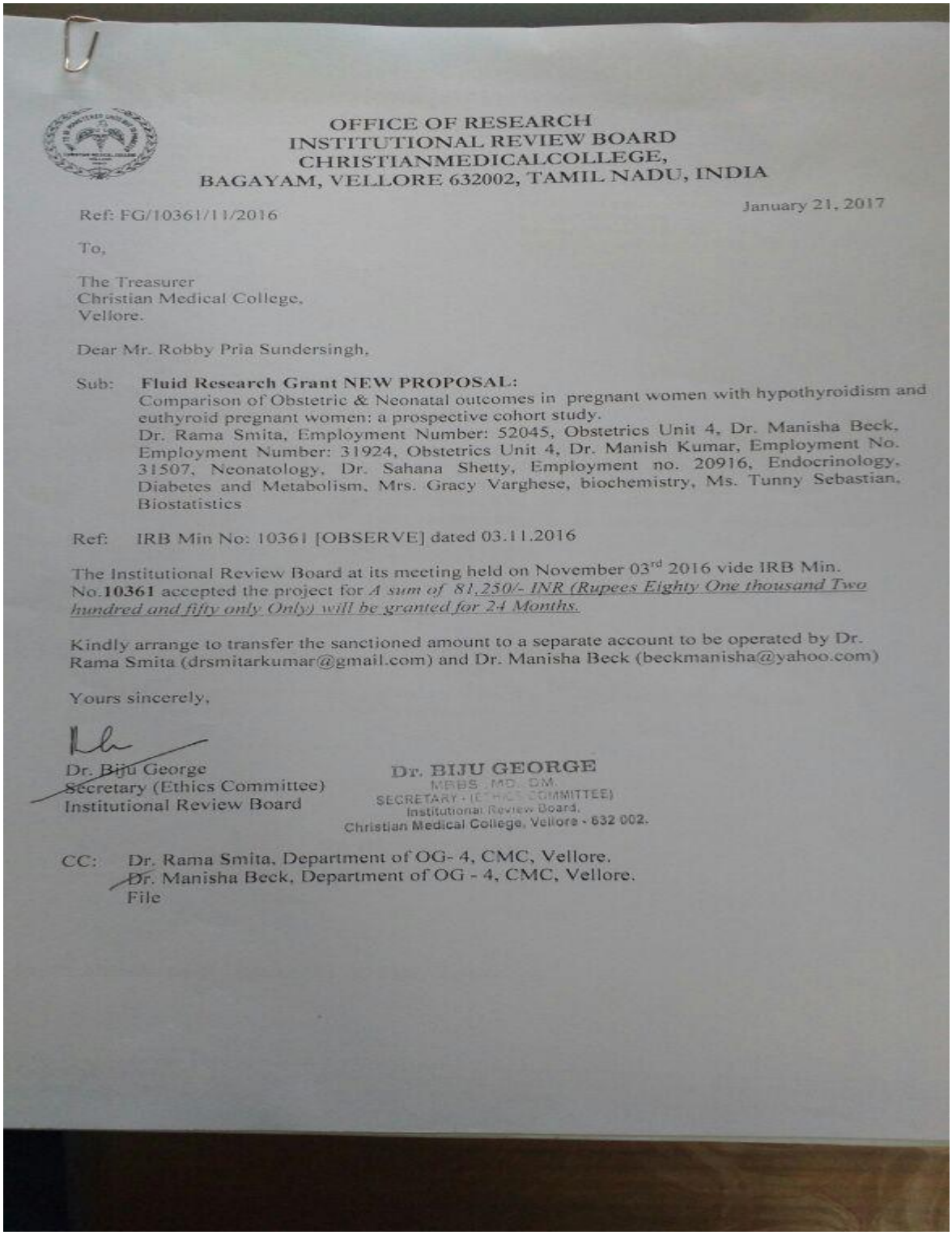
ANNEXURES



OFFICE OF RESEARCH
INSTITUTIONAL REVIEW BOARD
CHRISTIANMEDICALCOLLEGE,
BAGAYAM, VELLORE 632002, TAMIL NADU, INDIA

Ref: FG/10361/11/2016

January 21, 2017

To,

The Treasurer
Christian Medical College,
Vellore.

Dear Mr. Robby Pria Sundersingh,

Sub: **Fluid Research Grant NEW PROPOSAL:**
Comparison of Obstetric & Neonatal outcomes in pregnant women with hypothyroidism and euthyroid pregnant women: a prospective cohort study.
Dr. Rama Smita, Employment Number: 52045, Obstetrics Unit 4, Dr. Manisha Beck, Employment Number: 31924, Obstetrics Unit 4, Dr. Manish Kumar, Employment No. 31507, Neonatology, Dr. Sahana Shetty, Employment no. 20916, Endocrinology, Diabetes and Metabolism, Mrs. Gracy Varghese, biochemistry, Ms. Tunny Sebastian, Biostatistics

Ref: IRB Min No: 10361 [OBSERVE] dated 03.11.2016

The Institutional Review Board at its meeting held on November 03rd 2016 vide IRB Min. No.**10361** accepted the project for *A sum of 81,250/- INR (Rupees Eighty One thousand Two hundred and fifty only Only) will be granted for 24 Months.*

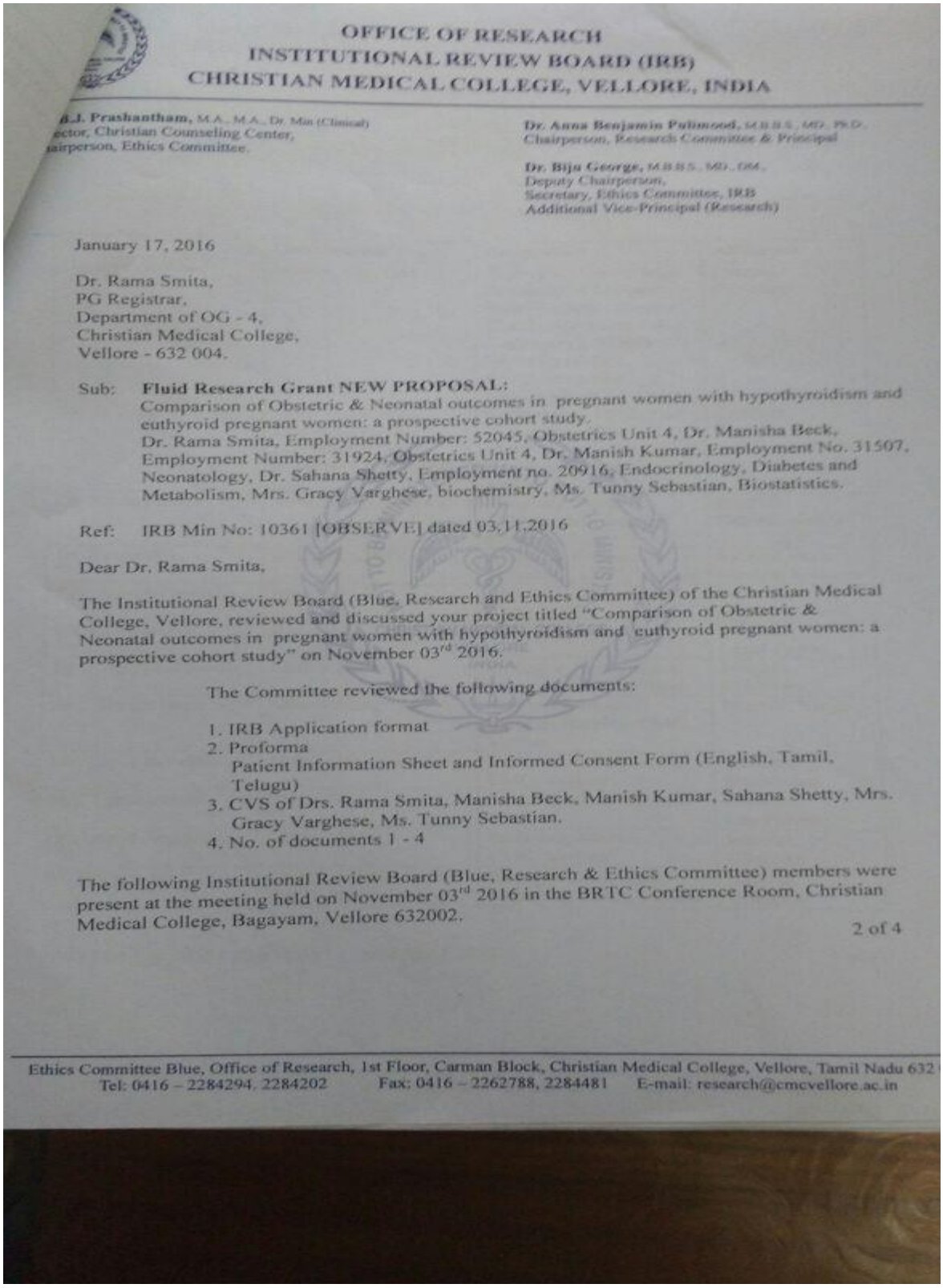
Kindly arrange to transfer the sanctioned amount to a separate account to be operated by Dr. Rama Smita (drsmitarkumar@gmail.com) and Dr. Manisha Beck (beckmanisha@yahoo.com)

Yours sincerely,

Dr. Biju George
Secretary (Ethics Committee)
Institutional Review Board

Dr. BIJU GEORGE
MBBS., MD., DM.
SECRETARY - (ETHICS COMMITTEE)
Institutional Review Board,
Christian Medical College, Vellore - 632 002.

