IMPACT OF PREEXISTING AND NEWLY DETECTED MATERNAL HYPOTHYROIDISM ON MATERNAL AND PERINATAL OUTCOMES DURING PREGNANCY, AN OBSERVATIONAL STUDY

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MASTER OF SURGERY IN OBSTETRICS AND
GYNAECOLOGY

LIST OF ABBREVIATIONS

TSH- THYROID STIMULATING HORMONE

FT4- FREE THYROXINE

T3-TRIIODOTHYRONINE

T4- THYROXINE

TPO-THYROID PEROXIDASE

TPOA- THYROID PEROXIDASE ANTIBODY

TGA- THYROGLOBULIN ANTIBODIES

APGAR- APPEARANCE, PULSE, GRIMACE, ACTIVITY, RESPIRATION

IU/ML- INTERNATIONAL UNITS PER MILILITRE

TBG- THYROID BINDING GLOBULIN

HCG- HUMAN CHORIONIC GONADOTROPIN

SCH- SUBCLINICAL HYPOTHYROIDISM

DM- DIABETES MELLITUS

ATP- ADENOSINE TRIPHOSPHATE

IGF- INSULIN-LIKE GROWTH FACTOR

GH- GROWTH HORMONE

HTN-HYPERTENSION

PPH-POSTPARTUM HEMORRHAGE

HCT- HUMAN CHORIONIC THYROTROPIN

ACOG- AMERICAN COLLEGE OF OBSTETRICS AND GYNECOLOGY

FGR- FETAL GROWTH RESTRICTION

NICU- NEONATAL INTENSIVE CARE UNIT

LGA- LARGE FOR GESTATIONAL

SGA- SMALL FOR GESTATIONAL AGE

BMI- BODY MASS INDEX

LT4- LEVOTHYROXINE

BMR-BASAL METABOLIC RATE

CNS- CENTRAL NERVOUS SYSTEM

μg- MICRO GRAMS

I¹³¹- IODINE ION 131

SH-R- SHORTROOT

POG-PERIOD OF GESTATION

CLIA- CHEMILUMINESCENCE IMMUNO ASSAY

ATA- AMERICAN THYROID ASSOCIATION

ELFA- ENZYME LINKED FLURESCENT ASSAY

TSB- TOTAL SERUM BILIRUBIN

SD- STANDARD DEVIATION

CBC- COMPLETE BLOOD COUNT

PE-PRE-ECLAMPSIA

PTD- PRETERM DELIVERY

FGR-FETAL GROWTH RESTRICTION

IUD- INTRAUTERINE DEATH

MAS- MECONIUM ASPIRATION SYNDROME

AGE- ACUTE GASTROENTERITIS

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ABSTRACT

Background

It is well-known that maternal hypothyroidism has a negative impact on the pregnancy's obstetrical and neonatal outcomes.

Preterm births, oligohydramnios, postpartum haemorrhage, abruption placenta, and other obstetric consequences such hypertensive diseases of pregnancy

Poor birth weight, a low APGAR score, neonatal infections, foetal mortality, and other events related to birth

Methodology

Between January 2021 to April 2022, we conducted a 1.5-year prospective observational study in which we enrolled 427 cases who were admitted to labour wards after 28 weeks of gestation in the Department of Obstetrics and Gynaecology at BLDE (DEEMED TO BE UNIVERSITY) Shri B.M. Patil's Medical College, Hospital and Research Centre, Vijayapura, following clearance from the ethics committee. All 427 patients underwent a thyroid function test to check for hypothyroidism. If the TSH level was greater than 3 micro-IU/LITRE, FT4 and TPO antibodies were supplied and classified as subclinical and overt hypothyroidism. Preterm births, oligohydramnios, postpartum haemorrhage, and hypertensive disorders of pregnancy were documented as obstetrical outcomes, and low birth weight, foetal growth restriction, intrauterine mortality, neonatal sepsis, among other neonatal events, were observed and assessed for statistical significance

Results

In our investigation, premature births (p value 0.003), abruption of the placenta (p value 0.039), and hypertensive disorders of pregnancy (p value 0.0014) were found to be clinically significant in terms of negative obstetrical outcomes.

In terms of poor new born outcomes, a low APGAR score was discovered to be clinically significant (p value-0.015).

Patients with TPO antibodies more than 0.5 IU/ML have a positive correlation with pregnancy-related hypertension problems and this correlation is clinically significant (p value- 0.015)

Conclusion

Hence Preterm births, abruption of the placenta, and hypertensive disorders of pregnancy are all common in cases of maternal hypothyroidism, which negatively impacts pregnancy in a substantial way.

Patients with maternal hypothyroidism had poorer neonatal outcomes in terms of low APGAR scores.

The prevalence of both obstetrical and neonatal outcomes is higher in mothers with maternal hypothyroidism who also have large levels of TPO antibodies.

INTRODUCTION

The thyroid goes through a stress test throughout pregnancy, and the hormonal environment of the thyroid gland will alter dramatically during this time. There are a multitude of changes seen in the gland during pregnancy that help the gland adapt to the rising metabolic demands. These changes include an increase in the size of the gland and an increase in thyroid hormone production. This increase in hormone synthesis, in turn, results in a parallel increase in iodine requirement. The changes occur as a result of many physiologic events. There appears to be an oestradiol-driven increase in thyroid-binding globulin (TBG), pronounced during the first trimester and plateauing during the second and third trimesters. This leads to increased T3 and T4 binding and a concomitant reduction in free T3 and T4. In addition, the action of human chorionic gonadotropin (hCG), whose beta subunit has a structural similarity to thyroid-stimulating hormone (TSH), stimulates the TSH-receptor, resulting in an increase in T3, T4, and a decrease in TSH during the first trimester. This effect persists until the end of the first trimester. Along with these changes, there are other ones, like modifications in the peripheral metabolism of thyroid hormones and an associated increase in renal loss of iodine due to a rise in glomerular filtration rate.

TSH in the first trimester decreases as a result of these modifications, which also result in a minor decrease in free T3 and T4 and an increase in total T3 and T4. Throughout pregnancy, the levels of total T3 and T4 are raised. After mid-pregnancy, TSH and free T3/T4 levels are probably going to near to pre-pregnancy levels. ²

Thyroid disorders are the second most common endocrinological disorders in women of reproductive age, second only to diabetes.³ Subclinical hypothyroidism (SCH) is the most common thyroid dysfunction during pregnancy.⁴ According to western data, the prevalence of hypothyroidism in pregnancy is approximately 2.5%, whereas Indian studies have shown a higher prevalence of up to 13%.⁵ In SCH, the TSH level is elevated while the T4 level is normal. SCH is common during pregnancy, particularly in areas prone to iodine deficiency. Apart from iodine deficiency, the other most common cause is due to anti-TPO antibodies.⁶

Most thyroid dysfunctions are treatable but may adversely affect the mother and foetus if not evaluated and managed appropriately. Maternal adverse hypothyroidism-related events include threatened abortion, preterm labor, placental abruption, and postpartum hemorrhage.⁷

The developing fetus exclusively depends on maternal thyroid hormones during the first 12 weeks, after which the fetal thyroid gland starts producing hormones. Maternal SCH can lead to poor foetal outcomes like low birth weight, foetal growth retardation, stillbirths, neonatal deaths, neonatal hyperbilirubinemia, and perinatal mortality. Also, maternal SCH is associated with an increased risk of intellectual and motor developmental delay, attention deficit hyperactivity disorder, and language, vision, and hearing impairment.

There is a lacuna in the studies showing the effect of hypothyroidism on foetal and maternal outcomes and the prevalence of maternal hypothyroidism. Hence, this study was undertaken to assess maternal hypothyroidism's impact on obstetrical and perinatal outcomes.

AIMS AND OBJECTIVES

AIM:

To study the effect of hypothyroidism on obstetrical and perinatal outcomes.

Objectives of the study:

To evaluate the effect of maternal hypothyroidism on obstetrical outcomes concerning

- Hypertensive disorders of pregnancy
- Abruptio placenta
- Postpartum haemorrhage
- Preterm delivery
- Oligohydramnios
- Gestational DM
- Need for Cesarean delivery
- Anemia

To evaluate the effect of maternal hypothyroidism on perinatal outcome with respect to

- Fetal growth restriction
- Intrauterine death
- Low birth weight
- Low APGAR score
- Neonatal hyperbilirubinemia
- Neonatal sepsis
- Early neonatal death

REVIEW OF LITERATURE

Development of the Thyroid Gland:

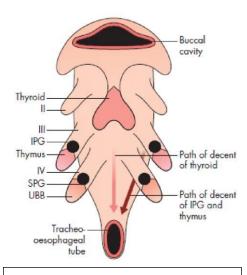


Fig 1- Thyroid gland structure

Just caudal to the tuberculum impar, a median endodermal thyroid diverticulum that grows down in front of the neck from the primitive pharynx's floor gives rise to the thyroid gland. The diverticulum's bottom end enlarges to become the gland. The thyroglossal duct is the name of the remaining, thin portion of the diverticulum. The majority of the duct soon vanishes. The position of the tongue's upper end is indicated by the foramen caecum, while the bottom end frequently endures as the pyramidal lobe. In the third month of development, the gland starts to produce hormones.

The medial and lateral thyroid anlagen, which are paired and produce calcitonin together, develop from the fourth branchial pouch and the fourth branchial pouch, respectively. In the eleventh week of pregnancy, colloid production occurs inside thyroid follicles.

At the end of the first trimester of pregnancy, throughout the life of the foetus, the thyroid gland completes its development. Before that, the only sources of thyroid hormones for the growing baby were maternal-free T4 and maybe T3 at low amounts.¹⁰

Between 16 and 20 weeks of gestation, the embryo will begin to secrete large amounts of thyroid hormones. The hypothalamus and pituitary are part of the regulatory feedback system, which does not fully develop until after birth. Iodine builds up in the developing thyroid, where

it is utilised to make thyroid hormones that are essential for myelination of nerve cells, brain development, and other processes throughout the foetal life. Iodine is used to create thyroid hormones and crosses the placenta.¹⁰

Anatomy of the Thyroid Gland:

An endocrine gland, the thyroid is located in the neck's anterior triangle (butterfly shape). It sits right below the cricoid cartilage and is made up of two lobes on either side that are joined by an isthmus. Between the sternomastoid muscle and the carotid sheath, the anterior lobes expand laterally.

The gland weighs between 6 and 20 grammes, depending on the person's body weight, age, and pregnancy status.¹²

On the back of the lateral lobe, where the thyroid gland is made up of many follicles wrapped in a capsule, are the parathyroid glands. Between 28 and 55 percent of people have a third lobe, which is now known as the pyramidal lobe of the thyroid gland. ¹³ The thyroglossal duct ¹⁴ is still present in the pyramidal lobe, also referred to as Lalouette's pyramid. It can occasionally separate into two or more pieces or become unattached. ¹⁵ It moves when swallowing because of its firmly fastened attachment to the trachea.

The superior thyroid artery, inferior thyroid artery, and occasionally the thyroid IMA artery, which branches off the subclavian artery, all supply blood to the thyroid.

Venous drainage: Venous blood leaves the body through the superior thyroid veins into the internal jugular vein and the inferior thyroid veins into the plexus of the left brachiocephalic vein.

Lymphatic drainage: into the pre- and paratracheal lymph nodes, as well as the lateral deep cervical lymph nodes.

The nerve supply is composed of parasympathetic nerve input from the superior and recurrent laryngeal nerves.¹⁷

Microscopy:

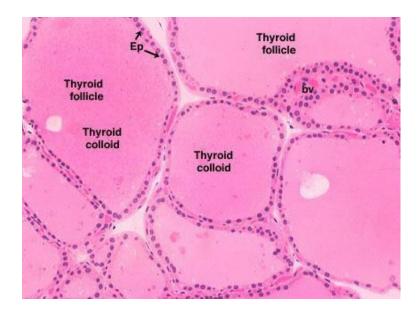


Fig 2- Microscopy of thyroid gland

Each of the numerous follicles of the thyroid gland is bordered by follicular cells, which exude colloid, a protein-rich fluid that acts as a precursor to thyroid hormones. The follicles are rather tiny and the cells are cuboidal or columnar when the gland is active. ^{18,19} The thyroid follicular cells are polarised, with an apical surface facing the follicular lumen and a basolateral surface facing the bloodstream. Thyroglobulin (Tg) resorption from the follicular lumen takes place if there is an increase in the demand for thyroid hormones, and there is proteolysis within the cell to create thyroid hormones that are eventually released into the bloodstream. ^{18,20}

The thyroid gland produces hormones.

Follicular cell-derived iodothyronines

Calcitonin is produced by parafollicular cells.

The term "iodothyronines" refers to two hormones. 21,22

Thyroxine or 3,5,3,5-tetraiodothyronine (T4)

3,5,3'-triiodothyronine (T3)

Each year, dietary iodine as iodides is needed to generate adequate levels of thyroxine in order to prevent iodine insufficiency.

Iodides that have been consumed are absorbed from the digestive tract into the blood and the majority of them are quickly eliminated by the kidneys. However, thyroid gland cells systematically remove 1/5 of ingested iodides from the circulating blood, which is then used to synthesise thyroid hormones. ²²

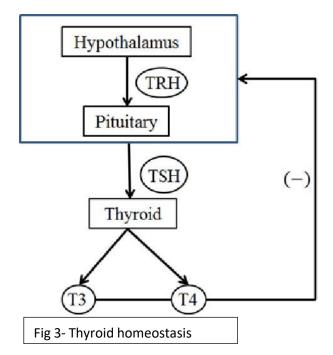
TRAPPING WITH IODIDE OR PUMPING WITH IODIDE

Iodide from the blood is absorbed by follicular cells of the thyroid gland. Due to the negative charge of follicular cells and the iodide ion, this process—known as "iodide trapping"—occurs against an electrochemical gradient.

Iodide trapping is an active process that necessitates a specific channel called Na+K+ATPase, and this concentration ratio can increase to 250 times when the gland is completely dynamic. Follicular cells have an iodide concentration that is 30 times higher than that of blood. ^{19,20}

TSH stimulates and hypophysectomy inhibits the thyroid cells' iodide pump, which affects how quickly iodide is trapped. ²²

The overview of thyroid homeostasis can be summarised in the diagram given below:



The above diagram shows the hypothalamus-pituitary-Thyroid axis with negative and positive feedback mechanisms.

Functions of thyroid hormones:

Effect on Growth

Thyroid hormones are necessary for development and growth. IGF (insulin-like growth factor) and GH are required for T4 and T3 to function (growth hormone). To allow the growth centre to mature, bones to restructure, and skeletal muscles to function appropriately, a foetus must have T4 and T3.¹⁹

THE IMPACT ON SEXUAL FUNCTION:

A lack of thyroid hormone in men can result in impotence and spermatogenesis problems as well as a loss of desire.

Hypothyroidism frequently results in amenorrhea and abnormal uterine bleeding in females. In addition, they are necessary for good follicular development, ovulation, and pregnancy development. 19,21

EFFECT ON PARTICULAR BODY MECHANISMS:

Stimulation of carbohydrate metabolism:

Nearly all phases of carbohydrate metabolism, including fast glucose uptake by cells, glucose breakdown, and glucose production, are stimulated by the thyroid hormone. All of these effects are most likely the outcome of the thyroid hormone-induced general increase in cellular metabolic enzymes.²²

Stimulation of fat metabolism:

The body's internal fat stores are reduced as a result of the thyroid hormone's mobilisation of lipids from fat tissue, which raises plasma levels of free fatty acids and sharply speeds up cellular oxidation of free fatty acids.²²

Increased basal metabolic rate:

Thyroid hormone stimulates metabolism in the majority of body cells, hence excess amounts of the hormone can raise BMR by 60% to 100% above the range considered normal. BMR decreases to around 1 12 times normal levels when thyroid hormone is not generated.¹⁸

Effect on sleep:

Hyperthyroid patients feel chronic fatigue as a result of the draining effects of thyroid hormone on the central nervous system- (CNS) and the excitable effects of thyroid hormone on synapses. Hypothyroidism, on the other hand, is characterised by "severe somnolence.²²

EFFECTS ON

Central nervous system

The multiplication and branching of nerve fibres in the cerebrum, as well as myelination, require T4 and T3. Nerve growth factor must be present for the fibre to branch. If not treated within a few months after life, thyroid insufficiency causes brain damage and cretinism and cannot be reversed by the subsequent injection of thyroid hormones. ¹⁹

Cardiovascular system

The cardiovascular system is impacted by the thyroid hormones in the following ways:

- -Increased cardiac output, heart rate, blood flow, and heart force
- -Average systolic blood pressure.²³

Respiratory system:

The hormone keeps the respiratory drive consistent. Thus, hyperthyroidism results in anxiety, tremors, and hyperreflexia while hypothyroidism results in sluggishness.