CC: Dr. Rama Smita, Department of OG- 4, CMC, Vellore.
Dr. Manisha Beck, Department of OG - 4, CMC, Vellore.
File



OFFICE OF RESEARCH
INSTITUTIONAL REVIEW BOARD (IRB)
CHRISTIAN MEDICAL COLLEGE, VELLORE, INDIA

B.J. Prashantham, M.A., M.A., Dr. Min (Clinical)
ector, Christian Counseling Center,
airperson, Ethics Committee.

Dr. Anna Benjamin Pulimood, M.B.B.S., MD., Ph.D.,
Chairperson, Research Committee & Principal

Dr. Biju George, M.B.B.S., MD., DM.,
Deputy Chairperson,
Secretary, Ethics Committee, IRB
Additional Vice-Principal (Research)

January 17, 2016

Dr. Rama Smita,
PG Registrar,
Department of OG - 4,
Christian Medical College,
Vellore - 632 004.

Sub: **Fluid Research Grant NEW PROPOSAL:**
Comparison of Obstetric & Neonatal outcomes in pregnant women with hypothyroidism and euthyroid pregnant women: a prospective cohort study.
Dr. Rama Smita, Employment Number: 52045, Obstetrics Unit 4, Dr. Manisha Beck, Employment Number: 31924, Obstetrics Unit 4, Dr. Manish Kumar, Employment No. 31507, Neonatology, Dr. Sahana Shetty, Employment no. 20916, Endocrinology, Diabetes and Metabolism, Mrs. Gracy Varghese, biochemistry, Ms. Tunny Sebastian, Biostatistics.

Ref: IRB Min No: 10361 [OBSERVE] dated 03.11.2016

Dear Dr. Rama Smita,

The Institutional Review Board (Blue, Research and Ethics Committee) of the Christian Medical College, Vellore, reviewed and discussed your project titled "Comparison of Obstetric & Neonatal outcomes in pregnant women with hypothyroidism and euthyroid pregnant women: a prospective cohort study" on November 03rd 2016.

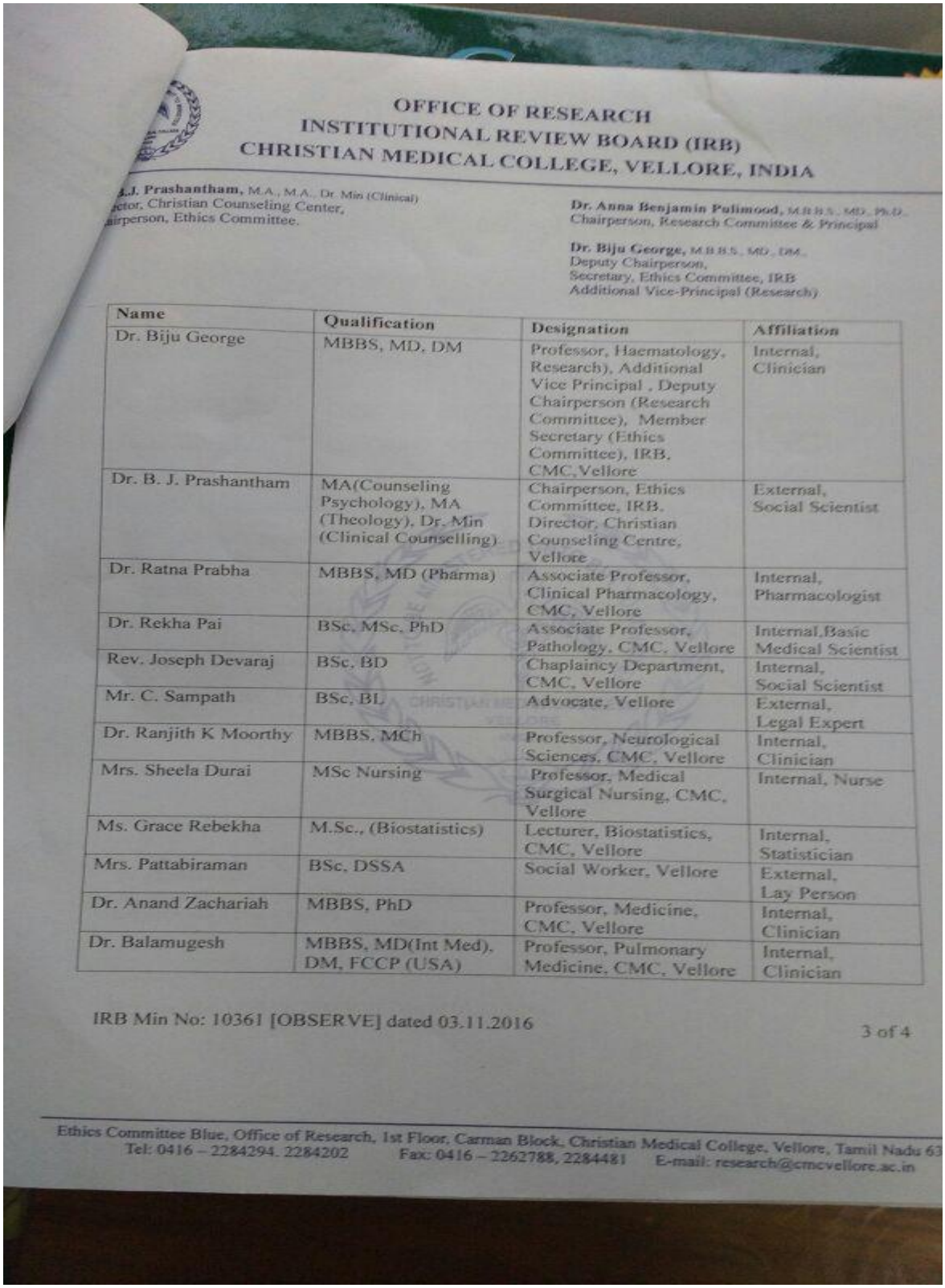
The Committee reviewed the following documents:

1. IRB Application format
2. Proforma
   Patient Information Sheet and Informed Consent Form (English, Tamil, Telugu)
3. CVS of Drs. Rama Smita, Manisha Beck, Manish Kumar, Sahana Shetty, Mrs. Gracy Varghese, Ms. Tunny Sebastian.
4. No. of documents 1 - 4

The following Institutional Review Board (Blue, Research & Ethics Committee) members were present at the meeting held on November 03rd 2016 in the BRTC Conference Room, Christian Medical College, Bagayam, Vellore 632002.

2 of 4

Ethics Committee Blue, Office of Research, 1st Floor, Carman Block, Christian Medical College, Vellore, Tamil Nadu 632
Tel: 0416 – 2284294, 2284202 Fax: 0416 – 2262788, 2284481 E-mail: research@cmcvellore.ac.in



OFFICE OF RESEARCH
INSTITUTIONAL REVIEW BOARD (IRB)
CHRISTIAN MEDICAL COLLEGE, VELLORE, INDIA

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ector, Christian Counseling Center,
airperson, Ethics Committee.

Dr. Anna Benjamin Pulimood, M.B.B.S., MD., Ph.D.,
Chairperson, Research Committee & Principal

Dr. Biju George, M.B.B.S., MD., DM.,
Deputy Chairperson,
Secretary, Ethics Committee, IRB
Additional Vice-Principal (Research)

Name	Qualification	Designation	Affiliation
Dr. Biju George	MBBS, MD, DM	Professor, Haematology, Research), Additional Vice Principal , Deputy Chairperson (Research Committee), Member Secretary (Ethics Committee), IRB, CMC,Vellore	Internal, Clinician
Dr. B. J. Prashantham	MA(Counseling Psychology), MA (Theology), Dr. Min (Clinical Counselling)	Chairperson, Ethics Committee, IRB. Director, Christian Counseling Centre, Vellore	External, Social Scientist
Dr. Ratna Prabha	MBBS, MD (Pharma)	Associate Professor, Clinical Pharmacology, CMC, Vellore	Internal, Pharmacologist
Dr. Rekha Pai	BSc, MSc, PhD	Associate Professor, Pathology, CMC, Vellore	Internal,Basic Medical Scientist
Rev. Joseph Devaraj	BSc, BD	Chaplaincy Department, CMC, Vellore	Internal, Social Scientist
Mr. C. Sampath	BSc, BL	Advocate, Vellore	External, Legal Expert
Dr. Ranjith K Moorthy	MBBS, MCh	Professor, Neurological Sciences, CMC, Vellore	Internal, Clinician
Mrs. Sheela Durai	MSc Nursing	Professor, Medical Surgical Nursing, CMC, Vellore	Internal, Nurse
Ms. Grace Rebekha	M.Sc., (Biostatistics)	Lecturer, Biostatistics, CMC, Vellore	Internal, Statistician
Mrs. Pattabiraman	BSc, DSSA	Social Worker, Vellore	External, Lay Person
Dr. Anand Zachariah	MBBS, PhD	Professor, Medicine, CMC, Vellore	Internal, Clinician
Dr. Balamugesh	MBBS, MD(Int Med), DM, FCCP (USA)	Professor, Pulmonary Medicine, CMC, Vellore	Internal, Clinician

IRB Min No: 10361 [OBSERVE] dated 03.11.2016

3 of 4

Ethics Committee Blue, Office of Research, 1st Floor, Carman Block, Christian Medical College, Vellore, Tamil Nadu 63
Tel: 0416 – 2284294, 2284202 Fax: 0416 – 2262788, 2284481 E-mail: research@cmcvellore.ac.in

OFFICE OF RESEARCH
INSTITUTIONAL REVIEW BOARD (IRB)
CHRISTIAN MEDICAL COLLEGE, VELLORE, INDIA

J. Prashantham, M.A., M.A., Dr. Min (Clinical)
Director, Christian Counseling Center,
Vice-Chairperson, Ethics Committee.

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Chairperson, Research Committee & Principal

Dr. Biju George, M.B.B.S., MD., DM.,
Deputy Chairperson,
Secretary, Ethics Committee, IRB
Additional Vice-Principal (Research)

Dr. Sneha Varkki	MBBS, DCH, DNB	Professor, Paediatrics, CMC, Vellore	Internal, Clinician
Mrs. Emily Daniel	MSc Nursing	Professor, Medical Surgical Nursing, CMC, Vellore	Internal, Nurse
Dr. Sathish Kumar	MBBS, MD, DCH	Professor, Child Health, CMC, Vellore	Internal, Clinician
Dr. Visalakshi. J	MPH, PhD	Lecturer, Biostatistics, CMC, Vellore	Internal, Statistician
Dr. Mathew Joseph	MBBS, MCH	Professor, Neurosurgery, CMC, Vellore	Internal, Clinician

We approve the project to be conducted as presented.

Kindly provide the total number of patients enrolled in your study and the total number of withdrawals for the study entitled: "Comparison of Obstetric & Neonatal outcomes in pregnant women with hypothyroidism and euthyroid pregnant women: a prospective cohort study"on a monthly basis. Please send copies of this to the Research Office (research@cmcvellore.ac.in).

Fluid Grant Allocation:

A sum of 81,250/- INR (Rupees Eighty One Thousand Two hundred and fifty Only) will be granted for 24 Months.

Yours sincerely,

Dr. Biju George
Secretary (Ethics Committee)
Institutional Review Board

Dr. BIJU GEORGE
SECRETARY (ETHICS COMMITTEE)
Christian Medical College
VELLORE - 632 002.

IRB Min No: 10361 [OBSERVE] dated 03.11.2016

Ethics Committee Blue, Office of Research, 1st Floor, Carman Block, Christian Medical College, Vellore, Tamil Nadu 63
Tel: 0416 – 2284294, 2284202 Fax: 0416 – 2262788, 2284481 E-mail: research@cmcvellore.ac.in

STUDY PROFORMA

Name: **Age**: **H. NO**:

Address: **BMI (prepregancy):** **Mobile:**

Booked / Unbooked :

Obstetric Score:

Previous Obstetric History:

Any comorbid Medical conditions:

A. known case of Hypothyroidism: **Y/N**

If Yes, Overt/subclinical:

Duration of disease:

On Thyroxine: Y/N Dose: Mcg

Compliance: Y/N

TFTs at diagnosis:

S.TSH:

S. T4 Levels:

S.FTc:

B.Diagnosed during pregnancy: Y/N

GA at diagnosis: weeks

TFTs at diagnosis:

S.TSH :

S.T4 Levels:

S.FTc:

Reason for ordering TFTs in pregnancy:

1) Symptomatic

2) Increased BMI (>40 Kg/m2)

3) Recurrent 1st Trimester miscarriages

4) Infertility

5) Family history of Thyroid disorder

6) History of head or neck radiation

7) Coming from area of Iodine deficiency

8) Type 1 Diabetes

Thyroxine started: Y/N Dose: Mcg

Repeat TFTs:

C. Alteration in dose of medications: Y/N

Dosage at 1st Trimester

2nd Trimester

3rd Trimester

D. Maternal complications: Y/N.

If yes, specify:

a)Miscarriages

b)Gestational HTN/ Preeclampsia

c) Preterm labor

d) Anemia

e)Gestational DM

f)Antepartum / Postpartum hemorrhage

g)Others

Any intervention done for above complications: Y/N

Specify

E. Labor and delivery details.

GA at delivery:

Preterm Labor:

Mode of delivery: **NVD / Instrumental delivery/ LSCS**

If LSCS/ instrumental delivery, indication:

Postpartum Haemorrhage : Y/N

F. Neonatal Outcome

Gestational age

Gender:

APGAR score:

Birth weight at delivery:

Cord blood TSH levels:

G. Neonatal complications:

Fetal growth restriction:

Intrauterine demise:

Admission to Neonatal ICU :

Neonatal Jaundice

Neonatal Death with cause

Data Entry Sheet

SLNO	IDNO	GROUP	AGE	age_r	BMI	bmi_r	CHECKUP	OBSTETRI	PREOBST	deleteCOMORBID	HT	DM	BA	HYPOTHY	HYPOYES	DURATION	THYROXIN	THYXODOS	COMPLIAN	STSH	ST4	SFTC	DIAGPREG	GADIAG	GA_diag_r
8		1	25	2	28.60	2	1	2	1	2			0	2									1	20.0	2
114		1	25	2	24.60	1	1	2	5	2			0	2			2						1	12.0	1
2		1	24	1	38.40	4	1	1	4	2			0	2									1	8.0	1
7		1	27	2	23.90	1	1	1	4	2			0	2									1	4.0	1
10		1	26	2	25.00	2	1	1	4	2			0	2									1	12.0	1
11		1	23	1	28.00	2	1	1	4	2			0	2			2		2				1	12.0	1
15		1	31	3	32.40	3	1	2	2	2			0	2									1	12.0	1
19		1	24	1	24.00	1	1	1	4	2			0	2									1	12.0	1
25		1	27	2	23.40	1	1	1	4	2			0	2			2						1	4.0	1
28		1	25	2	36.10	4	1	2	1	2			0	2			2						1	8.0	1
30		1	28	2	30.80	2	1	1	4	2			0	2			2						1	12.0	1
32		1	30	3	22.10	1	1	1	4	2			0	2			2						1	12.0	1
33		1	31	3	28.60	2	1	1	4	2			0	2			2						1	10.0	1
34		1	28	2	26.10	2	1	2	5	1			0	2			2						1	4.0	1
35		1	32	3	28.40	2	1	2	3	2			0	2			2						1	4.0	1
37		1	22	1	22.20	1	1	1	4	2			0	2			2						1	12.0	1
40		1	26	2	27.30	2	1	2	1	2			0	2			2						1	8.0	1
41		1	26	2	23.90	1	1	1	4	2			0	2			2						1	12.0	1
47		1	31	3	24.30	1	1	2	1	2			0	2			2						1	16.0	2
51		1	30	3	28.60	2	1	2	3	2			0	2			2						1	18.0	2
52		1	25	2	29.30	2	1	1	4	2			0	2									1	4.0	1
53		1	22	1	18.40	1	1	1	4	2			0	2			2						1	19.0	2
54		1	23	1	19.50	1	1	1	4	2			0	2			2						1	4.0	1
60		1	27	2	30.30	2	1	1	4	2			0	2			2						1	10.0	1
62		1	21	1	26.50	2	1	1	4	2			0	2			2						1	12.0	1
63		1	29	2	25.00	2	1	2	1	2			0	2			2						1	12.0	1
64		1	26	2	30.10	2	1	1	4	2			0	2			2						1	8.0	1
65		1	25	2	28.00	2	1	2	1	2			0	2			2						1	12.0	1
66		1	22	1	24.40	1	1	1	4	2			0	2			2						1	16.0	2
67		1	26	2	28.30	2	1	2	3	2			0	2			2						1	8.0	1
68		1	31	3	29.40	2	1	1	4	2			0	2			2						1	12.0	1
69		1	28	2	40.20	5	1	2	1	2			0	2			2						1	16.0	2
70		1	25	2	24.80	1	1	1	4	2			0	2			2						1	4.0	1
71		1	19	0	20.80	1	1	1	4	2			0	2			2						1	4.0	1
72		1	22	1	30.00	2	1	1	4	2			0	2			2						1	12.0	1
74		1	35	4	28.30	2	1	2	1	2			0	2			2						1	16.0	2
75		1	28	2	25.70	2	1	1	4	2			0	2			2						1	16.0	2
76		1	22	1	40.20	5	1	2	1	2			0	2			2						1	16.0	2
77		1	28	2	21.00	1	1	2	5	2			0	2			2						1	20.0	2
79		1	37	4	26.50	2	1	2	5	2			0	2			2						1	12.0	1
81		1	26	2	26.70	2	1	1	4	2			0	2			2						1	12.0	1
84		1	29	2	31.60	3	1	1	2	2			0	2			2						1	12.0	1
87		1	31	3	32.00	3	1	2	3	2			0	2			2						1	12.0	1
89		1	22	1	29.20	2	1	1	4	2			0	2			2						1	12.0	1
91		1	23	1	30.50	2	1	2	1	2			0	2			2						1	12.0	1
94		1	28	2	27.00	2	1	2	3	2			0	2			2						1	12.0	1
99		1	23	1	28.59	2	1	1	4	2			0	2			2						1	12.0	1
105		1	29	2	29.80	2	1	1	4	2			0	2			2						1	16.0	2
106		1	31	3	25.40	2	1	1	4	2			0	2			2						1	19.0	2
110		1	23	1	36.20	4	1	1	4	2			0	2			2						1	20.0	2
112		1	19	0	20.70	1	1	1	4	2			0	2			2						1	10.0	1
113		1	32	3	27.50	2	1	2	3	2			0	2			2						1	16.0	2
115		1	27	2	28.00	2	1	2	3	2			0	2			2						1	12.0	1
117		1	24	1	28.70	2	1	2	3	2			0	2			2						1	8.0	1
118		1	35	4	14.80	1	1	1	4	2			0	2			2						1	19.0	2
119		1	24	1	27.00	2	1	1	4	2			0	2			2						1	12.0	1
125		1	28	2	28.40	2	1	2	1	2			0	2			2						1	12.0	1
161	36	2	20	1	26.10	2	1	1	4	2			0	2			2						1	20.0	2
168	43	2	20	1	29.10	2	1	1	4	2			0	2			2						1	20.0	2