PREGNANCY THYROID GLAND PHYSIOLOGY

Thyrotropin-releasing hormone- (TRH), which is secreted by the hypothalamus on the maternal side, stimulates the anterior pituitary's thyrotrope cells to produce thyroid-stimulating hormone- (TSH), also referred to as thyrotropin.

A typical pregnancy does not cause an increase in TRH levels. However, TRH does penetrate the placenta and may act as an inducer of TSH secretion by the foetal pituitary.

To satisfy the demands of the mother and foetus, the thyroid gland increases thyroid hormone production by 40 to 100%. ²³

During pregnancy, the thyroid gland has a little expansion brought on by epithelial hyperplasia and increased vascularity. At term, the mean thyroid volume increases to 15 mL from 12 mL in the first trimester. ²⁴

Enlargement of the Thyroid Glands

The definition of goitre and the research area have an impact on its prevalence. In Aberdeen, 184 pregnant-women had a thyroid enlargement that was both visible and palpable, according to Crooks et al.- (1964), who used a standardised approach to make their findings. This was in contrast to 37% of age- and gender-matched non-pregnant controls.²⁵

Parity, mother age, or gestational stage had little effect on the distribution of goitre throughout pregnancy. There was no evidence of pregnancy-related thyroidal enlargement in a comparative study done by Crooks et al. in Iceland, a nation with a high iodine consumption, and goitre was equally common (20 percent) in both the control and pregnant groups. ²⁶ Histologically, the presence of sizable follicles and an excess of colloid is a sign that thyroid hormones are being produced and secreted actively (Burrow, 1975). ²⁷

Turnbridge and Hall assert that in addition to the possible stimulant of relative iodine deficit, some -of the enlargement may also be caused by increased blood flow, which can occasionally result in a vascular bruit.²⁸

Hypervolemia is brought on by pregnancy. Thyroid volume and total body water have a positive correlation in healthy persons. ²⁹ Body weight and BMI increase as water volume increases. Any goitre needs to be evaluated because a healthy pregnancy does not usually result in substantial thyromegaly. ³⁰

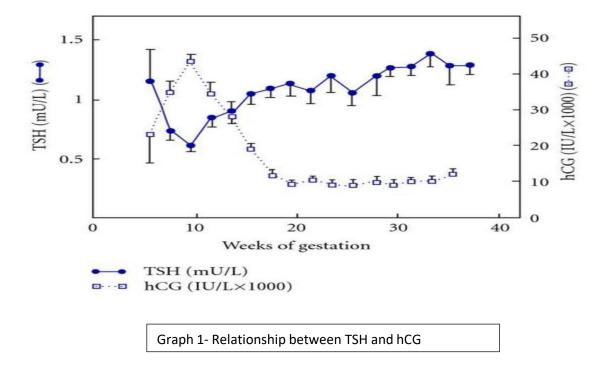
Variations in TSH and its relationship with hCG

Some women with thyroid glands that are otherwise healthy may see a drop in TSH levels during the first trimester. TSH levels in under 10% of females are below average. ³¹

Due to the structural closeness of the alpha subunits in both glycoproteins, hCG possesses intrinsic thyrotropic action, which is why high serum hCG levels activate the thyroid. The hCG and serum TSH levels also alter with gestational age. ²⁷

During the first trimester of pregnancy, HCG levels higher than 50,000 IU/l are linked to a briefly subnormal TSH. ³² TSH levels increased and peaked in the third trimester, peaking 3–4 days after delivery, regardless of the availability of iodine. At HCG levels greater than 400,000 IU/l, TSH is decreased in 100% of women while fT4 is elevated in 80% of them. ³⁴

In the first and third trimesters, TSH levels in smokers were lower than those in nonsmokers.³⁵

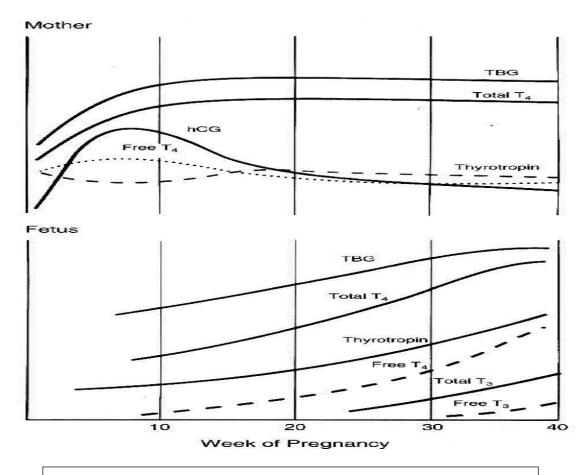


Variations of T3 and T4 (total and free)

Thyroid-binding globulin (TBG) levels begin to increase early in the first trimester and reach their peak at 20 weeks of gestation. Due to enhanced hepatic synthesis rates brought on by oestrogen stimulation and decreased metabolic rates brought on by increased sialylation and glycosylation of TBG, the concentrations of TBG are higher.

These higher TBG levels raise the concentrations of total serum T3, particularly T4 (which rise sharply between 6 and 9 weeks of gestation and reach a plateau at 18 weeks). However, they have no impact on the serum free T4 and free T3 levels, which are crucial in physiology. In the second half of pregnancy, fT4 levels drop and, less typically, fT3 levels do as well.

Even if significantly higher TBG concentration does not cause significantly lower fT4, its higher binding affinity does.³⁰



Graph 2- Relative changes in maternal and fetal thyroid function across

Alterations in Iodine Handling

An increase in glomerular filtration rate causes a surge in renal iodine clearance during pregnancy and for up to 6 weeks following birth. The drop in plasma inorganic iodine content is caused by a number of reasons, including hemodilution, an increased iodine distribution volume, and maternal losses to the foetus. Iodine is cleared from the thyroid more frequently as a result. Production of the thyroid hormone in the foetus increases in the second half of pregnancy. Following birth, iodide is also transmitted to breast milk, but total iodine intake is unaffected. ³⁶

According to the clinical practise recommendations of the Endocrine Society, iodine consumption before pregnancy should be 150 g/day to maintain adequate iodine stores of thyroid. A daily intake of 250 g of iodine is recommended throughout pregnancy and lactation. Iodine shortages do not affect women living in countries with sufficient and ongoing programmes to iodize all salt.³⁷

Summary of thyroid hormone physiology in pregnancy.³⁸

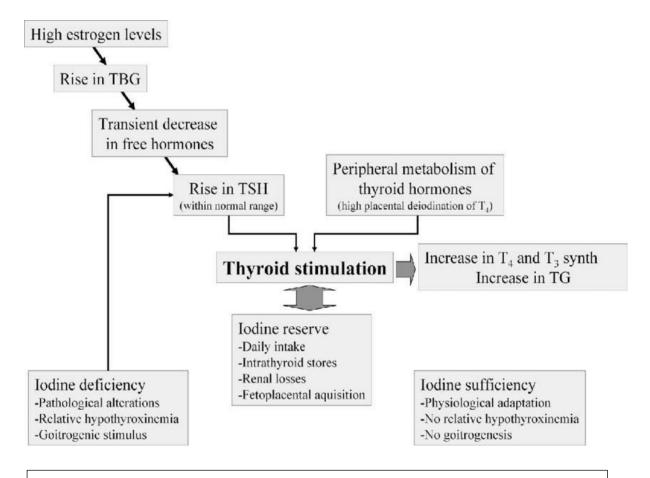


Fig 4- thyroid hormones physiology in pregnancy

Physiologic changes th	nat influence thyroid functi	on in pregnancy
Physiologic Change	Effect on Thyroid Function Test Results	Impact on Interpretation of Thyroid Function Tests
† Thyroxine-binding globulin	↑ Serum total T3 and T4 concentrations	Total thyroid hormone levels may be misleading; need to rely on free thyroid hormone levels.
† Human chorionic gonadotrophin secretion	↑ Free T4 and ↓ TSH	High human chorionic gonadotrophin levels may result in gestational thyrotoxicosis. This usually only requires symptomatic treatment but needs to be distinguished from pathologic thyroid disease. Response possibly impaired in TPO antibody–positive women.
† lodine excretion	↓ Thyroid hormone production in iodine-deficient areas	Need to be mindful of iodine deficiency and ensure optimal intake ideally before conception.
↑ Plasma volume	↑ T3 and T4 pool size	
Increased type 3 5-deiodinase (inner ring deiodination) activity from the placental	† T3 and T4 degradation	May explain in part the increasing thyroid demand in pregnancy.
Thyroid enlargement (in some women)	Increased thyroglobulin	Small goiters is common in pregnancy, but may be a sign of low thyroid function so merits thyroid function testing.

Table 1- Physiological changes that influence thyroid function in pregnancy

Causes of hypothyroidism

Primary

- Hashimoto's thyroiditis and atrophic thyroiditis are examples of autoimmune hypothyroidism.
- Iatrogenic conditions include ¹³¹I treatment, partial or complete thyroidectomy, and external neck radiation for cancer or lymphoma.
- Iodine overload (including amiodarone and iodine-containing contrast media), lithium, anti-thyroid
 medications, p-aminosalicylic acid, interferon and other cytokines, aminoglutethimide, and sunitinib
 are among the pharmaceuticals.
- congenital hypothyroidism: SH-R mutation, missing or ectopic thyroid gland
- Iodine shortage
- Amyloid disease, sarcoidosis, hemochromatosis, scleroderma, cystinosis, and Riedel's thyroiditis are examples of infiltrative illnesses.
- Type 3 deiodinase overexpression in infantile hemangiomas

Transient

- Postpartum thyroiditis is a type of silent thyroiditis.
- chronic thyroiditis
- withdrawal of thyroxine therapy in those with a healthy thyroid
- Following 131I therapy or partial thyroid gland resection for Graves' disease,

Secondary

- Tumors, pituitary radiation or surgery, infiltrative illnesses, Sheehan's syndrome, trauma, and inherit forms of combined pituitary hormone deficits can all cause hypopituitarism.
- isolated TSH inactivity or deficit
- Treatment for bexarotene
- Tumors, trauma, infiltrative illnesses, and idiopathic hypothalamic disease

signs and symptoms of hypothyroidism, ³⁹

symptoms	signs
 Tiredness and weakness Dry skin Coldness Loss of hair Difficulty concentrating and poor memory Difficult to pass stools Weight gain with poor appetite Shortness of breath Voice alteration Heavy menstrual bleeding (later, 	 Having rough, dry skin and cold outer extremities Puffy hands, feet, and face Hair loss that is wide spread Decrease in heart rate Leg swelling delayed relaxation of the tendon reflex over use strain injury effusions of serous cavities
 Heavy menstrual bleeding (later, infrequent menses or loss of menses) 'Pins and needles' sensation Hearing impairment 	

Table 2- Signs and symptoms of hypothyroidism

Types of hypothyroidism

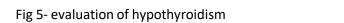
Hypothyroidism is a clinical condition characterised by decreased production of thyroid hormones.

Primary hypothyroidism: decreased thyroid hormone production due to intrinsic pathology in the thyroid gland. 40

Secondary hypothyroidism: caused by pituitary gland dysfunction. 40
Tertiary hypothyroidism: because of dysfunction in the hypothalamus. ⁴⁰
Thyroid hormone resistance hypothyroidism is most commonly caused by a mutation in the gene encoding thyroid hormone receptor B (TRB gene mutation). 40
<u>Subclinical hypothyroidism</u> is defined as usual FT4 production with elevated serum TSH values. ⁴⁰
Overt hypothyroidism is characterised by low serum T4 and elevated serum TSH. ^{38, 40}
<u>Isolated hypothyroxinemia during pregnancy</u> : low FT4 below the 2.5 th percentile with normal TSH ⁴¹

EVALUATION OF HYPOTHYROIDISM Measure TSH (Elevated) Normal Measure unbound T₄ Pituitary disease suspected? Yes (Normal) No Low Mild Primary Measure unbound T₄ No further hypothyroidism hypothyroidism tests (Low) Normal TPOAb+ or TPOAb+ TPOAb-TPOAb-, no symptomatic symptoms No further Autoimmune Rule out other tests

Evaluation of hypothyroidism, ⁴²



causes of hypothyroidism

Rule out drug effects, sick euthyroid syndrome,

then evaluate anterior pituitary function

VARIOUS TYPES OF HYPOTHYROIDISM'S EFFECTS ON OBSTETRIC OUTCOMES

hypothyroidism

T₄ treatment

Effects of overt hypothyroidism

T₄ treatment

Annual follow-up

Overt or symptomatic hypothyroidism complicates between 0.2 and 1.2 % of pregnancies. 43 Overt hypothyroidism, which is newly detected, occurs in approximately 0.2% – 0.66% of pregnant women. .44,45

Adverse obstetric outcomes with overt hypothyroidism include fetal loss, premature delivery, low birth weight, and pre-eclampsia.⁴⁶

- In a study done in 1993, Leung AS et al. found that overt hypothyroidism is linked to a higher incidence of problems such pre-eclampsia and low birth weight infants.⁴⁷
- Reid SM et al(2010) .'s study determined the necessity of intervention in the
 management of maternal hypothyroidism and discovered that levothyroxine treatment
 lowers the risk of pre-eclampsia in pregnant women who are overtly hypothyroid,
 suggesting that it has a positive impact on the treatment of hypothyroidism. However,
 it made no mention of how hypothyroidism affected the newborn. 48

Effects of subclinical hypothyroidism

TSH levels that are raised yet thyroxine (T4)levels in the free serum are normal are known as subclinical hypothyroidism (SCH).⁴⁰

According to numerous research ^{49–60}, there is a link between SCH and a higher risk of unfavourable pregnancy and neonatal outcomes. including

- preterm deliveries (13.5%)
- gestational hypertension- (11%)
- pre-eclampsia (6%)
- Abruptio placenta (4%)
- Cesarean delivery (27%)
- low birth weight (8.5%)
- low Apgar score (0.7%)
- Neonatal death (0.5%)

According to Kattah et al., there is a link between subclinical hypothyroidism and pregnant hypertension since patients with subclinical hypothyroidism who were identified with gestational hypertension and treated with levothyroxine medication had normalised blood pressure measurements.⁶¹

Out of 100 patients in an observational study by Sreelatha et al.⁶², 96 had subclinical hypothyroidism. Thyroid deficiency in pregnancy is associated with

• miscarriages (2.1%)

- Anemia (4.20%)
- Pregnancy-induced hypertension (14.7%)
- Gestational Diabetes Mellitus (4.2%)
- Preterm labours (3.1%)
- oligohydramnios (16.67%)
- Cesarean deliveries (22.9%)
- 6.3% had postpartum hemorrhage.

Contrarily, these two studies ^{58, 63} demonstrate no discernible advantage in reducing the risk of overall unfavourable pregnancy outcomes or hypertensive disorders of pregnancy, supporting the argument against routine subclinical hypothyroidism treatment.

Thyroid auto-immune diseases in pregnancy:

Hashimoto's thyroiditis:

Hashimoto's thyroiditis is one of the most common autoimmune illnesses in general; in women of reproductive age, the incidence of anti-thyroid antibodies is 10-15%, and it is considerably rising. ⁶⁴

The aetiology of autoantibodies depends on the cell-mediated immune response; throughout pregnancy, anti-TPO antibodies gradually decrease and reach their lowest levels in the third trimester. 65

A increased incidence of placental abruption, gestational HTN, PPH, miscarriages, as well as foetal problems, has been linked to overt or subclinical hypothyroidism.⁶⁶

Postpartum Thyroiditis:

The incidence of this condition ranges from 1.1 to 21.1 percent; it is described as thyroid gland dysfunction in the first year following birth, which can present as hypo- or hyperthyroidism. ⁶⁶ Those with Type 11 DM are affected 3 times more often and 6 times more frequently than women with increased TPOABb. ^{63, 67}

At six and twelve months following delivery, thyroid function tests are advised for these women.

Early on in the course of the illness, the hyperthyroid phase of the disease will manifest and endure for just 1-2 months. Later, the thyroid tissue is destroyed, resulting in the phase of hypothyroidism, which is more prevalent in TPOAb-positive patients. It could be temporary, with thyroid function returning in 12 months, or it could be permanent in 30% to 50% of patients);⁶⁸ Additionally, it was found that people who experience temporary hypothyroidism have a 20–60% likelihood of eventually acquiring permanent hypothyroidism. ⁶⁷

Effects of autoimmune hypothyroidism

Autoantibodies against almost 200 thyrocyte components are thought to be the primary cause of the majority of thyroid issues. These antibodies can cause thyroid inflammation, which may cause the death of follicular cells, or they can promote or inhibit thyroid activity.

The TSH receptor is bound and activated by thyroid-stimulating autoantibodies, also known as thyroid-stimulating immunoglobulins, which leads to thyroid hyperfunction and growth. Even though they are most frequently linked to Graves disease, this effect might be mitigated by concurrent generation of thyroid-stimulating blocking antibodies.⁶⁹

Through the activity of the enzyme thyroid peroxidase, the thyroid gland usually creates thyroid hormones (TPO).

5–15 percent of all gravidas have thyroid peroxidase antibodies, which are anti-TPO.^{56,70}

Human chronic gonadotropin (hCG) and human chronic thyrotropin (hCT), in particular, may interact with anti-thyroid antibodies or be an indication of immune system alterations in the mother.⁷¹

Anti-thyroid antibodies cause euthyroid non-pregnant women to repeatedly miscarry throughout their subsequent pregnancies.^{72,73}

According to Stagnaro-Green et al study .'s of 552 women who had thyroid antibody screening, the positive antibody group saw a rate of abortion of 17% compared to the negative antibody group's 8.4%.⁷²

- According to the study by Mecacci F et al,⁷⁴ the presence of TPO antibodies has an incidence of a three-fold increase in the risk of recurrent abortions and a four-fold increase in the risk of foetal death and pre-eclampsia syndrome, thus drawing a positive correlation between TPO antibody presence and adverse obstetrical outcomes.
- Another study, conducted by Abbassi-Ghanavati M et al.⁷⁵ enrolled 1000 women with positive TPO and found a link between TPO antibody presence and placental abruption.
- According to Anderson and Jameson's two studies ^{69,76}, patients with TPO antibodies
 have a higher risk of postpartum thyroiditis and a lifelong risk of permanent thyroid
 failure.
- TPO-Ab was positively linked with subclinical hypothyroidism (SCH) with hypertension but not with SCH without hypertension in the study by Shimizu Y et al.
- According to Matsua et al., women whose pregnancies terminated in miscarriages had significantly lower free T3 and T4 readings.⁷⁸
- 22 non-pregnant women with a problematic obstetric history underwent thyroid antibody screening by Bussen Steck et al. in 1996. They discovered that 36 percent of the population had thyroid antibodies, compared to controls who were multiparous (9%) and nulliparous (5%), respectively.

Relation of Maternal and Foetal thyroid hormonal status

Because the placenta is present and controls the transport of several chemicals, including T4, to the foetus, system of the hypothalamus, pituitary, and thyroid in the foetus develops rather independently of maternal influence under typical circumstances. The stage of foetal maturation is mostly reflected by circulating thyroid hormone levels. Up to week 10 of pregnancy, there are trace amounts of thyroid hormone receptors in the developing fetus's brain. Through week 16 of pregnancy, the concentrations of thyroid hormone receptors rapidly increase by a factor of 10. 80

At 12–14 weeks of development, total and free T4 levels in foetal serum are detected at low levels. ^{81,82,83} The mean foetal serum total T4 is two mcg/dL (26 nmol/L) at 12 weeks, and a

significantly more substantial fraction is in the free form, according to results obtained by foetal cord sample in typical pregnant women. By 36 weeks, the foetal incremental T4 concentration has normally increased to levels that are comparable to those of non-pregnant females (10 mcg/dL or138 nmol/L). This increase in serum TBG concentration and total T4 levels are principally related to the stimulation of the foetal liver by maternal oestrogen. Additionally, the concentration of foetal free T4 has increased as a result of the TSH receptor being upregulated. ⁸⁴

Contrary to T4, despite an increase in the amounts of T3's inactive metabolites reverse T3 (rT3) and T3 sulphate, the blood level of T3's active metabolite is consistently lower during foetal life than it is after delivery. At 12 weeks, the foetal blood T3 level is 6 ng/dL (0.09 nmol/L), and at 36 weeks, it is 45 ng/dL (0.68 nmol/L). 85

It highlights how crucial local T4 to T3 conversion is for brain health. Type 2 deiodinase, which converts T4 to T3, is expressed more often in the foetal cerebral cortex throughout the second trimester at the same time as T3 concentrations. One further possible T3 source for the pituitary is T3 sulphate. When a baby is full-term, the blood level of TSH climbs steadily from 4 mU/L at 12 weeks to 8 mU/L, and is frequently higher than the corresponding mother levels. S2,87 The specific cause is unknown, however it may be related to the fetus's high levels of TRH and the hypothalamic-pituitary-thyroid axis's immature development.

Role of placental transfer

During the early part of pregnancy, before the foetus' thyroid has fully developed, T4 can be found in the foetus' brain, proving that there are mechanisms in place for a modest but important transfer of maternal T4 to the foetus' circulation. 85,88

Transmission of the mother's thyroid hormone is essential when the foetus is hypothyroid. When neonates with severe congenital hypothyroidism received prompt treatment after delivery, the cognitive outcomes were predicted or almost normal. Even in neonates born without thyroid peroxidase due to a congenital condition, the cord serum T4 content is, for example, between 25 and 50% of normal. ⁸⁹ This maternal T4 swiftly exits the newborn circulation with a half-life of 3 to 4 days.

Congenital hypothyroidism and the importance of screening

One of the most treatable causes of intellectual disability is congenital hypothyroidism, which is why new born are routinely screened for the disease.⁹⁰

A third or so of the world's population lives in developing nations, where screening programmes are also being implemented. ⁹⁰ To avoid the misleading increase brought on by the physiologic spike in neonatal TSH, which takes place soon after birth, screening should be done between two and five days after birth. When samples show a value below a predetermined cut-off, several programmes use primary T4 screening with TSH testing. Others combine a reflex T4 method with a preliminary TSH measurement. Since congenital hypothyroidism screening virtually eliminated intellectual disability, postnatal levothyroxine therapy is started right away. ^{91,92}

The maternal thyroxine is passed to the foetus throughout pregnancy. 93

Importance of thyroid hormones on foetal growth

For healthy foetal brain development, particularly prior to the commencement of foetal thyroid gland function, maternal thyroxine is crucial. ^{94,95}

The maternal thyroxine contribution is still significant even if the foetal gland starts concentrating iodine and synthesising thyroid hormone after 12 weeks of gestation.

Thirty percent of the thyroxine in the term foetal serum comes from maternal sources. ⁹⁶ Even Nevertheless, little is known about the developmental hazards linked to maternal hypothyroidism after mid-pregnancy. ⁹⁷

Cortical grey matter volume in children is inversely correlated with maternal thyroid function during the first trimester. 98

Effects on the foetus

Some of the impacts on the foetus are

- 1. Fetal growth restriction
- 2. Low birth weight

- 3. Intrauterine deaths
- 4. Low APGAR scores
- 5. Fetal distress
- Untreated hypothyroid women are much more likely to develop pre-eclampsia, and insufficient therapy leads to LBW kids, according to the ACOG practise bulletin from 2001.
- N. Ohara et al. (2004) conducted a literature review on the role of thyroid hormone in trophoblastic function and neuronal development. They concluded that "Scrutiny of maternal thyroid hormones to ensure adequate hormone levels in early pregnancy is of prime importance in preventing miscarriage and neurodevelopmental deficits in infants." 100
- Using comparisons with the general population, Leung AS, Miller LK, et al. (1993) investigated maternal hypothyroidism's effects on pregnancy. When compared to the general population, they discovered that women who were sub clinically and obviously hypothyroid had a greater incidence of pregnancy-induced hypertension. The implications of untreated or improperly managed hypothyroidism during pregnancy were discussed. Gestational hypertension occurs in the majority of hypothyroid patients who are still hypothyroid after birth. Additionally, they discovered a higher prevalence of low birth weight in children born to hypothyroid moms, and they came to the conclusion that this was likely caused by the high rates of premature delivery in these mothers. ⁴⁷
- In a retrospective study of 167 pregnant women, Idris, Srinivasan R, Simm A, Page RC, et al. (2005) found a correlation between the outcomes and TSH levels in the first and third trimesters. When compared to non-hypothyroid individuals, effects like the caesarean section rate were higher in hypothyroid mothers (287 percent) (18 percent). A risk factor for low birth weight is hypothyroidism, as shown by the prevalence of low birth weight in moms with TSH > 5.5 and 5.5, respectively, being 15% and 4.8%. Low birth weight was less common in moms whose TSH was greater than 2 and who had their TSH under control in the third trimester. ¹⁰¹
- The prevalence of thyroid dysfunction during pregnancy was researched by Sahu MT et al. in 2009. They found that compared to controls, overtly hypothyroid women had an increased risk of pregnancy-induced hypertension (P = 0.04), intrauterine growth restriction (IUGR), and intrauterine demise (P = 0.0004). In addition, caesarean

sections were more frequently performed on hypothyroid moms, most likely because these patients had a higher frequency of foetal distress. Pregnant women with subclinical hypothyroidism had a significantly increased caesarean section rate for foetal distress (P = 0.04). Additionally, the overt hyperthyroidism group had significantly higher rates of both gestational diabetes and newborn problems (P = 0.03 and 0.04, respectively). P = 0.03

- When Hareesh MV et al. (2015) looked at the impact of maternal hypothyroidism on the foetus, they found that babies delivered to hypothyroid moms had a higher prevalence of NICU admissions. Even though they looked at how hypothyroidism affected foetal birth weight, they were unable to find a positive association, perhaps as a result of the smaller sample size. In the same study, it was discovered that there were more maternal difficulties, such as preterm labour and a higher chance that the mother would need a caesarean section. Whether the mother has subclinical or overt hypothyroidism, they discovered that these issues are frequent. ¹⁰³
- Out of 100 individuals, 96 had subclinical hypothyroidism, according to an observational study by Sreelatha et al. (2017). These patients also reported foetal adverse outcomes, such as low birth weight (21.9%), hyperbilirubinemia (9.4%), and NICU admissions (14.7%). 62
- L-T4 supplementation is advised to improve pregnancy outcomes since this study
 found that pregnant women with hypothyroidism and hypothyroxinemia are more
 likely to deliver children who are LGA (big for gestational age). Hypothyroid women
 often have a higher risk of preterm birth and low birth weight in their offspring, where
 the risk of SGA does not rise as gestational age is added. 104

Screening. 105

Risk factors that should prompt a pregnant thyroid disease screening in accordance with American Thyroid Association recommendations

- Women who have had thyroid problems in the past or had thyroid surgery, as well as those with a family history of thyroid issues such goitres, ought to be examined.
- Thyroid antibodies and symptoms or clinical indications of hypothyroidism are present in women.

- Women with type I diabetes who have a history of miscarriage or early delivery, as
 well as other autoimmune conditions such vitiligo, adrenal insufficiency, and
 hypoparathyroidism that are usually connected to autoimmune thyroid dysfunction,
- In females who have undergone therapeutic head or neck radiation in the past,
- The prevalence of hypothyroidism rises with age in females with a body mass index (BMI) of 40 kg/m2 and an age of 30 years or older.
- Women on amiodarone and lithium, as well as those who recently (within the last six weeks) came into contact with iodinated radiological contrast agents.

Management of hypothyroidism in pregnancy

Levothyroxine taken orally is the suggested treatment for hypothyroidism in pregnant women. Other thyroid supplements, such desiccated thyroid or triiodothyronine (T3), shouldn't be taken while pregnant.¹⁰⁶

Serial hCG and TSH administration to women with known hypothyroidism is ineffective in promoting thyroxine production at the proper amount. Due to an increase in demand during pregnancy, an incorrect adjustment in exogenous levothyroxine therapy will result in maternal hypothyroidism. According to clinical research, the need for thyroxine (or exogenous levothyroxine) increases as early as the first four to six weeks of pregnancy. ¹⁰⁷

These requirements steadily increase between weeks 16 and 20 of pregnancy before plateauing till delivery. The timing of TSH follow-up intervals for patients who have received treatment as well as recommendations for LT4 dosage modifications for pregnant women who are affected are based on these findings.

Half of TPOAB-positive pregnant women experience postpartum thyroiditis, and 1/3 to 1/2 of these women experience persistent hypothyroidism within the first year after giving birth.

68 The thyroid function of pregnant TPOAb-positive women must therefore be monitored both during and after the pregnancy. Between three and twelve months after delivery, the postpartum thyroiditis hypothyroid phase sets in. TSH and FT4 levels should be checked on women with TPOAB every three months for postpartum thyroiditis. If TSH levels are normal at delivery, they should be checked again between 6 and 9 months afterwards. 108

Follow-up. 107

Women who have subclinical and overt hypothyroidism or who are at risk for it (euthyroid patients with the presence of TPO or TGAB or treated with radioactive iodine) should have their serum TSH levels routinely 'checked every four weeks until mid-gestation' and at least once close to 30 weeks of pregnancy.

MATERIALS AND METHODS

1. Source of data

- Shri B.M. Patil's Medical College, Hospital, and Research Centre, Vijayapura.
 Patients are seen in the labour ward of the Department of Obstetrics and Gynecology in BLDE (deemed to be a university).
- Patients will get complete information about the trial, and formal informed consent will be obtained.
- After receiving ethical clearance, the period of contemplation would last from January 2021 until April 2022.

2. INCLUSION CRITERIA

- Pregnant women with a known case of hypothyroidism
- Women who Develop Hypothyroidism During Pregnancy
- All pregnant women beyond the 28-week period of gestation get admitted to the labour ward.

3. EXCLUSION CRITERIA:

- Patients have undergone radioiodine therapy.
- Patients have undergone a partial thyroidectomy.
- Multifetal gestation

4. **SAMPLE SIZE**:

The study will involve all expectant mothers who are past the 28-week mark in gestation.

The study would need a sample size of 301⁵ if it were assumed that 13% of the population's subjects have the factor of interest.

for calculating the predicted proportion with an accuracy of 5% absolute and a confidence level of 98%.

N = Z2p*q/d2 was the formula used.

Where does the z=z statistic fall within the significance threshold?

d2 is the absolute error.

P is the proportional rate.

q = 100-p

Methodology (Study Design):

- If they meet the inclusion and exclusion criteria, pregnant women who are over 28 weeks POG and present to the labour room are recruited for the study after giving their informed consent.
- The following patient conditions were looked into after a pertinent history and examination:
- There will be a TSH analysis on blood samples.
- TSH levels were evaluated using the CLIA (CHEMILUMINESCENCE IMMUNOASSAY) method.
- Further testing for fT4 and anti-TPO antibodies will be done in patients with abnormal TSH levels.
- CLIA performed the fT4

- created by ELFA was an anti-TPO antibody (enzyme-linked fluorescent assay)
- Patients will be evaluated for hypothyroidism following the investigations.

and the study's reference range will be based on ATA 2011 recommendations. When trimester-specific policies are unavailable, the ATA states that the following reference ranges will be applied: ¹⁰⁵

- First trimester: 0.1–2.5 microIU/liter
- Second trimester: 0.2–3.0 microIU/liter
- Third trimester: 0.3–3 microIU/liter
- TPO antibody levels and free T4 levels are assessed if TSH levels are high; the reference range is as follows:
- 0.7–2.5 ng/dl of free t4.¹⁰⁹
- Antibodies to TPO: 0.5 IU/ml ¹¹⁰
- TSH levels are determined in infants by heel-prick blood collection.

TSH is routinely measured between 2 and 5 days of age, but it is also checked in preterm and critically ill term babies at 1 hour of age. The reference range for TSH in newborns is: 111

- Premature infants (28-36 weeks): 1st week of life: 0.7-27 mIU/l
- TERM INFANTS: 1-17.6 Miu/L FROM BIRTH TO 4 DAYS
- The maternal outcomes are in terms of the following:
 - o Anemia: Hb <11g/dL
 - Hypertensive disorders in pregnancy
 - o Abruptio placenta,
 - Postpartum hemorrhage,
 - o Preterm deliveries: 37 completed weeks
 - o incidence of caesarean section
 - Gestational diabetes mellitus
 - Oligohydramnios <5
- The perinatal outcome in terms of

- Fetal growth restriction: a baby weighing less than 90% of other babies at the same gestational age
- intrauterine death
- LBW: <2.5kg
 - o Low APGAR score: If the score at 1 minute is < 7
 - o Other neonatal outcomes noted:
 - Neonatal hyperbilirubinemia:

Neonatal hyperbilirubinemia will be considered pathological if: 112, 113

- 1. If jaundice manifests itself within the first 24 hours of life,
- 2. Total serum bilirubin (TSB) level >5 on the first day of life
- 3. >10 TSB on the second day of life
- 4. >15 TSB on the third day of life

The method used to measure bilirubin levels is the DIAZO method.