ga_diag_r1	PREGSTSH	PREGST4	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX1	THYROXDO	REPSTSH	REPST4	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r2	PREGSTSH	PREGST5	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX2	THYROXDO	REPSTSH	REPST5	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r3	PREGSTSH	PREGST6	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX3	THYROXDO	REPSTSH	REPST6	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r4	PREGSTSH	PREGST7	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX4	THYROXDO	REPSTSH	REPST7	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r5	PREGSTSH	PREGST8	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX5	THYROXDO	REPSTSH	REPST8	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r6	PREGSTSH	PREGST9	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX6	THYROXDO	REPSTSH	REPST9	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r7	PREGSTSH	PREGST1(	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX7	THYROXDO	REPSTSH	REPST1(	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r8	PREGSTSH	PREGST11	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX8	THYROXDO	REPSTSH	REPST11	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r9	PREGSTSH	PREGST12	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX9	THYROXDO	REPSTSH	REPST12	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r10	PREGSTSH	PREGST13	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX10	THYROXDO	REPSTSH	REPST13	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r11	PREGSTSH	PREGST14	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX11	THYROXDO	REPSTSH	REPST14	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r12	PREGSTSH	PREGST15	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX12	THYROXDO	REPSTSH	REPST15	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r13	PREGSTSH	PREGST16	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX13	THYROXDO	REPSTSH	REPST16	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r14	PREGSTSH	PREGST17	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX14	THYROXDO	REPSTSH	REPST17	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r15	PREGSTSH	PREGST18	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX15	THYROXDO	REPSTSH	REPST18	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r16	PREGSTSH	PREGST19	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX16	THYROXDO	REPSTSH	REPST19	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r17	PREGSTSH	PREGST2(	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX17	THYROXDO	REPSTSH	REPST2(	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r18	PREGSTSH	PREGST21	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX18	THYROXDO	REPSTSH	REPST21	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r19	PREGSTSH	PREGST22	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX19	THYROXDO	REPSTSH	REPST22	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r20	PREGSTSH	PREGST23	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX20	THYROXDO	REPSTSH	REPST23	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r21	PREGSTSH	PREGST24	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX21	THYROXDO	REPSTSH	REPST24	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r22	PREGSTSH	PREGST25	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX22	THYROXDO	REPSTSH	REPST25	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r23	PREGSTSH	PREGST26	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX23	THYROXDO	REPSTSH	REPST26	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r24	PREGSTSH	PREGST27	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX24	THYROXDO	REPSTSH	REPST27	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r25	PREGSTSH	PREGST28	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX25	THYROXDO	REPSTSH	REPST28	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r26	PREGSTSH	PREGST29	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX26	THYROXDO	REPSTSH	REPST29	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r27	PREGSTSH	PREGST3(	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX27	THYROXDO	REPSTSH	REPST3(	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r28	PREGSTSH	PREGST31	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX28	THYROXDO	REPSTSH	REPST31	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r29	PREGSTSH	PREGST32	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX29	THYROXDO	REPSTSH	REPST32	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r30	PREGSTSH	PREGST33	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX30	THYROXDO	REPSTSH	REPST33	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r31	PREGSTSH	PREGST34	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX31	THYROXDO	REPSTSH	REPST34	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r32	PREGSTSH	PREGST35	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX32	THYROXDO	REPSTSH	REPST35	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r33	PREGSTSH	PREGST36	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX33	THYROXDO	REPSTSH	REPST36	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r34	PREGSTSH	PREGST37	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX34	THYROXDO	REPSTSH	REPST37	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r35	PREGSTSH	PREGST38	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX35	THYROXDO	REPSTSH	REPST38	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r36	PREGSTSH	PREGST39	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX36	THYROXDO	REPSTSH	REPST39	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r37	PREGSTSH	PREGST4(	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX37	THYROXDO	REPSTSH	REPST4(	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r38	PREGSTSH	PREGST41	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX38	THYROXDO	REPSTSH	REPST41	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r39	PREGSTSH	PREGST42	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX39	THYROXDO	REPSTSH	REPST42	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r40	PREGSTSH	PREGST43	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX44(	THYROXDO	REPSTSH	REPST43	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r41	PREGSTSH	PREGST44	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX41	THYROXDO	REPSTSH	REPST44	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r42	PREGSTSH	PREGST45	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX42	THYROXDO	REPSTSH	REPST45	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r43	PREGSTSH	PREGST46	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX43	THYROXDO	REPSTSH	REPST46	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r44	PREGSTSH	PREGST47	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX44	THYROXDO	REPSTSH	REPST47	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r45	PREGSTSH	PREGST48	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX45	THYROXDO	REPSTSH	REPST48	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r46	PREGSTSH	PREGST49	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX46	THYROXDO	REPSTSH	REPST49	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r47	PREGSTSH	PREGST5(	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX47	THYROXDO	REPSTSH	REPST5(	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r48	PREGSTSH	PREGST51	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX48	THYROXDO	REPSTSH	REPST51	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r49	PREGSTSH	PREGST52	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX49	THYROXDO	REPSTSH	REPST52	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r50	PREGSTSH	PREGST53	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX5(	THYROXDO	REPSTSH	REPST53	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r51	PREGSTSH	PREGST54	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX51	THYROXDO	REPSTSH	REPST54	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r52	PREGSTSH	PREGST55	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX52	THYROXDO	REPSTSH	REPST55	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r53	PREGSTSH	PREGST56	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX53	THYROXDO	REPSTSH	REPST56	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r54	PREGSTSH	PREGST57	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX54	THYROXDO	REPSTSH	REPST57	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r55	PREGSTSH	PREGST58	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX55	THYROXDO	REPSTSH	REPST58	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r56	PREGSTSH	PREGST59	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX56	THYROXDO	REPSTSH	REPST59	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r57	PREGSTSH	PREGST6(	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX57	THYROXDO	REPSTSH	REPST6(	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r58	PREGSTSH	PREGST61	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX58	THYROXDO	REPSTSH	REPST61	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r59	PREGSTSH	PREGST62	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX59	THYROXDO	REPSTSH	REPST62	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN
ga_diag_r60	PREGSTSH	PREGST63	PREGSFTC	REASON	SYMPTOM	INCRESEB	TRIMESTE	INFERTIL	THYROID	HEADNECK	IODINE	DIABTYPE	THYROX6(	THYROXDO	REPSTSH	REPST63	REPSFTC	MEDICAT	TRIMEST1	TRIMEST2	TRIMEST3	MATERNAL	MISCARRI	GASTAHTN	GHTN