- > Sepsis
- > Early neonatal death

5. STATISTICAL ANALYSIS:

The acquired data will be placed into an Excel spreadsheet, where statistical analysis for the social sciences will be carried out (version 20).

A mean (or median) SD, counts and percentages, and graphs will be used to display the results.

An independent t-test will be used to compare regularly distributed continuous variables between two groups; Mann-Whitney u tests will be used for variables that are not normally distributed.

The chi-square test will be used to compare categorical variables.

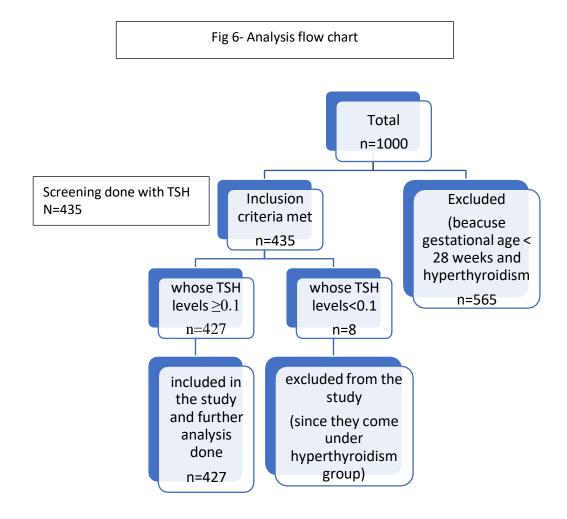
We will define statistical significance as P 0.5. Every statistical test will have two possible outcomes.

In this study, routine, standardised methods must be investigated or implemented. This research does not involve any animal testing.

INVESTIGATIONS:

In the mother, in the child

- 1. TSH
- 2. fT4 BILIRUBIN
- 3. Anti-TPO antibody
- 4. CBC

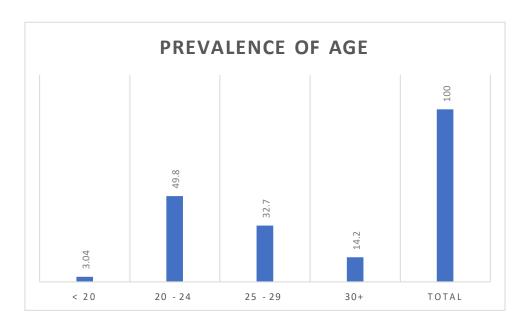


RESULTS AND OBSERVATIONS

Table 3: Age(Years)

The age range seen in the study is explained by the table and graph below. The majority of expectant mothers are between the ages of 20 and 24 years (49.8 %)

Age(Years)	No. of patients	Percentage
< 20	13	3.04
20 - 24	213	49.8
25 - 29	140	32.7
30+	61	14.2
Total	427	100



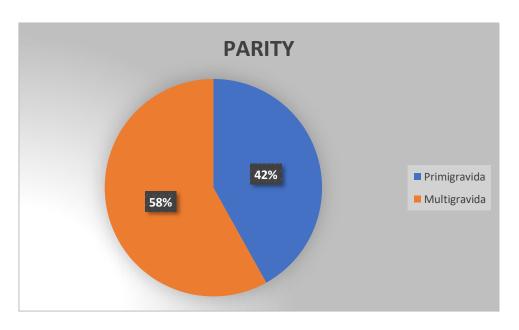
Graph 3

Table 4: Obstetric score

The prevalence of singleton and multiple pregnancies is explained in the table and graph below.

There are more multigravida (58.07%), with gravida 2 being the most common.

Obstetric score	No. of patients	Percentage
Primigravida	179	41.9
Multigravida	248	58.07
Total	427	100



Graph 4

The prevalence of various hypothyroidisms is explained in the table and graph below.

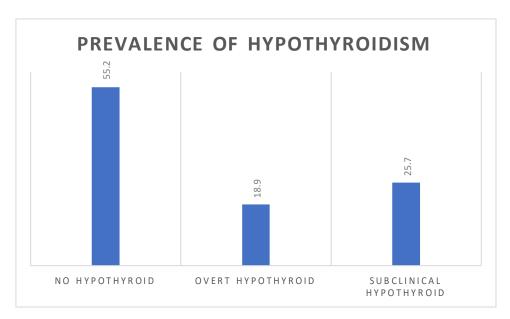
In our study, subclinical hypothyroidism was most frequently observed (25.7 percent)

With the 191 cases of hypothyroidism, 54 cases (or 59.3 percent) are already receiving therapy (pre-existing cases), and 137 cases (or 71.7 percent) are newly diagnosed cases.

TABLE 5- Prevalence of hypothyroidism

TYPES	NUMBER OF PATIENTS	PERCENTAGE
NO HYPOTHYROID	236	55.2
OVERT HYPOTHYROID	81	18.9
SUBCLINICAL	110	25.7
HYPOTHYROID		25.1
TOTAL	427	100.0

Graph 5



EFFECT OF MATERNAL HYPOTHYROIDISM ON OBSTETRICAL OUTCOMES

The impact of maternal hypothyroidism on pregnancy-related hypertension illnesses is described in the table and graph below.

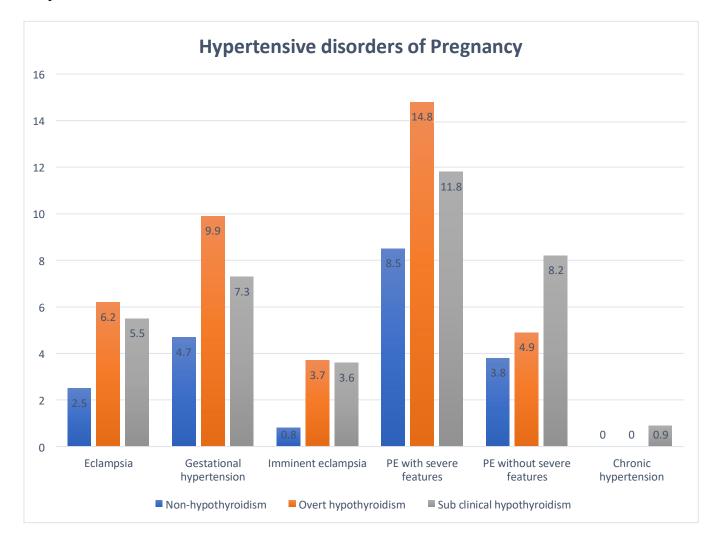
Both subclinical and overt hypothyroidism have positive association with hypertensive disorders of pregnancy and found to be statistically significant (p value <0.05)

Patients with hypothyroidism are most likely to experience PE with severe symptoms (26.6 percent)

TABLE 6- EFFECT ON HYPERTENSIVE DISORDERS OF PREGNANCY

HYPERTENSIVE		TYPES	OF HY	POTHY	ROIDIS	M	Chi-	P
DISORDERS	No		Overt		Subclin	ical	square	value
	Hypothyroid		Hypothyroid				test	
	N	%	N	%	N	%		
Eclampsia	6	2.5	5	6.2	6	5.5	31.923	0.0014
Gestational hypertension	11	4.7	8	9.9	8	7.3		
Imminent eclampsia	2	.8	3	3.7	4	3.6		
None	188	79.7	37	45.7	57	51.8		
PE with severe features	20	8.5	12	14.8	13	11.8		
*PE without severe features	9	3.8	4	4.9	9	8.2		
chronic hypertension	0	0	0	0	1	.9		
Statistically significa	int						I	

Graph 6



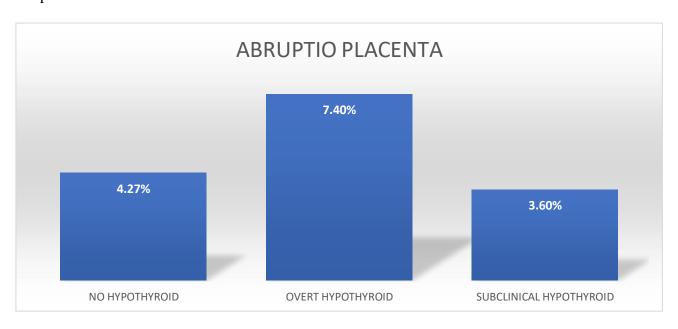
The table and graph that follows demonstrates the association between maternal hypothyroidism and placental abruption

Both overt and subclinical hypothyroidism are positively corelated with abruptio placenta and found to be statistically significant (p value <0.05)

TABLE 7- EFFECT OF MATERNAL HYPOTHYROIDISM ON ABRUPTIO PLACENTA

				ABRU	PTIO PL	ACENTA		
	NO		OVERT		SUBCLI	SUBCLINICAL		P
	HYPOTI	HYROID	HYPOTHYROID		HYPOTHYROID		SQUARE	(SIGNIFICANCE)
							VALUE	
	N	%	N	%	N	%	113.084	<0.00001
YES	10	4.27	6	7.4	4	3.6		
NO	226	100	75	92.6	106	96.4		
TOTAL	236	100	81	100	0 110 100			
Statistica	lly signific	cant						

Graph 7



The table and graph that follows demonstrates the impact of maternal hypothyroidism on preterm births

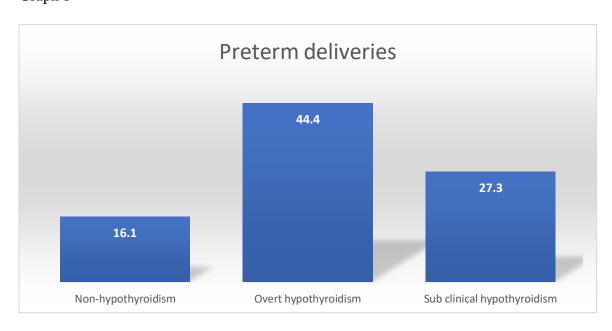
Both overt and subclinical hypothyroidism have affected preterm deliveries and shown to be statistically significant (p value < 0.05)

In our study, late preterm, defined as between 34 + 0/7 and 37 + 6/7, is the most typical (55.5percent)

TABLE 8- EFFECT OF MATERNAL HYPOTHYROIDISM ON PRETERM DELIVERIES

					PRETER	RM		
	NO		OVERT		SUBCLI	SUBCLINICAL		P
	HYPOTHYROID		HYPOTHYROID		HYPOTHYROID		SQUARE	(SIGNIFICANCE)
							VALUE	
	N	%	N	%	N	%	11.890	0.003
YES	38	16.1	36	44.4	36	27.3		
NO	198	83.9	45	55.6	74	72.7		
TOTAL	236	100	81	100	00 110 100			
Statistica	lly signific	cant						

Graph 8



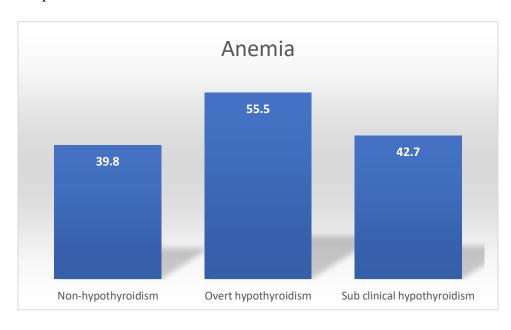
The prevalence of anaemia among hypothyroid individuals is described in the table and graph below.

Microcytic hypochromic anaemia is the most common type (30.8%), and moderate anaemia (42.2%), which is between 7 and 10 gm/dl, is the most common kind among hypothyroid patients. Both overt and subclinical hypothyroidism have positive association with anemia and found to be statistically insignificant (p value >0.05).

TABLE 9- EFFECT OF MATERNAL HYPOTHYROIDISM ON ANEMIA

					ANEMI	A		
	NO		OVERT		SUBCLINICAL		CHI	P
	НҮРОТ	HYPOTHYROID		HYPOTHYROID		HYPOTHYROID		(SIGNIFICANCE)
							VALUE	
	N	%	N	%	N	%	2.503	0.286
YES	94	39.8	45	55.5	47	42.7		
NO	142	60.2	36	44.5	63	57.3		
TOTAL	236	100	81	100 110		100		
Statistica	lly insigni	ficant						

Graph 9



The table and graph below describe the association between need for caesarean deliveries in hypothyroid patients.

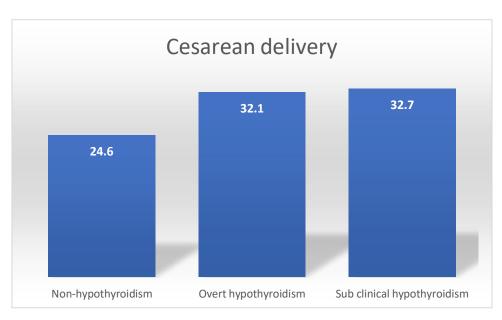
Both overt and subclinical hypothyroidism have impacted on need for caesarean delivery However, using chi square test, statistically no significant difference was observed in our study (p value>0.05)

The most typical of these is the multigravida who has a history of prior caesarean deliveries and elects to have another one.

TABLE 10- NEED FOR CESAREAN DELIVERY IN MATERNAL HYPOTHYROIDISM

				CESA	REAN S	ECTION		
	NO		OVERT		SUBCLI	NICAL	CHI-	P
	HYPOT	HYPOTHYROID		HYPOTHYROID		HYPOTHYROID		(SIGNIFICANCE)
							VALUE	
	N	%	N	%	N	%	1.898	0.755
YES	58	24.6	26	32.1	36	32.7		
NO	178	75.4	55	67.9	74	67.3		
TOTAL	236	100	81	100	00 110 100			
Statistica	lly insigni	ficant						

Graph 10



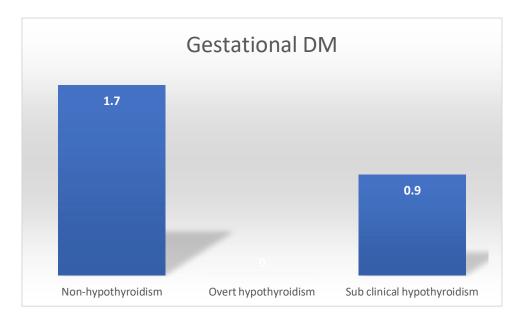
The effect of hypothyroidism on gestational diabetes is described in the table and graph below.

In the subclinical hypothyroidism group, there was just one patient (0.9%) with gestational diabetes, and it was determined to be statistically insignificant (p value >0.05)

TABLE 11- EFFECT OF MATERNAL HYPOTHYROIDISM ON GESTATIONAL DM

				GES	TATION	AL DM		
	NO		OVERT		SUBCLI	NICAL	CHI-	P
	HYPOTHYROID		HYPOTHYROID		HYPOTHYROID		SQUARE	(SIGNIFICANCE)
							VALUE	
	N	%	N	%	N	%	1.303	0.521
YES	4	1.7	0	0	1	0.9		
NO	232	98.3	81	100	109	99.1		
TOTAL	236	100	81	100	110	100		
Statistica	lly insigni	ficant						

Graph 11



This table and graph illustrate the association between maternal hypothyroidism af oligohydramnios.

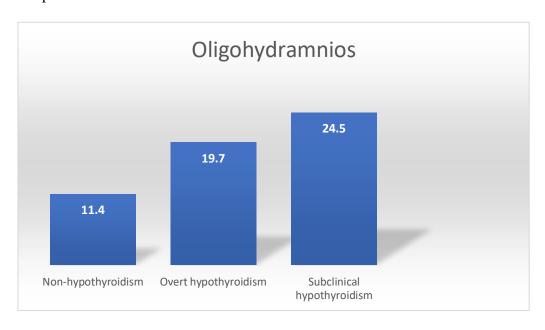
In the subclinical hypothyroidism group, 24.5 percent of oligohydramnios were found compared to 19.7 percent in the overt hypothyroidism group and both have an impact on oligohydramnios

However, it is determined to be statistically insignificant (p value >0.05) using the chi square test

TABLE 12- EFFECT OF MATERNAL HYPOTHYROIDISM ON OLIGOHYDRAMNIOS

				OLIG	OHYDR	AMNIOS					
	NO		OVERT		SUBCLI	NICAL	CHI-	P			
	HYPOTHYROID		HYPOTHYROID		HYPOTHYROID		SQUARE	(SIGNIFICANCE)			
							VALUE				
	N	%	N	%	N	%	3.621	0.164			
YES	27	11.4	16	19.7	27	24.5					
NO	209	88.6	65	80.2	83	75.5					
TOTAL	236	100	81	100	110	100					
Statistica	Statistically insignificant										

Graph 12



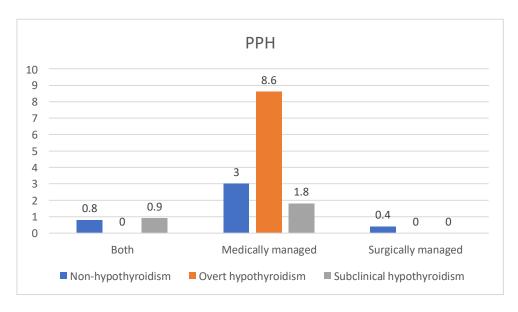
This chart and table illustrate the association of hypothyroidism on PPH.