PE	PRELABOR	ANEMIA	GESTADM	ANTEPART	OTHERS	INTERVEN	SPECIFY	GADELIVE	PREELABO	DELIMODE	LSCS	LSCSINST	POSTPART	GESTAGE	GA	BABYGEND	APGARSCO	APGAR2	BIRTHWT	bwt	CORDBLOO	cord_ish	NEONAT	FETALGRO	INTRAUTE
2	2	2	1	2		1	MNT	37.1	2	3	1	NRFS	2	37.1	2	2	9	10	2.540	2	7.25	2	1	1	2
2	2	2	2	2		2		39.1	2	1	2		2	39.1	2	1	9	10	2.460	1	18.82	2	1	1	2
2	2	2	2	2		1	BP MONITORING	39.4	2	1	2		2	39.4	2	1	9	10	3.310	2	13.52	2	1	2	2
2	2	2	1	2		1	MNT	39.3	2	2	2	NRFS	2	39.3	2	1	9	10	2.670	2	17.74	2	2	2	2
2	2	2	2	2		2		39.4	2	2	2	NRFS	2	39.4	2	2	9	10	2.650	2	6.72	2	2	2	2
2	2	2	2	2		2		40.3	2	2	2	CAT 2 NRFS	2	40.3	2	2	9	10	3.000	2	8.07	2	2	2	2
2	2	2	1	2		1	MNT	38.6	2	3	1	PREVIOUS LSCS	2	38.6	2	1	9	10	3.420	2	4.26	2	2	2	2
1	1	2	2	2		1	ANTIHYPERTENSIVE DRUGS, MGSO4	35.4	1	3	1	NRFS	2	35.4	1	1	9	9	1.120	1	16.23	2	1	1	2
2	2	2	2	2		2		40.3	2	3	1	NRFS	2	40.3	2	2	9	9	3.460	2	4.37	2	2	2	2
2	2	2	2	2		1	ANTIHYPERTENSIVE DRUGS	38.2	2	1	2		2	38.2	2	1	9	10	3.120	2	17.50	2	2	2	2
2	2	2	2	2		2		39.1	2	2	2	NRFS	2	39.1	2	1	9	10	3.160	2	4.02	2	2	2	2
2	2	2	1	2		1	MNT	39.1	2	1	2		2	39.1	2	2	9	10	3.070	2	5.36	2	1	2	2
2	2	2	1	2		1	MNT	38.4	2	2	2	NRFS	2	38.4	2	2	9	10	2.540	2	4.51	2	1	1	2
2	2	2	2	2		2		38.4	2	1	2		2	38.4	2	1	9	10	3.320	2	6.17	2	2	2	2
1	2	2	2	2		1	BP MONITORING	36.6	1	3	1	PREVIOUS LSCS, WITH BREECH	2	36.6	1	1	9	10	2.860	2	5.79	2	2	2	2
2	2	1	2	2		1	ORAL IRON	39.6	2	2	2	CAT 2 NRFS	2	39.6	2	1	9	10	2.000	1	5.94	2	1	1	2
2	2	1	2	2		1	ORAL IRON	38.3	2	1	2		2	38.3	2	2	9	10	2.800	2	5.79	2	1	2	2
2	2	1	1	2		1	MNT, ORAL IRON	38.4	2	3	1	NRFS, THINMSAF	2	38.4	2	1	9	10	2.540	2	4.90	2	1	2	2
2	2	1	2	2		1	ORAL IRON	39.0	2	1	2		2	39.0	2	1	9	10	2.700	2	6.11	2	2	2	2
2	2	2	2	2		2		39.3	2	1	2		2	39.3	2	1	9	10	2.960	2	5.43	2	2	2	2
2	2	1	2	2		1	BP MONITORING, ORAL IRON	38.5	2	2	2	NRFS	2	38.5	2	2	9	10	2.800	2	24.69	2	1	2	2
2	2	2	2	2		2		38.0	2	2	2	NRFS	2	38.0	2	2	9	10	2.030	1	4.27	2	1	1	2
2	2	1	2	2		1	ORAL IRON	39.2	2	1	2		2	39.2	2	2	9	10	2.800	2	9.63	2	2	2	2
2	2	2	2	2		2		38.6	2	1	2		2	38.6	2	1	9	10	2.830	2	4.83	2	2	2	2
2	2	2	2	2		2		38.5	2	1	2		2	38.5	2	2	9	10	3.330	2	5.05	2	2	2	2
2	2	1	2	2		1	ORAL IRON	38.1	2	1	2		2	38.1	2	2	9	10	2.680	2	10.10	2	2	2	2
2	2	2	2	2		2		38.4	2	3	1	CAT 2 NRFS	2	38.4	2	2	9	10	2.300	1	7.72	2	1	1	2
2	2	2	2	2		2		39.1	2	3	1	FAILED INDUCTION	2	39.1	2	2	9	9	3.220	2	15.23	2	2	2	2
1	2	2	2	2		1	MGSO4	40.1	2	3	1	ARREST OF DESCENT	2	40.1	2	2	9	9	3.600	2	6.22	2	2	2	2
2	2	2	1	2		1	BP MONITORING, OHA + INSULINE	38.5	2	3	1	PREVIOUS LSCS	2	38.5	2	2	9	9	3.420	2	5.52	2	2	2	2
2	2	2	2	2		2		39.3	2	2	2	CAT 2 NRFS	2	39.3	2	2	9	10	2.790	2	4.18	2	2	2	2
2	2	2	2	2		2		39.4	2	1	2		2	39.4	2	2	9	10	2.900	2	17.86	2	2	2	2
2	2	2	1	2		1	MNT	39.4	2	1	2		2	39.4	2	1	9	9	3.680	2	7.00	2	2	2	2
2	2	2	2	2		2		37.2	2	1	2		2	37.2	2	2	9	10	6.690	2	2.11	2	1	1	2
2	2	2	2	2		1	BP MONITORING	38.2	2	2	2	PROLONG 2ND STAGE	2	38.2	2	1	9	10	3.780	2	5.57	2	2	2	2
2	1	2	1	2		1	OHA + MNT	36.4	1	1	2		2	36.4	1	2	9	10	2.360	1	6.85	2	2	2	2
2	2	2	2	2		2		38.6	2	1	2		2	38.6	2	2	9	10	2.980	2	10.71	2	2	2	2
1	2	2	2	2		1	ANTIHYPERTENSIVE DRUGS	36.3	1	2	2	NRFS	2	36.3	1	1	9	10	2.800	2	29.62	1	2	2	2
2	2	1	2	2		1	ORAL IRON	38.0	2	3	1	PREVIOUS LSCS NOT WILLING FOR VBAC	2	38.0	2	2	9	10	3.000	2	3.30	2	2	2	2
2	2	1	2	2		1	ANEMIA RX WITH ORAL IRON, GHTN ON BP MONITORING	39.1	2	3	1		2	39.1	2	1	9	10	3.120	2	2.64	2	2	2	2
2	2	2	1	2		1	BP MONITORING	39.0	2	1	2		2	39.0	2	2	9	10	3.160	2	7.30	2	2	2	2
2	2	1	2	2		1	ORAL IRON	38.2	2	3	1	PROTRACTED DILATATION	2	38.2	2	2	9	10	2.460	1	11.23	2	1	1	2
2	2	2	2	2		2		39.5	2	3	1	PREVIOUS LSCS	2	39.5	2	1	9	9	3.680	2	8.12	2	2	2	2
2	2	2	2	2		2		39.5	2	3	1	CAT 2 NRFS	2	39.5	2	2	9	10	3.360	2	6.83	2	2	2	2
2	2	2	2	2		2		38.4	2	1	2		2	38.4	2	1	9	10	3.140	2	10.44	2	2	2	2
2	2	2	1	2		1	MNT	38.4	2	3	1	DOUBTFUL SCAR INTEGRITY	2	38.4	2	1	9	9	3.140	2	4.98	2	2	2	2
2	2	1	2	2		1	BP MONITORING, ORAL IRON	39.0	2	2	2	NRFS	2	39.0	2	2	9	10	2.800	2	24.69	2	1	2	2
2	2	1	2	2		1	ORAL IRON	37.1	2	2	2	NRFS	2	37.1	2	2	9	10	2.270	1	13.23	2	1	1	2
2	2	2	2	2		2		38.3	2	1	2		2	38.3	2	1	9	10	2.770	2	13.18	2	2	2	2
1	2	1	2	2		1	ORAL IRON, MGSO4 DELIVERY	38.0	2	3	1	SPE WITH DETERORATION MATERNAL CONDITION	2	38.0	2	1	9	10	2.600	2	4.14	2	1	2	2
2	2	2	2	2		2		39.4	2	1	2		2	39.4	2	1	9	10	3.550	2	3.54	2	2	2	2
2	2	2	1	2		1	OHA	39.0	2	1	2		1	39.0	2	2	9	10	3.100	2	9.25	2	2	2	2
2	2	1	2	2		1	ORAL IRON	38.3	2	2	2	NRFS	2	38.3	2	1	7	8	3.250	2	10.34	2	2	2	2
2	2	2	2	2		2		40.2	2	1	2		2	40.2	2	2	9	10	2.850	2	7.12	2	2	2	2
2	1	2	2	2		1	ANTIHYPERTENSIVE DRUGS	33.4	1	3	1	SEVEN PRE ECLAMPSIA	2	33.4	1	2	9	10	1.420	1	8.56	2	1	2	2
2	2	1	2	2		1	ORAL IRON	39.6	2	3	1	NRFS	2	39.6	2	1	9	9	2.560	2	14.13	2	1	2	2
2	2	2	2	2		2		40.3	2	2	2	NRFS		40.3	2	1	7	9	3.440	2	9.32	2	1	2	2
2	2	1	2	2		1	ORAL IRON	41.1	2	1	2		2	41.1	2	2	9	10	2.560	2	12.71	2	2	2	2
1	2	2	2	2		1	ANTIHYPERTENSIVE DRUGS	39.3	2	3	1	NRFS	2	39.3	2	1	9	10	2.800	2	8.42	2	2	2	2

NEONATIC	Preterm	LBW	Jaundice	Asphyxia	Renalpelvisdilataion	Sepsis	Hypogylcemia	TTN	PDA	Microcephaly	TSH	NEONATJA	NEONATDE	DEATHYES	filter_$
2	2	2	2	2	2	2	2	2	2	2	2	2	2		1
2	2	2	2	2	2	2	2	2	2	2	2	2	2		1
1	2	2	2	2	2	2	2	2	2	2	2	2	2		1
2	2	2	2	2	2	2	2	2	2	2	2	2	2		1
2	2	2	2	2	2	2	2	2	2	2	2	2	2		1
2	2	2	2	2	2	2	2	2	2	2	2	2	2		1
2	2	2	2	2	2	2	2	2	2	2	2	2	2		1
1	1	2	2	2	2	2	2	2	2	2	2	2	2		1
2	2	2	2	2	2	2	2	2	2	2	2	2	2		1
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2	2	2	2	2	1	2	2	2	2	2	2	2	2		1
1	2	2	2	2	2	2	2	2	2	2	2	2	2		1
2	2	2	2	2	2	2	2	2	2	2	2	2	2		1
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1	2	2	1	2	2	2	2	2	2	2	2	2	2		1
1	2	2	2	2	2	2	2	2	2	1	2	2	2		1
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2	2	2	2	2	2	2	2	2	2	2	2	2	2		1
2	2	2	2	2	2	2	2	2	2	2	2	2	2		1
2	2	2	2	2	2	2	2	2	2	2	2	2	2		1
2	2	2	2	2	2	2	2	2	2	2	2	2	2		1
2	2	2	2	2	2	2	2	2	2	2	2	2	2		1
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2	2	2	2	2	2	2	2	2	2	2	2	2	2		1
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1	2	2	2	2	2	2	2	2	2	2	1	2	2		1
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1	2	2	2	2	2	2	1	2	2	2	2	2	2		1
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1	2	2	2	2	2	2	2	1	2	2	2	2	2		1
1	2	2	2	2	2	2	2	1	2	2	2	2	2		1
1	2	2	2	1	2	2	2	2	2	2	2	2	2		1
2	2	2	2	2	2	2	2	2	2	2	2	2	2		0
2	2	2	2	2	2	2	2	2	2	2	2	2	2		0