It is determined to be statistically insignificant (p value >0.05) using the chi square test because only 10 out of 191 hypothyroid individuals (11.3 percent) had PPH, and both overt and subclinical hypothyroidism have an impact on PPH with medically controlled PPH being the most common type.

TABLE 13- EFFECT OF MATERNAL HYPOTHYROIDISM ON PPH

			1	POSTPAR	TUM HE	'UM HEMORRHAGE			
	NO		OVERT	OVERT		SUBCLINICAL		P	
	HYPOTHYROID		НҮРОТ	HYPOTHYROID		HYROID	SQUARE	(SIGNIFICANCE)	
							VALUE		
	N	%	N	%	N	%	9.745	0.136	
ВОТН	2	0.8	0	0	1	0.9			
MEDICALLY	7	3	7	8.6	2	1.8			
MANAGED									
SURGICALLY	1	0.4	0	0	0	0			
MANAGED									
NO	226	95.8	74	91.3	107	97.3			
TOTAL	236	100	81	100	110	100			
Statistically insig	nificant	<u> </u>	l			<u> </u>			

Graph 13



The impact of maternal hypothyroidism on other maternal problems is depicted in this chart and graph.

Among other problems, polyhydramnios (2.5%) and gestational thrombocytopenia (4.3%) were frequently seen in hypothyroid patients

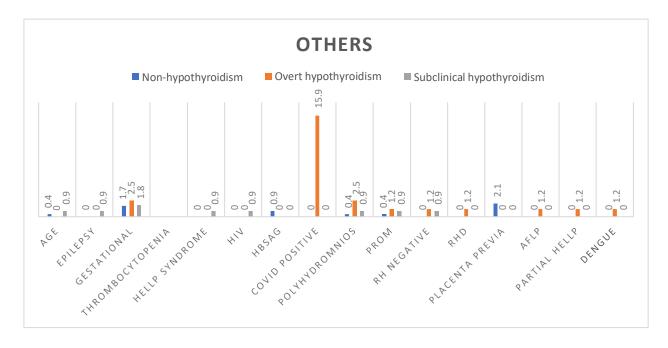
Chi square analysis shows that it has no statistical significance (p value>0.05)

TABLE 14- EFFECT OF MATERNAL HYPOTHYROIDISM ON OTHER MATERNAL COMPLICATIONS

			ГО	THERS				
	NON		OVERT		SUBCI	LINICAL	CHI -	P
	HYPOT	HYR	HYPOTHYROIDI		HYPOTHYROI		SQUA	(SIG
	OIDISM		SM		DISM		RE	NIFI
							VALU	CAN
							Е	CE)
	N	%	N	%	N	%	40.363	0.366
Acute	1	0.4	0	0	1	0.9		
gastroenteritis								
Epilepsy	0	0	0	0	1	0.9		
Gestational	4	1.7	2	2.5	2	1.8		
Thrombocytope								
nia								
HELLP	0	0	0	0	1	0.9		
SYNDROME								
HIV positive	0	0	0	0	1	0.9		
HBSAG	1	0.9	0	0	0	0		
positive								
Covid Positive	0	0	13	15.9	0	0		
Polyhydramnios	1	0.4	2	2.5	1	0.9		
PROM	1	0.4	1	1.2	1	0.9		
Rh negative	0	0	1	1.2	1	0.9	1	
status								

RHD(rheumatic	0	0	1	1.2	0	0	
heart disease)							
Placenta Previa	5	2.1	0	0	0	0	
Acute Fatty	0	0	1	1.2	0	0	
Liver of							
Pregnancy							
Partial HELLP	0	0	1	1.2	0	0	
Dengue	0	0	1	1.2	0	0	
NONE	223	94.1	58	71.9	101	91.9	
TOTAL	236	100	81	100	110	100	
Statistically insignificant							

Graph 14



EFFECT OF MATERNAL HYPOTHYROIDISM ON NEONATAL OUTCOMES

This below graph and table illustrate the association of foetal growth restriction with maternal hypothyroidism.

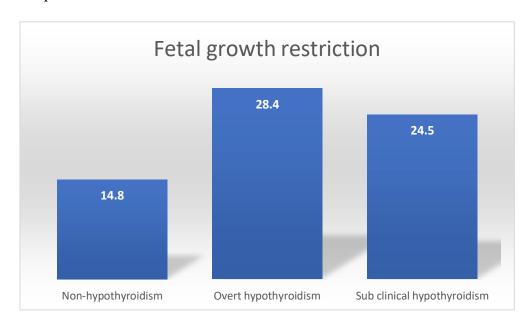
Both subclinical and overt hypothyroidism have affected fetal growth restriction

Despite the fact that there is a clinical difference, the chi square test found no statistically significant difference (p value >0.05)

TABLE 15- EFFECT OF MATERNAL HYPOTHYROIDISM ON FETAL GROWTH RESTRICTION

		FETAL GROWTH RESTRICTION									
	NO		OVERT		SUBCLINICAL		CHI-	P			
	HYPOT	HYPOTHYROID		HYPOTHYROID		HYPOTHYROID		(SIGNIFICANCE)			
							VALUE				
	N	%	N	%	N	%	0.054	0.973			
YES	35	14.8	23	28.4	27	24.5					
NO	201	85.2	58	71.6	83	75.5		,			
TOTAL	236	100	81	100 110 100							
Statistica	lly insigni	ficant									

Graph 15



The effects of maternal hypothyroidism on intrauterine death are depicted in this graph and chart.

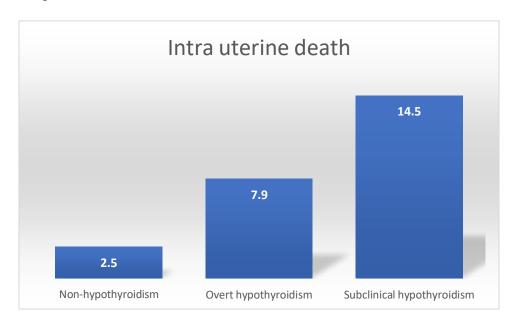
Both overt hypothyroidism and subclinical hypothyroidism have positive corelation with intrauterine death

And in our analysis, these deaths were most frequently observed in preterm patients and it is statistically insignificant (p value>0.05)

TABLE 16- EFFECT OF MATERNAL HYPOTHYROIDISM ON INTRAUTRINE DEATH

		INTRAUTERINE DEATH									
	NO		OVERT		SUBCLI	SUBCLINICAL		P			
	HYPOT	HYROID	HYPOTHYROID		HYPOT	HYPOTHYROID		(SIGNIFICANCE)			
							VALUE				
	N	%	N	%	N	%	5.306	0.070			
YES	6	2.5	6	7.9	16	14.5					
NO	230	97.5	75	92.1	94	85.5					
TOTAL	236	100	81	100	110 100						
Statistica	Statistically insignificant										

Graph 16



We showed the association between maternal hypothyroidism and low birth weight in this chart and graph.

Both subclinical and overt hypothyroidism have affected low birth weight

The most prevalent weight range, 1500–2000 grams, accounts for 18.9% of those between 2000–2500 grams (8.4%)

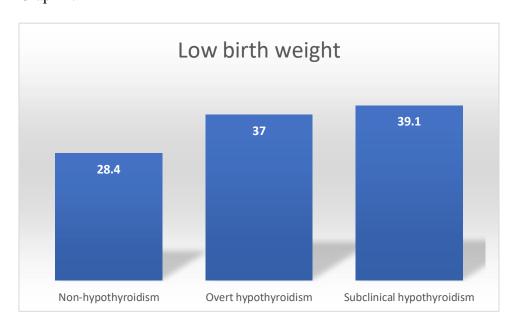
1000-1500 (very LBW) - 6%, 500-1000 (extremely LBW) - 1.7%

Even though there is a clinical difference, the chi square test reveals that it is statistically unimportant (p value>0.05)

TABLE 17- EFFECT ON OF MATERNAL HYPOTHYROIDISM LOW BIRTH WEIGHT

		LOW BIRTH WEIGHT									
	NO		OVERT		SUBCLINICAL		CHI-	P			
	HYPOT	HYROID	HYPOTHYROID		HYPOTHYROID		SQUARE	(SIGNIFICANCE)			
							VALUE				
	N	%	N	%	N	%	5.609	0.061			
YES	67	28.4	30	37	43	39.1					
NO	169	71.6	51	63	67	60.9					
TOTAL	236	100	81 100 110 100								
Statistica	Statistically insignificant										

Graph 17



The graph and figure below illustrate the impact of maternal hypothyroidism on neonatal hyperbilirubinemia.

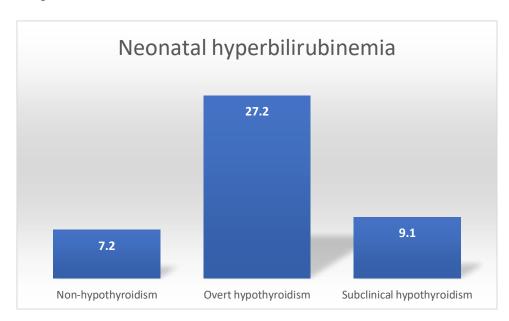
Both overt and subclinical hypothyroidism have affected neonatal hyperbilirubinemia

Using chi square test, clinical significance was determined to be unimportant (p value>0.05)

TABLE 18- EFFECT ON NEONATAL HYPERBILIRUBINEMIA

		NEONATAL HYPERBILIRUBINEMIA									
	NO		OVERT		SUBCLINICAL		CHI-	P			
	HYPOTI	HYROID	HYPOTHYROID		HYPOTHYROID		SQUARE	(SIGNIFICANCE)			
							VALUE				
	N	%	N	%	N	%	3.565	0.168			
YES	17	7.2	22	27.2	10	9.1					
NO	219	92.8	59	72.8	100	90.9					
TOTAL	236	100	81	31 100 110 100							
Statistica	Statistically insignificant										

Graph 18



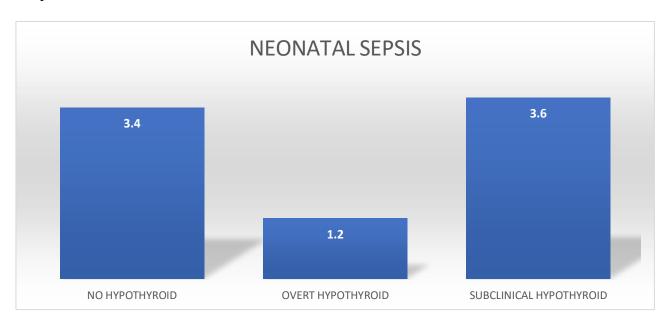
The impact of maternal hypothyroidism on new born sepsis is shown in this table and graph.

Both subclinical and overt hypothyroidism have affected neonatal sepsis, however this effect was shown to be clinically insignificant (p value>0.05)

TABLE 19- EFFECT OF MATERNAL HYPOTHYROIDISM ON NEONATAL SEPSIS

		NEONATAL SEPSIS									
	NO		OVERT		SUBCLINICAL		CHI-	P			
	HYPOTI	HYROID	HYPOTHYROID		HYPOTHYROID		SQUARE	(SIGNIFICANCE)			
							VALUE				
	N	%	N	%	N	%	0.948	0.623			
YES	8	3.4	1	1.2	4	3.6					
NO	228	96.6	80	98.8	106	96.4					
TOTAL	236	100	81	81 100 110 100							
Statistica	Statistically insignificant										

Graph 19



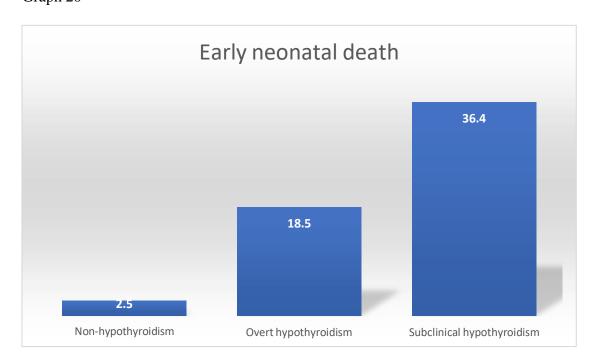
This chart and graph demonstrate the impact of maternal hypothyroidism on early neonatal death.

In our study, the majority of early neonatal deaths—fetuses who pass away within the first six days after birth—occur in the group of people who have both overt and subclinical hypothyroidism and it is statistically insignificant (p value>0.05%)

TABLE 20- EFFECT OF MATERNAL HYPOTHYROIDISM ON EARLY NEONATAL DEATH

		EARLY NEONATAL DEATH									
	NO		OVERT		SUBCLI	NICAL	CHI-	P			
	HYPOT	HYROID	HYPOT	HYROID	HYPOTHYROID		SQUARE	(SIGNIFICANCE)			
						VALUE					
	N	%	N	%	N	%	3.875	0.144			
YES	6	2.5	15	18.5	7	6.4					
NO	230	97.5	66	81.5	103	93.6					
TOTAL	236	100	81	100	110 100						
Statistica	Statistically insignificant										

Graph 20



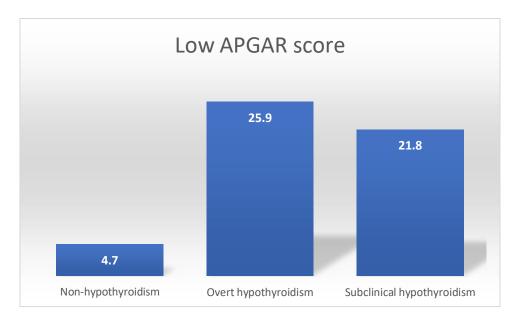
This graph and table demonstrate maternal hypothyroidism effects on APGAR scores.

Both overt and subclinical hypothyroidism have affected APGAR scores. It is determined in our study to be clinically significant using the chi square test (p value- 0.015)

TABLE 21- EFFECT OF MATERNAL HYPOTHYROIDISM ON LOW APGAR SCORE

		LOW APGAR SCORE									
	NO		OVERT		SUBCLI	SUBCLINICAL		P			
	HYPOTI	HYROID	HYPOTHYROID		HYPOTHYROID		SQUARE	(SIGNIFICANCE)			
						VALUE					
	N	%	N	%	N	%	8.414	0.015			
YES	11	4.7	19	25.9	24	21.8					
NO	226	95.3	62	74.1	86						
TOTAL	236	100	81 100 110 100								
Statistica	lly signific	cant									

Graph 21



The effects of maternal hypothyroidism on additional new born problems are depicted in this graph and chart.

These all were affected by overt and subclinical hypothyroidism and determined to be clinically significant (p value>0.05) using the chi square test.