16		1	28	2	30.80	2	1	1	4	1		0	1	2	2.00	1	50	1	7.500	10.2	1.15	2		0
18		1	25	2	26.60	2	1	1	4	1		0	1	2	1.06	1	50	1	9.600			2		0
39		1	30	3	28.20	2	1	1	4	1		0	1	2	10.00	1	25	1	8.500	7.2	1.01	2		0
44		1	32	3	30.80	2	1	2	3	1		0	1	2	1.00	1	50	1	6.150			2		0
58		1	24	1	25.00	2	1	1	4	1		0	1	2	1.06	1	25	1	5.600			2		0
59		1	26	2	28.20	2	1	2	3	1		0	1	2	1.06	1	50	1	8.000	8.3	1.12	2		0
61		1	30	3	24.50	1	1	2	1	1		0	1	2	1.06	1	75	1	9.390			2		0
82		1	33	3	30.20	2	1	1	4	1		0	1	2	2.00	1	50	1	8.283	8.5	1.15	2		0
104		1	28	2	25.60	2	1	2	3	1		0	1	2	3.00	1	50	1	8.230	8.0	1.10	2		0
108		1	29	2	23.00	1	1	2	1	1		0	1	2	1.06	1	50	1				2		0
122		1	41	5	32.00	3	1	1	4	1		0	1	2	1.06	1	50	1	9.000	7.5	1.15	2		0
124		1	25	2	22.30	1	1	1	4			0	1	2	2.00	1	50	1	9.360	10.2	1.23	2		0
1		1	27	2	33.30	3	1	1	4	1		0	1	1	13.00	1	200					2		0
3		1	22	1	31.60	3	1	1	4	1		0	1	1	1.06	1	50	1				2		0
4		1	30	3	27.50	2	1	1	4	1		0	1	1	7.00	1	150	1				2		0
5		1	31	3	23.00	1	1	2	3	1		0	1	1	0.08	1	100		16.520			2		0
6		1	21	1	35.30	3	1	1	4	1		0	1	1		1	50	1	11.360	1.2		2		0
9		1	25	2	36.20	4	1	2	3	1		0	1	1		1	75	1	74.933	8.9	0.67	2		0
12		1	29	2	28.00	2	1	1	4	1		0	1	1	2.06	1	125	1				2		0
13		1	23	1	21.40	1	1	2	5	1		0	1	1	1.00	1	100	1	12.000			2		0
17		1	35	4	30.50	2	1	1	4	1		0	1	1	4.00	1	125	1				2		0
20		1	28	2	21.20	1	1	2	1	1		0	1	1	1.06	1	50	1	11.000			2		0
21		1	23	1	25.50	2	1	2	5	1		0	1	1	1.00	1	75	1	10.000			2		0
22		1	29	2	23.30	1	1	1	4	1		0	1	1	10.00	1	100	1				2		0
23		1	29	2	32.50	3	1	1	4	1		0	1	1	12.00	1	75	1	15.200	12.3	1.33	2		0
26		1	33	3	27.50	2	2	2	5	1		0	1	1	3.00	1	50	1	10.010			2		0
27		1	27	2	25.00	2	1	2	3	1		0	1	1	2.00	1	100	1	12.500			2		0
29		1	22	1	32.50	3	1	1	4	1		0	1	1	12.00	1	100	1	20.600			2		0
31		1	29	2	33.50	3	1	1	4	1		0	1	1	2.00	1	75	1	40.507	3.2	0.60	2		0
38		1	30	3	31.20	3	1	2	3	1		0	1	1	2.00	1	200		160.000			2		0
43		1	28	2	19.90	1	1	1	4	1		0	1	1	3.00	1	75	1	12.000			2		0
45		1	29	2	34.20	3	1	2	3	1		0	1	1	5.00	1	50	1	10.520			2		0
46		1	28	2	30.20	2	2	2	2	1		0	1	1	3.00	1	100	1	19.127			2		0
48		1	30	3	28.30	2	1	2	5	1		0	1	1	6.00	1	150	1	23.400			2		0
49		1	34	3	30.20	2	1	2	3	1		0	1	1	6.00	1	100		20.500			2		0
55		1	32	3	26.70	2	1	2	5	1		0	1	1	5.00	1	75	1	9.500	10.5	1.02	2		0
56		1	26	2	27.30	2	1	1	4	1		0	1	1	3.00	1	100	1	23.000	1.8	0.28	2		0
57		1	28	2	32.40	3	1	2	3	1		0	1	1	2.00	1	75	1	10.520			2		0
73		1	33	3	27.00	2	1	2	5	1		0	1	1	2.00	1	100	1	14.640	10.6	1.23	2		0
78		1	32	3	28.60	2	1	2	3	1		0	1	1	6.00	1	100	1	22.300			2		0
80		1	28	2	28.10	2	1	1	4	1		0	1	1	7.00	1	125	1	20.000			2		0
85		1	27	2	29.50	2	1	1	4	1		0	1	1	5.00	1	75	1				2		0
86		1	29	2	28.20	2	1	2	5	1		0	1	1	4.00	1	100	1	11.200			2		0
88		1	29	2	38.40	4	1	2	3	1		0	1	1	5.00	1	50	1				2		0
90		1	28	2	24.70	1	1	2	3	1		0	1	1	14.00	1	50	1				2		0
92		1	28	2	32.40	3	1	1	4	1		0	1	1	11.00	1	75		14.423	12.2	1.18	2		0
93		1	24	1	24.70	1	1	2	1	1		0	1	1	2.00	1	50	1				2		0
95		1	31	3	29.10	2	1	1	4	1		0	1	1	3.00	1	150	1				2		0
96		1	25	2	28.50	2	1	1	4	1		0	1	1	2.00	1	50	1	11.400			2		0
97		1	23	1	23.80	1	1	1	4	1		0	1	1	1.06	1	100	1	27.000			2		0
98		1	36	4	32.40	3	1	1	4	1		0	1	1	6.00	1	150	1	20.400			2		0
100		1	27	2	23.90	1	1	2	5	1		0	1	1	6.00	1	50	1	11.887	12.5	0.92	2		0
101		1	32	3	27.50	2	1	1	4	1		0	1	1	5.00	1	25	1	115.000			2		0
102		1	20	1	30.00	2	1	2	5	1		0	1	1	4.00	1	100	1				2		0
103		1	34	3	26.00	2	1	2	5	1		0	1	1	7.00	1	50	1	100.000			2		0
107		1	27	2	26.00	2	1	1	4	1		0	1	1	2.00	1	100	1	12.500	10.9	1.23	2		0
109		1	27	2	25.00	2	1	2	3	1		0	1	1	3.00	1	150	1	92.999	1.9	0.49	2		0
111		1	20	1	30.70	2	1	1	4	1		0	1	1	1.00	1	75					2		0
116		1	22	1	29.50	2	1	2	3	1		0	1	1	10.00	1	200		217.000	7.2	0.54	2		0
120		1	32	3	34.20	3	1	2	3	1		0	1	1	3.00	1	100	1	23.200			2		0