TABLE 22- EFFECT OF MATERNAL HYPOTHYROIDISM ON OTHER FACTORS

		A	ANY O	THERS,	SPECII	F Y		
	NO		OVE	RT	SUBC	LINIC	CHI-	P
	НҮРО	THYR	HYPOTHYR		AL		SQUA	(SIGNIFICA
	OID		OID		HYPO	THYR	RE	NCE)
					OID		VALU	
							Е	
	N	%	N	%	N	%	34.852	0.010
HIE	1	0.4	1	1.2	0	0		
MAS	2	0.8	1	1.2	1	0.9		
RDS	4	1.7	1	1.2	4	3.6		
HYPOGLYCE	0	0	0	0	1	0.9		
MIC								
SEIZURES								
MENINGITIS	0	0	1	1.2	0	0		
BIRTH	0	0	2	2.5	0	0		
ASPHYXIA								
CARDIAC	0	0	1	1.2	0	0	•	
ARREST								
CLEFT LIP	0	0	1	1.2	0	0		
PNEUMONIA	0	0	0	0	1	0.9		
NONE	229	97.1	73	90.3	103	93.7		
TOTAL	236	100	81	100	110	100		
Statistically sign	ificant	L	ı	L	ı	L		

Graph 22

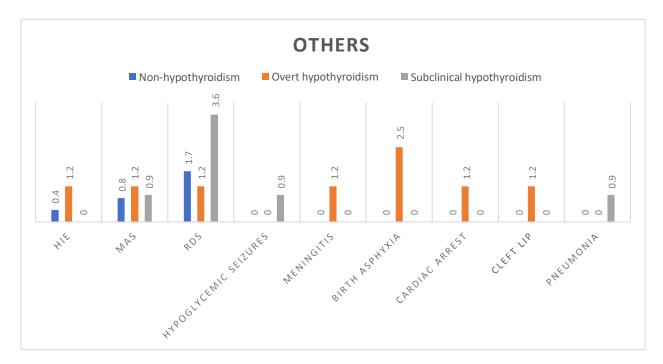


TABLE 23- RELATIONSHIP OF TPO ANTIBODIES AND HYPERTENSIVE DISORDERS OF PREGNANCY

The prevalence and association between TPO antibodies and hypertensive diseases are shown in this graph and chart.

The majority of individuals with TPO antibodies (16.1%) have PE, with severe symptoms being the most prevalent type.

A clinically significant finding was made as well and it is statistically significant (p value>0.05)

	NO TPO	TPO ANTIBODIES PRESENT	CHI- SQUARE VALUE	P (SIGNIFICANCE)
ECLAMPSIA	6 (2.5%)	10 (8.1%)	15.791	0.015
GESTATIONAL HYPERTENSION	11 (11%)	13 (10.5%)		
IMMINENT ECLAMPSIA	2 (0.8%)	5 (4%)		
PE WITH SEVERE FEATURES	20 (8.5%)	20 (16.1%)		
PE WITHOUT SEVERE FEATURES	9 (3.8%)	8 (6.5%)		
CHRONIC HYPERTENSION	0	1 (0.8%)		
Statistically signific	ant			

Graph 23

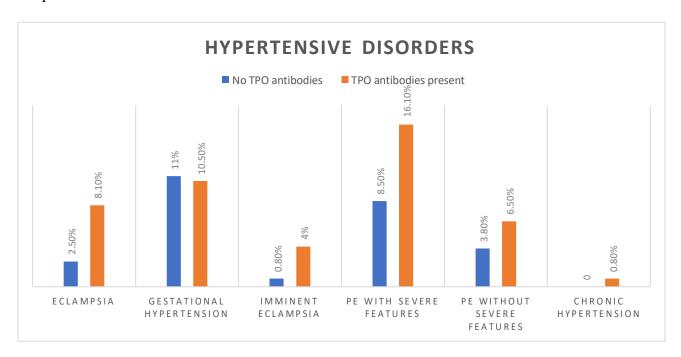


TABLE 24- OBSTETRICAL OUTCOMES CONCERNING TPO ANTIBODIES

The various obstetrical outcomes related to TPO antibodies are displayed in the table and chart below.

Patients with TPO antibodies experience more unfavourable obstetrical outcomes than those who do not, with anaemia (43.5%) and preterm births being the most prevalent (21.8 percent)

Even if there is clinical significance, using chi square charts has no statistical relevance (p value>0.05)

	NO TPO ANTIBODIES	TPO ANTIBODIES
		PRESENT
PPH	10 (4.2%)	6 (4.8%)
ABRUPTION	5 (2.1%)	9 (7.3%)
PRETERM DELIVERY	38 (16.1%)	27 (21.8%)
ANEMIA	94 (39.8%)	54 (43.5%)
CESAREAN SECTION	56 (23.7%)	22 (17.7%)
GESTATIONAL DM	4 (1.7%)	1 (0.8%)
OLIGOHYDRAMNIOS	27 (11.4%)	18 (14.5%)

Graph 24

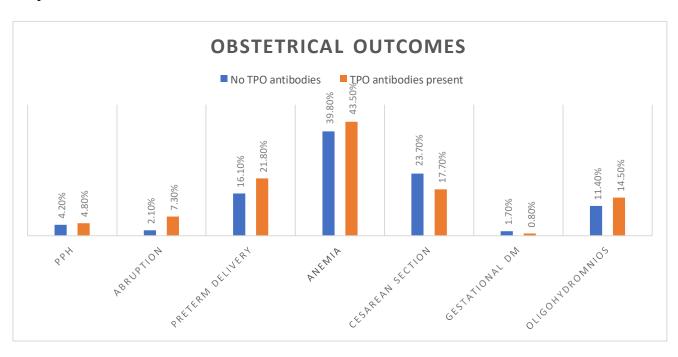


TABLE 25- NEONATAL OUTCOMES CONCERNING TPO ANTIBODIES

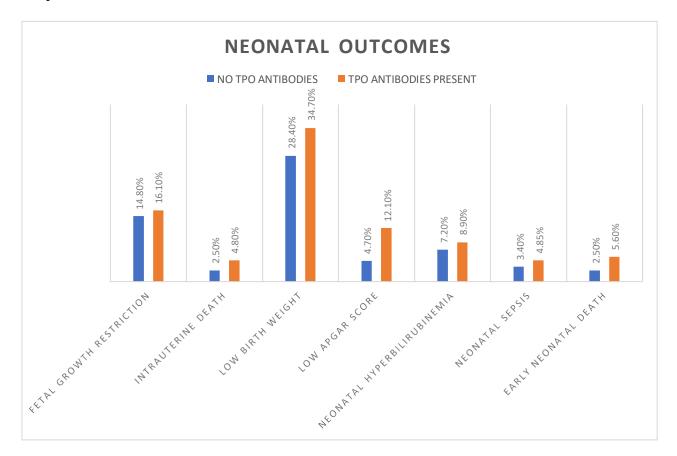
The table and graph below show some negative neonatal outcomes related to TPO antibodies.

Low birth weight (34.7 percent) and foetal development restriction are the most prevalent neonatal outcomes in patients with TPO antibodies (16.1 percent)

No statistical significance (p value>0.05) was discovered despite the clinical importance.

	NO TPO ANTIBODIES	TPO ANTIBODIES PRESENT
FETAL GROWTH	35 (14.8%)	20 (16.1%)
RESTRICTION		
INTRAUTERINE DEATH	6 (2.5%)	6 (4.8%)
LOW BIRTH WEIGHT	67 (28.4%)	43 (34.7%)
LOW APGAR SCORE	11 (4.7%)	15 (12.1%)
NEONATAL	17 (7.2%)	11 (8.9%)
HYPERBILIRUBINEMIA		
NEONATAL SEPSIS	8 (3.4%)	6 (4.850
EARLY NEONATAL	6 (2.5%)	7 (5.6%)
DEATH		

Graph 25



DISCUSSION

This observational study was carried out at B. M. Patil Medical College and Research Institute in Vijayapura. A total of 427 patients underwent screening for hypothyroidism.

The primary goal is to research how maternal hypothyroidism affects obstetrical and neonatal outcomes.

Our study group's average age ranges from 20 years to 24 years.

Primagravida and multigravida with prevalence rates of 41.9 and 58.07 percent, respectively.

In our study, the prevalence of overt hypothyroidism is 18.9%, but Sahu et alsurvey .'s ¹⁰² found only 4.56 percent, which is 13% lower than our study's prevalence.

The prevalence of overt hypothyroidism in the other investigations by Weiping Teng et al. ¹¹⁴, Tuija Mannisto et al. ¹¹⁵, and Stagnaro et al. ¹¹⁶ is substantially lower than in our study at less than 1%, 1.3 percent, and 0.4 percent, respectively.

TABLE 26- Prevalence of Overt Hypothyroidism in various studies

Our study	18.9%
Sahu et al	0.7%
Tuija Mannisto et al	1.3%
Weiping et al	1%
Stagnaro et al	0.4%

Our study found that 25.7 percent of participants had subclinical hypothyroidism, which is similar to the 23 percent found in the survey conducted by Casey BM et al. ¹¹⁷

The prevalence of subclinical hypothyroidism was quite low in the other studies by Sahu et al.¹⁰², Tuiha Mannisto et al.¹¹⁵, and Weiping et al.¹¹⁴, coming in at 6.47 percent, 3.5 percent, and 3.5 percent, respectively.

TABLE 27- Prevalence of subclinical hypothyroidism in various studies

Our study	25.7%
Casey B. Miller and colleagues	23%
Sahu et al.	6.47%
Tuija Mannisto et al.	3.5%
Weiping et al.	3-5%

TABLE 28- Prevalence of subclinical and overt hypothyroidism

	PREVALENCE											
STUDIES	OVERT	SUBCLINICAL										
	HYPOTHYROIDISM	HYPOTHYROIDISM										
Our study	18.9%	25.7%										
Sahu et al	0.7%	6.47%										
Tuija et al	1.3%	3.5%										
Weiping et al	1%	3-5%										

Adverse Obstetric and Neonatal Outcomes in Hypothyroidism: Overt and Subclinical

In our study, the incidence of adverse obstetrical outcomes in overt and subclinical hypothyroidism, respectively, was

• Hypertensive disorders of pregnancy: 20.3%, 48.2%

• Pre-eclampsia: 19.7%, 20%

• Abruption: 7.4%, 3.6%

• Preterm deliveries: 44.4%, 27.3%

• Anemia: 55.5%, 42.7%

• Cesarean deliveries: 52.1%, 32.7%

• Oligohydramnios: 19.7%, 24.5%

• PPH- 8.6%, 2.7%

The incidence of adverse neonatal outcomes is higher in overt hypothyroidism than in subclinical hypothyroidism.

- FGR- 28.4%, 24.5%
- IUD- 7.9%, 14.5%
- LBW- 37%, 39.1%
- Neonatal hyperbilirubinemia: 27.2%, 9.1%
- Neonatal sepsis: 1.2%, 3.6%
- Early neonatal deaths: 18.5%, 6.4%
- Low APGAR scores: 25.9%, 21.8%

OVERT HYPOTHYROIDISM

In the study done by Sahu MT et al.,¹⁰² the incidence of complications like PE (20.7%), preterm deliveries (4.7%), and FGR (13.8%)

In another similar study by Leung et al. ¹¹⁸, incidences were 22% for PE and 22% for LBW, which is very similar to our study.

In the study by Abolovich et al. ¹¹⁹, complications such as placental abruption (19%) and LBW (6%) were identified.

In our study, there is a significant association between overt hypothyroidism and complications like hypertensive disorders of pregnancy, preterm deliveries, abruption placenta, low APGAR scores, pneumonia, and birth asphyxia.

The incidence of complications varied in different studies, but some complications, like preeclampsia and abruption incidences, are comparable.

TABLE 29- Incidence of adverse obstetrical and neonatal outcomes in various studies of overt hypothyroidism

STUDIES	PE	ABRUPTIO	PRETERM	FGR	LBW
		PLACENTA	DELIVERIES		
Our study	19.7%	7.4%	44.4%	28.4%	37%
Leung et al	22%	-	-	-	22%
Sahu MT et al	20.7%	-	4.7%	13.8%	-
Ablovich et al	-	19%	-	-	6%

SUBCLINICAL HYPOTHYROIDISM

In a study conducted by Leung et al. ¹¹⁸, the incidence of adverse outcomes such as PE (15%) was very comparable to our study, whereas the incidences of PTD (9%), LBW (9%), and other outcomes were very low compared to our study.

In the other similar study by Sahu et al. ¹⁰² there were complications like PE (9.8%), PTD (10.35), and FGR (2.4%), but in these two studies there were no complications like abruption and low APGAR scores, which were very significant in our study at 3.6% and 21.8%, respectively.

TABLE 30- Incidence of adverse obstetrical and neonatal outcomes in various studies of subclinical hypothyroidism

STUDIES	PE	ABRUPTIO	PTD	FGR	LBW	LOW
		PLACENTA				APGAR
						SCORE
Our study	20%	3.6%	27.3%	24.5%	39.1%	21.8%
Leung et al	15%	-	9%	-	-	-
Sahu et al	9.8%	-	10.3%	2.4%	2.5%	-

The presence of TPO antibodies and adverse obstetrical and neonatal outcomes

In our study, for all women whose TSH levels were >3 microIU/L, TPO antibody levels were measured, and we found that there was a higher incidence of adverse obstetrical and neonatal outcomes in women with the presence of TPO antibodies compared to women without antibodies.

When antibody levels exceeded 0.9 IU/ml, the negative effects became more pronounced.

In our study, a significant correlation was drawn between the presence of TPO antibodies and hypertensive disorders of pregnancy.

- TPO antibodies were found to increase the risk of pre-eclampsia in a cohort of over 600 women participating in a comparable study by Van De Boogard et al. 46 (1996).
- Similar to our findings, in other study ¹²⁰ found that a patient with a high titer of TPO antibodies had a three-fold increased probability of experiencing an abruption (7.3 percent).
- In addition, Haddow et al. conducted a large cohort study.

Two big sample size meta-analyses:-

- He et al. 122 and
- Thangaratinam et al. 123

Both demonstrated an increased risk of preterm birth with the presence of antibodies, which is very similar to our study (21.8%).

In our study, the incidence of LBW associated with TPO antibodies was 34.7%, as similarly proved by the other research conducted by Chen et al.¹²⁴

LIMITATIONS

To accept our weaknesses,

- Because TSH is assessed after 72 hours, according to our study, neonates who passed away within that time frame could not have had their TSH levels checked.
- Regarding the treatment of hypothyroidism, there was no intervention in our study.
- Patients with high free T4 and TSH levels were not examined collectively.
- The prevalence and unfavourable outcomes of multifetal gestation pregnancy could not be evaluated since it was not included in the study.

CONCLUSION

The prevalence of thyroid problems in our study, including overt and subclinical hypothyroidism, is very high. In order to forecast negative obstetrical and neonatal outcomes and provide prompt care, we advise routine thyroid screening for all pregnant women, especially in the third trimester.

SUMMARY

- At the B. M. Patil Medical College and Research Institute in Vijayapura, the current study was carried out.
- An observational research was conducted.
- 427 pregnant women in their third trimester who were arriving at the labour and delivery wards for birth were part of the screening process.
- TSH levels were assessed in each woman.
- To investigate the frequency of overt, subclinical, and autoimmune hypothyroidism and its effects on obstetrical and neonatal outcomes, free T4 and TPO antibodies were supplied if TSH was greater than 3 microIU/litre.
- 18.9 percent of people have overt hypothyroidism, whereas 25.7 percent have subclinical hypothyroidism.
- Thirty-seven percent of the women who were examined had TPO antibodies.
- In our study, the effects of both overt and subclinical maternal hypothyroidism on obstetrical outcomes, respectively, were: hypertensive disorders of pregnancy: 20.3%, 48.2%; preeclampsia: 19.7%, 20%; abruption: 7.4%, 3.6%; preterm deliveries: 44.4%, 27.3%; anemia: 55.5%, 42.7%; caesarean sections: 52.1%, 32.7%; oligohydramnios: 19.7%, 24.5%; PPH: 8.6%, 2.7%
- In our study, the effects of both overt and subclinical maternal hypothyroidism on neonatal outcomes were, respectively, FGR: 28.4%, 24.5%, IUD: 7.9%, 14.5%, LBW: 37%, 39.1%, Neonatal hyperbilirubinemia: 27.2%, 9.1%, Neonatal sepsis: 1.2%, 3.6%, Early neonatal deaths: 18.5%, 6.4%; low APGAR scores: 25.9%, 21.8%
- In our study, we found that there is a strong association between hypertensive disorders and the presence of TPO antibodies.
- Women with TPO antibodies had a higher incidence of adverse obstetrical and neonatal outcomes than women without TPO antibodies.

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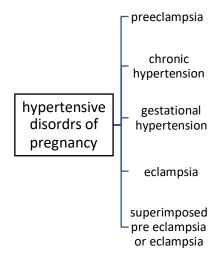
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ANNEXURE I

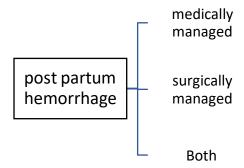
PROFORMA

Case No: Name: Age: ADDRESS:	IP No. : PHONE NO :
A. OBSTETRIC HISTORY	
Obstetric Score-	Months of amenorrhoea-
B. MENSTRUAL HISTORY	
POG by dates:	
POG by 1 st -trimester scan	
C. GENERAL PHYSICAL EXPANSION: Icterus:	XAMINATION Pulse: B.P:
Oedema :	BMI – weight in kg/(height in
metres) ²	
1. PERSONAL HISTORY: I 1. YES 2. NO	IS SHE K/C/O HYPOTHYROIDISM?
2. ADVERSE OUTCOMES	



ABRUPTIO PLACENTA

0



- o PRETERM DELIVERIES
- \circ ANEMIA (HB<11g/dl)
- o INCIDENCE OF CAESAREAN SECTION
- o GESTATIONAL DIABETES MELLITUS
- o OLIGOHYDROMNIOS
- o ANY OTHERS(SPECIFY)

3. INVESTIGATIONS

Thyroid Hormone profile

1. TSH • Hb

If TSH is abnormal these investigations will be

Performed:

MCH

MCV

2. FREE T4

• MCHC

3. TPO ANTIBODIES

• PLT

BABY PROFORMA

• WBC

- > BIRTH WEIGHT
- ➤ APGAR SCORE AT 1 MINUTE
- TIME OF PASSAGE OFMECONIUM AFTER BIRTH
- ➤ PRESENCE OF ICTERUS /JAUNDICE
- > PHYSICAL APPEARANCE (COARSE FACIES)
- ➤ COARSE CRY
- > UMBILICAL HERNIA
- > LARGE FONTANELLE

ADVERSE PERINATAL OUTCOMES

- o FETAL GROWTH RESTRICTION
- o INTRAUTERINE DEATH
- LOW BIRTH WEIGHT
- LOW APGAR SCORE
- o NEONATAL HYPERBILIRUBINEMIA
- o SEPSIS
- o EARLY NEONATAL DEATH
- ANY OTHERS(SPECIFY)

INVESTIGATIONS

 \circ TSH

If clinically icterus is present then, this will be performed:

o BILIRUBIN

ANNEXURE II

INFORMED CONSENT FOR PARTICIPATION IN DISSERTATION/RESEARCH

I, the undersigned,	, S/O D/O W/O_		,	aged_	years,
ordinarily resident of	do hereby state/de	eclare that	Dr. GAURI	BAN	KAPUR
of Shri. B. M. Patil Medical Co	ollege Hospital and R	esearch Co	entre have e	examin	ed me
thoroughly on	at	(place)	Further	DR	GAURI
BANKAPUR has informed me th	at he/she is conducting	dissertatio	n/research ti	itled "I	MPACT
OF PREEXISTING AND NEW	LY DETECTED MA	TERNAL	НҮРОТНҮ	ROIDI	SM ON
MATERNAL AND PERINATA	L OUTCOME DURI	NG LATE	PREGNAN	CY AN	1
OBSERVATIONAL STUDY" un	nder the guidance of DI	R RAJASR	I G YALIW	AL. A	ccording
to this my blood sample will be ta	ken to assess my thyro	id function	and I will be	e moni	tored for
any fetal or maternal adverse even	nts. Further Doctor has	s informed	me that my	partici	pation in
this study will help to get useful in	formation that can help	in the futu	re treatment	of suc	h similar
cases.					

The Doctor has also informed me that information given by me, observations made/photographs/video graphs taken upon me by the investigator will be kept secret and not assessed by the person other than me or my legal hirer except for academic purposes.

The Doctor did inform me that though my participation is purely voluntary, based on the information given by me, I can ask for any clarification during treatment/study related to diagnosis, the procedure of treatment, result of treatment, or prognosis. At the same time, I have been informed that I can withdraw from my participation in this study at any time if I want or the investigator can terminate me from the study at any time study but not the procedure of treatment and follow-up unless I request to be discharged.

The doctor did inform me that though my baby's participation is purely voluntary and also, I can ask for any clarification during treatment/study related to diagnosis, the procedure of treatment, result of treatment, or prognosis. At the same time, I have been informed that I can withdraw the consent for my baby's participation in this study at any time if I want or the investigator can terminate me from the study at any time but not the procedure of treatment and follow-up unless I request to be discharged

After understanding the nature of dissertation or research, diagnosis made, mode of treatment. I am giving consent for the blood investigations and also for the follow-up.

I the undersigned Shri/Smt	_under my	full con	SC10U	is state
of mind agree to participate in the said research/dissertation	and also	agree to	my	baby's
participation in this said research/dissertation				
Signature of patient:				
Signature of doctor:				
Witness: 1.				
Date:				
Place:				

ANNEXURE III

ETHICAL COMMITTIEE CLEARANCE



B.L.D.E. (DEEMED TO BE UNIVERSITY) Date - 22 101 2001

(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act, 1956)

The Constituent College

SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Institutional ethical committee of this college met on 11-01-2021 at 11-00 am to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

Title: Impact of preexisting & newly detected maternal hypothyroidism on maternal & perinatal outcome during late pregnancy an observational study.

Name of PG student: Dr Gauri Bankapur, Department of Obst/Gynaec

Name of Guide/Co-investigator: Dr Rajasri.G.Yaliwal, Associate Professor of Obst/Gynaec

CHAIRMAN, IEC

Institutional Ethical Committee B L D'E (Deemed to be University) Shri B.M. Patil Medical College, VIJAYAPUR-596103 (Karnataka)

Following documents were placed before Ethical Committee for Scrutinization:

- 1. Copy of Synopsis / Research project
- 2. Copy of informed consent form
- 3. Any other relevant documents.

5

ANNEXURE IV

MASTER CHART

age	address	obstetric score	Period of gestation K/C/O hypothyroidism	hypertensive disorders of pregnancy	post partum hemorrhage	abruptio placenta	preterm delivery	incidence of C- Section	gestational DM	oligohydromnios	any others, specify	hemoglobin	MCV	PLATELETS	TSH	free T4	not done	Which type of hypothyroidism	birth weight	APGAR score at 1 fetal growth restriction	intrauterine death	low birth weight	neon	neonatal sepsis early neonatal death	specify, if others	baby TSH
2 4	Indi	10a	40 no	reatures	None	no	no n	o no	no	no	none	11. 7	78. 7	2.21	2.3	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	no 1	no no	none	not done
3	Indi	primigra vida	37 no	PE with severe features	None	no	no y	no	no	no	none	6.9	85. 4	1.9	1.93	not done	not done	No hypothyroidism	1500-2000 gms	$7 \begin{vmatrix} ye \\ s \end{vmatrix}$	no	ye s no	no 1	no no	none	not done
2 5	Vijayapura	multigrav ida	42 no	none	None	no	no n	o no	no	no	Rh negative status	13. 1	85. 3	2.57	2.1	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no l	no no	none	not done
2 7	Vijayapura	multigrav ida	32 no	imminent eclampsia	None	no	ye s	o ge	no	ye s	none	13	83. 2	2.04	1.6	not done	not done	No hypothyroidism	1000-1500 gms	7 no	no	ye no	no 1	no no	none	not done
2 4	Sindagi	primigra vida	29 no	imminent eclampsia	None	no	ye y	no	no	no	none	10	89. 6	1.51	1.4	not done	not done	No hypothyroidism	500-1000gms	3 no	no	ye ye s s	no 1	no ye	none	not done
2 0	Vijayapura	multigrav ida	40 no	none	None	no	no n	ye s	no	no	none	12. 8	90. 2	2.4	2.3	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no 1	no no	none	not done
2 2	Sindagi	multigrav ida	41 no	PE with severe features	None	no	ye y	e ye	no	no	none	8.5	69. 5	1.96	1.7	not done	not done	No hypothyroidism	2000-2500 gms	7 no	no	ye s no	no 1	no no	RDS	not done
2 7	Vijayapura	multigray	35 no		None	no	ye y	e no	no	no	none	9.1	79. 3	1.71	1.15	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	no 1	no no	RDS	not done
2 2	Vijayapura	multigray	44 no	none	None	no	no y	e no	no	no	none	8.4	105 .4	2.52	2.75	not done	not done	No hypothyroidism	2000-2500 gms	5 ye	no	ye ye	no 1	no ye	none	not done
2 5	Babaleshwara	neimiara	39 no	None	None	no	no n	o no	no	ye	none	11.	84.	2.45	1.78	not done	not done	No hypothyroidism	more than 3000 gms	8 no	ye	no no	no 1	no no	none	not done
3	Bijapur	multiamary	39 no	None	None	ye	no n	o no	no	no	none	11.	92. 8	2	1.7	not done	not done	No hypothyroidism	2500-3000 gms	8 no	no	no no	no 1	no no	none	not done
2	Bijapur	primiaro	39 no	None	None	no	no y	e no	no	no	none	10.	80.	1.8	2.29	not	not done	No hypothyroidism	2000-2500 gms	7 no	no	no no	no 1	no no	none	not done
3	Bijapur	multigray	40 no	None	None	no		e ye	no	no	none	9.9	89	2.12	1.02	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	no 1	no no	none	not done
1	Jamkandi	primiaro	29 no	None	None	no	ye y	e ye	no	ye	none	9.1	84.	3.03	1.9	not done	not done	No hypothyroidism	1000-1500 gms	4 no	no	ye ye		no ye	none	not done
3	Itagi	multigray	40 no	None	None	no	no y	e ye	no	no	none	8.6	81.	1.65	1.91	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	ye 1	no no	none	not done
2 8	Bijapur	multigray	39 no	None	None	no	no n	o no	no	no	none	11	79. 4	2.3	2.45	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no 1	no no	none	not done
2 3	Tadavalaga	multigray	38 no	None	both	no	no y	e ye	no	no	none	10.	108	1.68	2.9	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no 1	no no	none	not done
2 7	Bijapur	multigray	39 no	None	None	no	no n	o no	no	ye s	none	13.	86.	1.88	2.02	not done	not done	No hypothyroidism	2000-2500 gms	7 no	no	no no	no 1	no no	none	not done
2 0	Bijapur	nnimiano	39 no	None	None	no	no y	e no	no	no	Gestational thrombocytopenia	9.1	73	1.1	2.67	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	no 1	no no	none	not done
3	Indi	multigray	36 no	None	None	no	ye n	o no	no	no	none	13.	89.	1.99	2.17	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	ye 1	no no	none	not done
2 6	Bijapur	muinai ana	40 no	None	None	no	no n	o no	no	no	none	14.	92. 4	1.51	1.92	not done	not done	No hypothyroidism	2000-2500 gms	7 ye	no	ye no	no 1	no no	none	not done
3	Bijapur	mimiano	38 no	None	None	no	no y	e no	no	no	none	7.9	77.	1.01	1.3	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	no 1	no no	none	not done
$\begin{bmatrix} 2 \\ 0 \end{bmatrix}$	Indi	primiara	38 no	None	None	ye	no n	o no	no	ye	none	13	90	2.5	1.6	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	ye 1	no no	none	not done
2	Bijapur	mys1ti amary	37 no	None	None	no	no n	ye ye	no	ye	none	12	87	2.62	2.3	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	ye 1	no no	none	not done
2	Bijapur	14.i o	39 no	None	None	no	no n	ye s	ye	no	none	12. 2	89	2.62	2.6	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no 1	no no	none	not done
2	Indi	multigray	39 no	gestational hypertension	None	no	no y	e ye	no	no	none	10.	72. 3	1.67	2.11	not done	not done	No hypothyroidism	2500-3000 gms	6 no	no	no no	no 1	no no	none	not done
2	Bijapur	multigray	40 no	V 1	None	no	no y	e no	no	no	none	10.	79. 5	2.09	1.67	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	ye s	no no	none	not done
3 0	Gulbarga	multigrav ida	39 no	None	None	no			no	no	none	12	90. 4	3.04	1.9	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no		no no	none	not done

$\begin{bmatrix} 2 \\ 3 \end{bmatrix}$	Todalabagi	multigrav 39 no	None	None	no	no n	o ye	no	ye	none	12.	91. 7	1.56	2.08	not	not	No hypothyroidism	more than	7 no	o no	no l	no n	o no	no	none	not
2	Indi	ida 39 no multigrav ida 38 no		None	no	no y	e no	o no	no	Central placenta previa	9.8	86.	2.07	2.2	not	not done	No hypothyroidism	3000 gms 2500-3000	7 no	o no	no 1	no n	o no	no	none	not done
2 2	Kalburgi	primigra vida 38 no	None	None	no	no n	o no	o no	ye	none	12.	83.	1.94	2.18	not	not done	No hypothyroidism	gms more than 3000 gms	7 no	o no	no 1	no n	o no	no	none	not done
2 5	Bijapur	multigrav ida 40 no	None	None	no	no n	o ye	no	ye	Placenta previa	14	89. 4	2.1	2.6	not done	not done	No hypothyroidism	more than 3000 gms	7 no	o no	no 1	no n	o no	no	none	not done
2 5	Bijapur	primigra vida 38 no	None	None	no	no y	e no	o no	no	Rh negative status	10.	92	2.42	1.9	not done	not done	No hypothyroidism	more than 3000 gms	7 no	o no	no 1	no n	o no	no	none	not done
2 4	Bijapur	multigrav 31 no	None	None	no	ye s	o no	o no	ye	none	13.	86. 4	2.6	1.09	not done	not done	No hypothyroidism	1000-1500 gms	6 no	o no	ye s	no n	o no	no	none	not done
2 8	Chadchan	multigrav do no	None	None	no	no n	o ye	no	no	none	11.	86	2.45	1.3	not done	not done	No hypothyroidism	2500-3000 gms	7 no	o no	no 1	no n	o no	no	none	not done
2 5	Hathahalli	multigrav ida 29 no	None	None	no	ye y	e no	o no	no	none	6.7	84. 1	2.23	1.45	not done	not done	No hypothyroidism	1000-1500 gms	0 no	o ye		ye s	o no	no	none	not done
2 6	Bijapur	multigrav ida 38 no	None	None	ye s	no y	e no	no	no	none	8.1	70. 1	1.35	2.6	not done	not done	No hypothyroidism	2000-2500 gms	7 yo	no	ye s	no n	o no	no	none	not done
2 6	Muddebihal	primigra vida 40 no		None	no	ye s n	o no	no	no	none	13. 3	92. 7	1.46	1.8	not done	not done	No hypothyroidism	more than 3000 gms	7 no	o no	no i	no n	o no	no	none	not done
2 6	Bijapur	multigrav ida 40 no	PE with severe features	None	no	no y	e no	no	no	none	7.6	78. 2	89	1.02	not done	not done	No hypothyroidism	2500-3000 gms	7 no	o no	no 1	no n	o no	no	none	not done
2 6	Bijapur	multigrav ida 39 no	None	None	no	no n	o s	no	no	none	13.	94.	1.35	1.22	not done	not done	No hypothyroidism	2500-3000 gms	7 no	o no		no n	o no	no	none	not done
2 6	Bijapur	multigrav da no	None	None	no	no n	o s	no	no	none	13.	90. 1	2.2	1.99	not done	not done	No hypothyroidism	2500-3000 gms	7 no	o no	ye s	no n	o no	no	none	not done
2 6 2	Tikota	primigra vida 40 no	None	None	no	no n	o no	o no	no	none	11. 7	79. 4	3.3	1.90 7	done	not done	No hypothyroidism	more than 3000 gms	7 no	o no	no i	no n	o no	no	none	done
6 2	Bijapur	multigrav 41 no	None PE with severe	None	ye s		o no	no	no	none	12. 2	81. 3	3.01	2.03	not done	not done	No hypothyroidism	2500-3000 gms 2000-2500	7 no	o no		no n	o no	no	none	not done
7	Indi	multigrav de	features	None	no	S	o no		no	Thrmbocytopenia	10.	77.	20	1.9	not done	done	No hypothyroidism	gms 2500-3000	7 no	o no	ye s	no n	o no	no	none	not done not
$\begin{bmatrix} 2\\2\\2 \end{bmatrix}$	Solapur	ida 37 no		None	no	no y	s s	no	no	none	6	6 96.	2.03	6	not done not	not done not	No hypothyroidism	gms 2500-3000	7 no	o no	no 1	no n	o no	no	none	done
3	Bijapur	ida 39 110		None	no	no n	Ve	5		none	12	1 87.	2.52		done	done	No hypothyroidism	gms 2000-2500			no 1	V	re.		none	done
3	Indi	ida 36 no	DE without savers	None	no	S	s s	по	no	none	5	94.		1.02	done	done	No hypothyroidism	gms more than			no 1	10	S IIO	no	none	done
5	Kalagi	ida 39 110	features	None	no ye	1	S		no	none	6	3 86.	1.97	2.92	done	done	No hypothyroidism	3000 gms more than			no 1			no	none	done
7 2	Bijapur	vida 38 no		None	S	no n				none	3 12.	9 93.	1.91	2.6	done	done	No hypothyroidism	3000 gms more than			no i			no	none	done not
5 2	Bagewadi Babaleshwara	vida 37 no multigrav 38 no	-	None None		no n	V	.	no	Rh negative status	3	5 81.	2.65	8 1.02	done not	done not	No hypothyroidism No hypothyroidism	3000 gms 2500-3000			no no no	v	re.	no	none	done not
2	Babaleshwara	primigra 38 no		medically		no n	S			none	5 12.	3 103	202	2.80	done	done not	No hypothyroidism	gms 2500-3000			no i		S		none	done not
3	Vijayapura	primigra 40 no		managed None		no n				none	7	.5 90.	2.82	0.25	done	done	No hypothyroidism	gms 2500-3000		o no				no	none	done
2	Bijapur	primigra 27 no		None		no n			ve	none	11	99.	2.78	2 17	not	not	No hypothyroidism	gms 1500-2000	6	e no	ve	no n			none	not
2	Indi	vida 37 no multigrav d1 no		None	no	v	e.		s no	none	9.7	81. 8	2.64	0.76	not	not	No hypothyroidism	gms more than	8 no	;	s no 1			no	none	not
2	Muddebihal	multigrav ida 38 no		None		1	8	o no		none	11.	91. 4	3.04	0.9	not	not done	No hypothyroidism	3000 gms 1500-2000		o no				no	none	not done
2 0	Middebihal	primigra vida 29 no	None	None	no	ye n	o no	o no	no	Polyhydromnios	13.	88.	2.17	1.24	not done	not done	No hypothyroidism	gms 500-1000gms	4 no	o no	ye	ye n	o no	ye	none	not done
2	Akhalkot	multigrav ida 38 no	None	None	no	no n	o ye	no	no	none	11.	79. 3	2.42	2.67	not done	not done	No hypothyroidism	2000-2500 gms	7 no	o no	ye s	no n	o no	no	none	not done
2 5	Indi	multigrav 39 no	None	None	no	no n	o ye	no	no	none	13.	94. 5	1.96	2.18	not done	not done	No hypothyroidism	2500-3000 gms	7 no	o no	no 1	no n	o no	no	none	not done
2 6	Bijapur	multigrav ida 37 no	gestational hypertension	None	no	no n	o ye	по	no	none	11.	87. 2	1.93	2.16	not done	not done	No hypothyroidism	more than 3000 gms	8 no	o no	no 1	no y	re no	no	none	not done
2 0	Bagewadi	primigra vida 40 no	**	surgically managed	no	no y	e ye	no	no	none	9.2	86	2.04	1.85	not done	not done	No hypothyroidism	more than 3000 gms	7 no	o no	no i			no	none	not done
2 3	Bijapur	multigrav ida 37 no	None	None	no	no n				none	11. 1	88. 6	1.56	1.10 9	not done	not done	No hypothyroidism	2000-2500 gms	7 no	o no	no 1	no n	o no	no	none	not done
2 3	Telabagal	multigrav ida 38 no	None	None	no	no n	o ye	no	no	none	11. 6	100 .5	2.43	1.88 9	not done	not done	No hypothyroidism	2000-2500 gms	7 no	o no	no l	no n	o no	no	none	not done