1.00				1	2	2	2	1	2	2	2	2			1.660	15.20	1.22	1	75			2	2	2	2
1.00				2	0	0		0					2		4.880			1		75		2	2	2	2
1.00				1	2	2	2	1	2	2	2	2	2		0.990			2				1	2	2	2
1.00				2	0	0		0					1		1.432	13.40	1.10	2				2	2	2	2
1.00				1	2	2	2	1	2	2	2	2	2		1.672			2				2	2	2	2
1.00				2	0	0		0					2		4.617	14.00	1.07	1	75			1	2	2	2
1.00				2	0	0		0					2		6.999			1	100			1	2	2	2
1.00				2	0	0		0					2		1.312			2				1	2	2	2
1.00				2	0	0		0					2		1.060	14.80	1.12	2				2	2	2	2
1.00				1	2	2	2	1	2	2	2	2	2		2.301			2				1	2	1	2
1.00				1	2	2	2	1	2	2	2	2	2		10.070			1	100			1	2	2	2
1.00				1	2	2	2	1	2	2	2	2	2		3.489			1		75		1	2	2	2
1.00				2	0	0		0							2.600			2				2	2	2	2
1.00				2	0	0		0							4.100	11.20	1.20	1		125		1	2	2	2
1.00				2	0	0		0							14.143	13.20	1.19	1		200		1	2	2	2
1.00				2	0	0		0							1.970			2				1	2	2	2
1.00				2	0	0		0					1	75	2.537			1	75			1	2	1	1
1.00				2	0	0		0							6.876			1		100		2	2	2	2
1.00				1	2	2	2	1	2	2	2	2			0.827			2				2	2	2	2
1.00				2	0	0		0							2.300			1	50			2	2	2	2
1.00				2	0	0		0							1.833	16.60	1.16	1	150			2	2	2	2
1.00				2	0	0		0							1.900			1		75		2	2	2	2
1.00				2	0	0		0					2					2				2	2	2	2
1.00				2	0	0		0					2		29.000			2				1	2	2	2
1.00				2	0	0		0					2		1.814			2				1	2	2	2
1.00				2	0	0		0					2		1.955	12.50	1.22	2				1	2	2	2
1.00				2	0	0		0					2		1.940			2				2	2	2	2
1.00				2	0	0		0					2		0.353			1		150		2	2	2	2
1.00				2	0	0		0					2		0.675			1	100			2	2	2	2
1.00				2	0	0		0					2		2.076	14.70	1.23	2				2	2	2	2
1.00				2	0	0		0					2		2.668	8.40	1.08	1	100			1	2	2	2
1.00				2	0	0		0					2					2				2	2	2	2
1.00				2	0	0		0					2		0.093	15.40	1.41	1		150		1	2	2	2
1.00				2	0	0		0					2		2.930			2				1	2	2	2
1.00				2	0	0		0					2		0.045			2				1	2	2	2
1.00	0.560	14.2	1.04	2	0	0		0					2		0.560	14.20	1.04	2				2	2	2	2
1.00				2	0	0		0					2		4.414			1		150		1	2	2	2
1.00				2	0	0		0					2		1.051	15.70	1.04	1			150	2	2	2	2
1.00				2	0	0		0					2		4.882	6.40	0.97	1	125			2	2	2	1
1.00				2	0	0		0					2		3.282			2				2	2	2	2
1.00				2	0	0		0					2		0.848			1	150			2	2	2	2
1.00				2	0	0		0					2		2.652			1	100			1	2	1	1
1.00				2	0	0		0					2		2.900			2				1	2	2	2
1.00				2	0	0		0					2		1.801			2				1	2	2	2
1.00				2	0	0		0					2		2.300	10.90	0.97	2				1	2	2	2
1.00				2	0	0		0					2		5.834			1	100			1	2	2	2
1.00				2	0	0		0					2		2.500			2				1	2	2	2
1.00				2	0	0		0					2		1.400		1.18	2				1	2	1	1
1.00				2	0	0		0					2		8.600			1	100			1	2	2	2
1.00				2	0	0		0					2		3.491	9.00	1.28	1			175	1	2	2	2
1.00				2	0	0		0					2		1.684	16.20	1.29	2				2	2	2	2
1.00				2	0	0		0					2		3.217			1		100		1	2	2	2
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1.00				2	0	0		0					2		4.600	8.60		1	75			1	2	2	2
1.00				2	0	0		0					2		2.587	14.90	1.05	2				2	2	2	2
1.00				2	0	0		0					2		0.011			2				1	2	2	2
1.00				2	0	0		0					2		0.983			2				1	2	2	2
1.00				2	0	0		0					2		3.600			1	250			1	2	2	2
1.00				2	0	0		0					2		0.562			1		100		2	2	2	2

2	2	2	2	2		2		38.0	2	2	2	NRFS	2	38.0	2	2	9	10	2.890	2	33.09	1	2	2	2
2	2	2	2	2		1	BIWEEKLY MONITORING, IOL	39.5	2	3	1	NRFS	2	39.5	2	2	9	9	2.300	1	5.68	2	1	1	2
2	2	1	1	2		1	ORAL IRON	38.1	2	3	1	CAPERATORRY FOR RUPTURE UTERUS	1	38.1	2	1	0	1	3.150	2	5.30	2	1	2	2
2	2	2	2	2		2		39.2	2	1	2		2	39.2	2	1	9	10	2.890	2	6.99	2	2	2	2
2	2	2	2	2		2		39.2	2	3	1	NRFS	2	39.2	2	1	9	10	2.980	2	6.88	2	1	2	2
2	2	2	1	2		1	MNT	37.3	2	1	2		2	37.3	2	1	9	10	3.340	2	6.10	2	1	2	2
2	2	2	1	2		1	OHA	38.0	2	3	1	FAILED INDUCTION	2	38.0	2	2	9	10	2.600	2	18.82	2	2	2	2
2	2	1	2	2		1	ORAL IRON	39.0	2	3	1	NRFS	2	39.0	2	2	9	10	3.000	2	8.40	2	2	2	2
2	2	2	2	2		2		40.2		1	2		2	40.2	2	2	9	10	3.260	2	5.12	2	2	2	2
1	2	2	2	2		1	ANTIHYPERTENSIVE DRUGS, MGSO4	40.0	2	2	2	PROLONG 2ND STAGE	2	40.0	2	1	9	10	2.780	2	8.32	2	2	2	2
2	1	2	2	2		2		36.3	1	3	1	IUGR WITH ABNORMAL DOPPLER	2	36.3	1	2	9	9	1.700	1	6.89	2	1	1	2
2	1	2	2	2		2		36.6	1	1	2		2	36.6	1	1	9	10	2.130	1	5.94	2	1	1	2
2	2	2	2	2		2		40.1	2	2	2	CAT 2 NRFS	2	40.1	2	1	9	10	2.700	2	10.70	2	2	2	2
2	2	2	1	2		1	OHA	38.6	2	2	2	CAT 2 NRFS	2	38.6	2	1	9	9	3.100	2	7.93	2	2	2	2
2	2	2	1	2		1	OHA	38.5	2	1	2		2	38.5	2	2	9	10	2.700	2	6.95	2	1	2	2
2	2	2	1	2		1	MNT	39.0	2	1	2		2	39.0	2	1	9	10	3.490	2	7.19	2	1	2	2
2	2	1	1	2		1	MNT, BP MONITORING, ORAL	38.4	2	3	1	BREECH WITH ECV	2	38.4	2	1	9	10	4.160	2	4.11	2	2	2	2
2	2	2	2	2		2		39.2	2	3	1	PREVIOUS LSCS WITH FAILED INDUCTION	2	39.2	2	1	9	9	2.920	2	3.74	2	2	2	2
2	2	2	2	2		2		40.3	2	3	1	UNFAVOURABLE CERVIX IVF PREGNANCY	2	40.3	2	1	9	10	2.920	2	5.93	2	2	2	2
2	2	2	2	2		2		40.1	2	3	1	NRFS	2	40.2	2	1	9	10	3.480	2	5.54	2	2	2	2
2	2	2	2	2		2		39.1	2	3	1	CAT 2 NRFS	2	39.1	2	2	9	10	3.100	2	6.59	2	2	2	2
2	2	2	2	2		2		40.0	2	3	1	ARREST OF DILATATION	2	40.1	2	2	9	10	3.060	2	4.23	2	2	2	2
2	2	2	2	2		2		40.2	2	1	2		2	40.2	2	1	9	10	3.410	2	20.65	2	2	2	2
2	2	2	1	2		1	MNT	38.4	2	2	2	CAT 2 NRFS	2	38.2	2	2	9	10	2.900	2	1.67	2	2	2	2
2	2	1	1	2		1	MNT, ORAL IRON	38.4	2	2	2	NRFS	2	38.4	2	2	9	10	2.960	2	1.67	2	2	2	2
2	1	1	1	2		1	MNT, ORAL IRON	35.3	2	1	2		2	35.3	1	2	9	10	2.840	2	10.77	2	2	2	2
2	2	2	2	2		2		39.2	2	1	2		2	39.2	2	1	9	10	3.520	2	4.10	2	2	2	2
2	2	2	2	2		2		39.2	2	3	1	CAT 2 NRFS	2	39.2	2	1	9	10	3.260	2	17.62	2	2	2	2
2	2	2	2	2		2		38.6		1	2			38.6	2	2	9	10	2.340	1	18.85	2	1	1	2
2	2	2	2	2		2		38.1	2	1	2		2	38.1	2	1	9	10	2.830	2	4.35	2	2	2	2
2	1	1	2	2	IUGR - 1.4KG AFI	1	ORAL IRON	34.3	1	3	1	FAILED INDUCTION	2	34.3	1	2	9	10	1.540	1	9.42	2	1	1	1
2	2	2	2	2		2		37.5	2	1	2		2	37.5	2	1	9	9	2.440	1	15.23	2	1	1	2
2	2	2	1	2		1	OHA	39.2	2	3	1	CORD PROLAPSE	2	39.2	2	1	9	10	3.280	2	4.33	2	2	2	2
2	2	1	2	2		1	ORAL IRON	40.1	2	1	2		2	40.1	2	2	9	10	3.500	2	4.46	2	2	2	2
2	2	2	1	2		1	OHA + INSULINE	38.6	2	1	2		2	38.6	2	1	9	10	2.740	2	13.97	2	1	2	2
2	2	2	2	2		2		39.0	2	1	2		2	39.0	2	2	9	10	2.950	2	7.76	2	2	2	2
2	2	1	1	2		1	MNT, ORAL IRON	40.0	2	2	2	CAT 2 NRFS	2	40.0	2	1	9	9	3.050	2	17.45	2	1	2	2
2	2	2	2	2		1		40.1	2	1	2		2	40.1	2	1	9	10	3.720	2	9.55	2	2	2	2
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2	2	2	2	2		2		39.0	2	1	2		2	39.0	2	2	9	10	3.280	2	6.25	2	2	2	2
2	2	2	2	2		2		39.2	2	1	2		2	39.2	2	1	9	10	3.712	2	7.12	2	2	2	2
2	2	2	1	2		1	MNT, BP MONITORING	37.2	2	3	1	BREECH WITH GHTN	2	37.2	2	1	9	10	3.060	2	7.15	2	2	2	2
2	2	1	2	2		1	ORAL IRON	40.1	2	1	2		2	40.1	2	2	9	10	3.500	2	4.46	2	2	2	2
2	2	2	1	2		1	OHA	39.3	2	1	2		2	39.3	2	1	9	10	2.950	2	67.58	1	1	2	2
2	2	2	1	2		1	MNT	40.5	2	1	2		2	40.5	2	2	9	10	3.440	2	8.81	2	2	2	2
2	2	1	2	2		1	ORAL IRON	38.3	2	3	1	ARREST OF DESCENT	2	38.3	2	2	9	10	3.960	2	3.31	2	1	2	2
2	2	2	1	2		2		37.4		1	2			37.3	2	2	9	10	2.630	2	21.65	2	2	2	2
2	2	1	1	2		1	GDM ON OHA, BP MONITORING, ORAL IRON	39.5	2	2	2	NRFS	2	39.5	2	2	9	10	2.900	2	14.08	2	2	2	2
2	2	2	1	2		1	MNT	39.5	2	3	1	CAT 2 NRFS	2	39.5	2	2	5	9	3.540	2	6.17	2	2	2	2
2	2	2	1	2		1	MNT	38.6	2	1	2		2	38.6	2	1	9	10	2.840	2	5.15	2	2	2	2
2	2	2	2	2		2		38.6	2	3	1	NRFS	2	38.6	2	1	9	9	2.820	2	5.68	2	2	2	2
2	2	1	2	2		1	ORAL IRON	39.4	2	2	2	PROLONG 2ND STAGE	2	39.4	2	2	9	9	3.240	2	6.67	2	2	2	2
2	2	1	2	2		1	ORAL IRON	39.0	2	3	1	CAT 2 NRFS	2	39.0	2	2	9	9	2.960	2	3.69	2	2	2	2
2	2	2	2	2		2		39.0	2	1	2		2	39.0	2	1	9	10	2.800	2	7.65	2	1	2	2
2	2	2	1	2		1	OHA	37.0	2	1	2		2	37.0	2	1	9	10	2.800	2	7.67	2	2	2	2
2	2	2	2	2		2		39.3	2	2	2	NRFS	2	39.3	2	2	9	10	2.820	2	22.22	2	2	2	2
2	2	1	2	2		1	ORAL IRON	37.4	2	3	1	PLACENTA PREVIA	2	37.4	2	2	9	10	2.840	2	3.09	2	2	2	2
2	2	2	1	2		1	OHA	33.6	1	3	1	FAILED INDUCTION FOR PPROM	2	33.6	1	1	9	10	2.400	1	8.57	2	1	2	2
2	2	1	1	2		1	MNT, ORAL IRON	38.4	2	1	2		2	38.4	2	2	9	10	2.470	1	4.65	2	2	2	2
2	2	2	2	2		2		39.5	2	1	2		2	39.5	2	2	9	10	3.160	2	8.32	2	2	2	2

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36		1	33	3	28.00	2	1	2	3	1		0	1	2	1.06	1	50	1	8.367	6.7	0.90	2		0
42		1	26	2	23.60	1	1	2	1	1		0	1	2	3.00	1	25	1				2		0
83		1	26	2	34.10	3	1	1	4	1		0	1	2	6.00	1	75	1				2		0
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123		1	29	2	36.50	4	1	2	1	2		0	2	2		1	25		5.200			2		0
126	1	2	26	2	26.50	2	1	2	5	2		0	2			2						2		0
127	2	2	19	0	40.50	5	1	2	1	1		0	2			2						2		0
128	3	2	21	1	28.20	2	1	1	4	2		0	2			2						2		0
129	4	2	28	2	24.50	1	1	1	4	2		0	2			2						2		0
130	5	2	20	1	23.60	1	1	1	4	2		0	2			2						2		0
131	6	2	18	0	20.30	1	1	1	4	2		0	2			2						2		0
132	7	2	30	3	17.20	1	1	2	3	2		0	2			2						2		0
133	8	2	23	1	21.50	1	1	1	4	2		0	2			2						2		0
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138	13	2	36	4	30.00	2	1	1	4	2		0	2			2						2		0
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141	16	2	24	1	20.50	1	1	1	4	2		0	2			2						2		0
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159	34	2	26	2	22.90	1	1	1	4	2		0	2			2						2		0
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162	37	2	22	1	23.00	1	1	2	3	2		0	2			2						2		0
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178	53	2	33	3	18.60	1	1	2	3	2		0	2			2						2		0
179	54	2	17	0	19.30	1	1	1	4	2		0	2			2						2		0

1.00				2	0	0		0					2		2.300			2				2	2	2	2										
1.00				2	0	0		0							1.298	15.70	1.05	2				1	2	1	1										
1.00				1	2	1	2	2	2	2	2	2	2		5.369			1		75		2	2	2	2										
1.00				2	0	0		0					2		2.221	12.70	1.03	1	75			2	2	2	2										
1.00				2	0	0		0					2		1.762	12.10	0.91	1		50		1	2	2	2										
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1.00				2	2	2	2	2	2	2	2	2	2					2				1	2	2	2										
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				2	0	0		0					2		2.350			2				2	2	2	2										
				2	0	0		0					2		2.300			2				1	2	2	2										
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2	2	1	2	2		1	BP MONITORING, ORAL IRON	39.3	2	3	1	CAT 3 NRFS	2	39.3	2	1	7	10	3.000	2	6.30	2	2	2	2
2	2	2	2	2		2		40.3	2	1	2		2	40.3	2	2	9	10	3.680	2	11.46	2	2	2	2
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2	2	2	2	2		2		38.1	2	2	2	CAT 2 NRFS	2	38.1	2	2	9	10	2.900	2	12.44	2	2	2	2
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2	2	2	1	2		1	MNT	29.0	1	3	1	BREECH WITH CHORIOAMNITIS	2	29.0	1	1	6	8	1.280	1	2.25	2	1	2	2
2	1	2	1	2		1	MNT	36.6	1	3	1	PREVIOUS LSCS	2	36.6	1	1	9	10	3.220	2	6.35	2	1	2	2
2	2	2	2	2		2		38.3	2	1	2		2	38.3	2	1	9	10	2.390	1	20.47	2	1	1	2
2	2	2	1	2		1	MNT	40.2	2	2	2	NRFS	2	40.2	2	2	9	10	2.960	2	8.52	2	2	2	2
2	2	2	1	2		1	OHA	38.1	2	3	1	PRIMI BREECH	2	38.1	2	1	9	10	3.100	2	2.82	2	1	2	2
2	2	2	2	2		2		38.4	2	1	2		2	38.4	2	2	9	10	2.350	1	11.45	2	1	1	2
2	1	2	2	2		2		33.3	1	3	1	FAILED INDUCTION	2	33.3	1	1	9	10	2.120	1	4.98	2	1	2	2
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2	2	2	2	2		2		39.4	2	3	1	PREVIOUS LSCS	2	39.4	2	1	9	10	2.800	2	8.52	2	2	2	2
2	2	2	2	2		2		39.0	2	3	1	FAILED INDUCTION	2	39.0	2	1	9	10	2.960	2	2.63	2	2	2	2
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2	2	1	2	2		1	ORAL IRON	38.4	2	3	1	PREVIOUS LSCS	2	38.4	2	2	9	10	2.640	2	3.26	2	2	2	2
2	2	2	2	2		2		38.3	2	1	2		2	38.3	2	1	9	10	3.140	2	13.12	2	2	2	2
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2	2	2	2	2		2		38.1	2	2	2	NRFS	2	38.1	2	1	9	10	2.340	1	5.93	2	2	2	2
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247	122	2	30	3	26.70	2	1	2	6	2		0	2			2						2		0
248	123	2	29	2	26.00	2	1	1	4	2		0	2			2						2		0
249	124	2	28	2	25.30	2	1	1	4	2		0	2			2						2		0
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2	2	2	2	2	2		40.3	2	3	1	NRFS	2	40.3	2	2	9	10	3.060	2	4.22	2	2	2	2
2	1	1	2	2	1	MNT, ANTIHYPERTENSIVE DRUGS, MGSO4	28.6	1	3	1	SEVEN PRE ECLAMPSIA, BREECH + IUGR	2	28.6	1	2	9	9	0.901	1	8.18	2	1	1	2
2	2	2	2	2	2		39.0	2	1	2		2	39.0	2	1	9	10	2.940	2	5.25	2	2	2	2
2	2	1	1	2	1	MNT, ORAL IRON	40.0	2	3	1	NRFS	2	40.0	2	1	9	10	4.380	2	1.55	2	2	2	2
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2	2	2	2	2	2		37.2	2	2	2	NRFS	2	37.2	2	2	9	10	2.290	1	12.99	2	1	1	2
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2	1	2	1	2	1	MNT	36.0	1	3	1	PREVIOUS LSCS	2	36.0	1	1	8	10	3.160	2	3.21	2	2	2	2
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