$\begin{bmatrix} 2 \\ 0 \end{bmatrix}$	Bijapur	primigra vida	41 no	None	None	no	no	ye	no l	no no	none	10.	80	2.18	2.62	not done	not done	No hypothyroidism	more than 3000 gms	7	no	no	no no	no	no	no	none	not done
2 4	Bijapur	primiaro	38 no	None	None	no	no	no	no 1	no no	none	11.	90. 5		1.32	not done	not done	No hypothyroidism	2500-3000 gms	7	no	no	no no	o no	no	no	none	not done
2	Athani	multigrav ida	39 no	gestational hypertension	None	no	no	no	no 1	no no	none	13.	90.		1.46	not done	not done	No hypothyroidism	more than 3000 gms	8	no	no	no no	o no	no	no	none	not done
2 3	Bijapur	primiaro	37 no	• 1	None	no	no	ye	no 1	no no	none	8.7	72. 6	. 3.64	1.74	not done	not done	No hypothyroidism	2000-2500 gms	7	no	no	no no	o no	no	no	none	not done
2	Muddebihal	primiaro	39 no	None	None	no	no	ye	no 1	no no	none	8.7	72. 9	. 2.31	2.34	not done	not done	No hypothyroidism	2500-3000 gms	7	no	no	no no	o no	no	no	none	not done
3 0	Chadchan	multigray	36 no	None	None	no	no	ye s	no 1	no no	none	10.	89. 6	. 2.02	2.26	not done	not done	No hypothyroidism	2500-3000 gms	7	no	no	no no	o no	no	no	none	not done
2 2	Bijapur	neimiara	38 no	None	None	no		ye s	no 1	no no	none	9.5	78. 7	3.15	2.2	not done	not done	No hypothyroidism	more than 3000 gms	7	no	no	no no	no	no	no	none	not done
2 3	Bijapur	multigray	41 no	None	None	no	no	ye s	no 1	no no	none	10	74. 1	. 2.72	2.12	not done	not done	No hypothyroidism	more than 3000 gms	7	no	no	no no	no	no	no	none	not done
2 1	Bijapur	multigrav ida	38 no	None	None	no	no	ye s	no 1	no no	none	9.8	76. 1	. 3.2	1.88	not done	not done	No hypothyroidism	more than 3000 gms	7	no	no	no no	no	no	no	none	not done
2 7	Indi	primigra vida	39 no	None	None	no	ye s	ye s	no 1	no no	none	10.	76. 5		2.22	not done	not done	No hypothyroidism	more than 3000 gms	7	no	no	no no	no	no	no	none	not done
3 3	Bagewadi	multigrav ida	37 no	None	None	no	no	no	ye s	no no	none	12. 1	79. 8		2.68	not done	not done	No hypothyroidism	more than 3000 gms	7	no	no	no no	no	no	no	none	not done
3 4	Bijapur	multigrav ida	37 no	eclampsia	None	no	no	no	no l	no no	none	12. 2	85. 7	4.23	1.87 5	not done	not done	No hypothyroidism	2000-2500 gms	7	ye s	no	ye s no	no	no	no	none	not done
2 5	Indi	multigrav ida	34 no	None	None	no	ye s	no	no 1	no no	none	13. 2	87. 6	2.14	0.79 5	not done	not done	No hypothyroidism	1500-2000 gms	7	no	no	ye ye	no	no	no	none	not done
2 4	Bujapur	vida	40 no	None	None	no	no	no	no 1	no ye	none	12	90		2.8	not done	not done	No hypothyroidism	2500-3000 gms	7	no	no	no no	no	no	no	none	not done
2 2	Babaleshwara	vida	40 no	None	None	no	no	no	no 1	no no	none	12. 2	86. 7	2.81	2.1	not done	not done	No hypothyroidism	2500-3000 gms	7	no	no	no no	ye s	no	no	none	not done
2 9	Bagewadi	lua	38 no		None	no	no	no	no 1	no no	none	12. 6	90. 3	3.42	1.81	not done	not done	No hypothyroidism	2500-3000 gms	7	no	no	no no	no	no	no	none	not done
2 2	Talikoti	primigra vida	35 no	PE without severe features	None	no	no	ye s	no 1	no no	none	7.7	81		1.2	not done	not done	No hypothyroidism	1500-2000 gms	7	no	no	ye s no	no	no	no	none	not done
5	Indi	viaa	39 no		None	no	no	no	no 1	no no	none	11.	89. 6	3.12	2.2	not done	not done	No hypothyroidism	2500-3000 gms	6	no	no	no no	no	no	no	none	not done
2 6	Bijapur	ıda	31 no	PE with severe features	None	ye s	no	ye s	no 1	no no	none	7.7	75. 8	2.54	1.61	not done	not done	No hypothyroidism	500-1000gms	5	no	no	ye ye s s	no	ye s	ye s	none	not done
2 3	Bagewadi	ıda	30 no	None	None	no	ye s	no	no 1	no no	none	12. 1	96. 4	1.98	1.09	not done	not done	No hypothyroidism	500-1000gms	6	no	no	ye s no	no	no	no	none	not done
5	Indi	ıda	40 no	None	None	no	no	no	ye s	no no	none	11.	87. 7	2.57	2.82	not done	not done	No hypothyroidism	2500-3000 gms	7	no	no	no no	no	no	no	none	not done
1	Indi	ıda	39 no	None	None	no			no 1	no no	none	11.	93. 5	2.5	2.1	not done	not done	No hypothyroidism	2500-3000 gms			no	no no			no	none	not done
2 2	Bijapur	vida	37 no	None	None	no	no	ye s	no 1	no no	Hellp syndrome	10.	85	00	2.65	not done	not done	No hypothyroidism	1500-2000 gms	6	ye s	no	ye s no	ye s	no	no	none	not done
2 4	Bijapur	primigra vida	35 no	None	None	no	ye s	no	no 1	no ye	none	11.	87. 2	2.9	1.58	not done	not done	No hypothyroidism	2000-2500 gms	7	no	no	ye s no	no	no	no	none	not done
2 2	Indi	primigra vida	39 no	None	None	no	no	no	no 1	no no	none	11	78. 5	3.19	2.66	not done	not done	No hypothyroidism	2500-3000 gms	7	no	no	no no	no	no	no	none	not done
2 4	Bijapur	vida	38 no	None	None	no	no	ye s	no 1	no no	none	8.8	69. 7	2.22	2.53	not done	not done	No hypothyroidism	2000-2500 gms	7	ye s	no	S	no	no	no	none	not done
3 2	Indi	iua	30 no	None	None	no	ye s	no	no 1	no no	none	12. 1	96. 4	1.98	1.09	not done	not done	No hypothyroidism	1000-1500 gms	0		ye s	ye ye s s	no	no	no	none	not done
2 8	Bijapur	vida	38 no	None	None	no			no 1	no no	none	12. 6	96. 4	2.6	0.8	not done	not done	No hypothyroidism	2000-2500 gms	7	ye s	no	no no			no	none	not done
2 0	Bijapur	vida	40 no	None	None	no	no	ye s		no no	none	10. 7	74. 6	2.4	2.64	not done	not done	No hypothyroidism	2500-3000 gms	7	ye s	no	ye s no	ye s	no	no	none	not done
0	Indi	iua	40 no	None	None	no	no	no	ye s	no no	none	12. 7	92. 4	2.44	2.45	not done	not done	No hypothyroidism	2500-3000 gms	7	no :	no	no no	no	no	no	none	not done
8	Indi	ıda	40 no	None	None	no	no	ye s	no 1	no no	none	9	94		1.13	done	done	No hypothyroidism	more than 3000 gms	7	no	no	no no	no	no	no	none	not done
2 1	Tikota	vida	38 no	None	None	no	no			no no		4	90. 4	1.7	1.2	done	done	No hypothyroidism	2500-3000 gms	7	no	no	no no	no	no	no	none	not done
5	Bijapur	lua	38 no		None	no	no	no	ye s	no no	none	12. 6	86			done	done	No hypothyroidism	2000-2500 gms	7	no	no	no no	no	no	no	none	not done
2 6	Sindagi	vida	39 no	reatures	None	no	no	no	no 1	no no		11.	79. 4	2.01	1.19	not done	not done	No hypothyroidism	more than 3000 gms	7	no	no	no no	no	no	no	none	not done
2 3	Kolapur	multigrav ida	36 no	PE with severe features	None	no	no	no	no 1	no ye	none	14. 6	94. 3		2.02	not done	not done	No hypothyroidism	2500-3000 gms	7	no	no	no no	no	no	no	none	not done

2 3	Indi	multigrav 37 no	None	None	no	no y	e no	o no	no	none	10. 2	84. 6	2.13	1.1	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no	no	none	not done
3	Athani	primigra vida 39 no	None	None	no		e no	o no	no	none	9.1	87. 9	2.81	1.9	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no	no	none	not done
3 0	Indi	primigra vida 38 no	None	None	no	no y	e no	o no	ye s	none	10. 2	91. 7	1.35	2.00	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no	no	none	not done
2 4	Kalgi	multigrav de	None	None	no	no n	o no	o no	no	none	11. 4	80. 2	2.91	1.81 2	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no	no	none	not done
2 8	Sindagi	multigrav ida 38 no	None	None	no	no n	o no	o no	no	none	12. 1	82. 1	2.4	1.3	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no	no	none	not done
1 9	Bijapur	primigra vida 36 no	None	None	no	no n	o no	o no	ye s	none	11. 9	83. 2	2.49	1.56	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no n	o no	no	no	none	not done
2 3	Bijapur	multigrav ida 39 no	None	None	no	no n	o no	o no	no	none	13. 5	88		1.67	not done	not done	No hypothyroidism	2500-3000 gms	8 no	no	no n	o ye s	no	no	none	not done
2	Talikote	multigrav ida 39 no	None	None	no			o no	no	none	11. 5	79. 8	2.72	1.1	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no n	o no	no	no	none	not done
5	Bagewadi	multigrav ida 37 no	None	medically managed	no	no y	e ye s s	no	no	none	9.2	81.	3.1	1.09	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no	no	none	not done
2 0	Chadchan	primigra vida 40 no	None	None	no		e no		no	none	10.	87. 3	4.39	1.08	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no n	o no	no	no	none	not done
2 1	Tikota	multigrav do no	None	None	no	no	e ye s s	no	no	none	10.	85. 3	2	2.32	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no n	o no	no	no	none	not done
3 2	Bijapur	multigrav ida 39 no	None	None	no	no y	e ye s s	no	no	none	10. 5	77. 6	2.36	2.7	not done	not done	No hypothyroidism	2500-3000 gms		no	no n	o no	no	no	none	not done
2 4	Bagewadi	primigra vida 37 no	None	None	no	no y	e s	o no	no	Rheumatic heart disease	9.9	70. 9	5.98	2.9	not done	not done	No hypothyroidism	2000-2500 gms	$\begin{array}{ c c c c }\hline 7 & ye \\ s & \end{array}$	no	ye s n	o no	no	no	none	not done
3	Kannur	multigrav ida 37 no	None	None	no	no n	o no	o no	no	none	12. 4	91.	3.17	1.6	done	done	No hypothyroidism	2500-3000 gms		no		o no	no	no	none	done
5	Bijapur	multigrav 35 no	None	None	no	no n	o no	o no	no	none	12. 6	90. 6	2.66	1.6	not done	not done	No hypothyroidism	1500-2000 gms	7 ye s	no	ye s n	o no	no	no	none	not done
2 5 2	Muddebihal	multigrav 39 no	None	None	no	no n		o no	no	none	11. 6	79. 2 78.	1.38	1.05	not done	not done	No hypothyroidism	3000 gms	7 no	110	110 11	o no	no	no	none	not done
0	Athani	multigrav 37 no	None	None	no	no y	e no		no	none	4	9 94.	2.92	2.50	not done	not done	No hypothyroidism	2000-2500 gms 2500-3000	$\begin{array}{ c c c c c c c c c c c c c c c c c c c$	no	ye s n	o no	no	no	none	not done
2 2	Bijapur	multigrav 39 no	None	None	no		o s	no	no	none	11. 4 10.	94. 5 96.	2.29	9	not done	done	No hypothyroidism	gms 2000-2500	7 no	no	no n	o no	no	no	none	not done
3	Aliyabad	primigra vida 37 no	None	None	no	no y	e no	o no	no	none	5 11.	90.	2.03	1.33	not done	not done	No hypothyroidism	gms 2500-3000	$\begin{array}{ c c c c c c c c c c c c c c c c c c c$	no	s n	o no	no	no	none	not done
4 2	Bilagi	multigrav do no multigrav do no	None	None	no	no n	o no	o no	no	none	7 12.	5	1.82	2.6	done	not done not	No hypothyroidism	gms more than	7 no	no	no n	o no	no	no	none	not done not
5	Hipparigi	ida 40 110	None		no	no n	o no	o no	no	none	6	92 83.		2.87 6.60	not done not	done	No hypothyroidism	3000 gms more than	8 no	no	no n	o no	no	no	none	done
9	Bijapur	vida 39 110	None	None	S	no n	o no	o no	no	none	12 10.	7	2.76	4	done	done	No hypothyroidism	3000 gms 2000-2500	7 no		no n	o no	no	no	none	done
0	Bijaput	vida 59 110	None	None	no	no 3	e no	o no	no	none	8	80		2.08	done	done	No hypothyroidism	gms 2000-2500	7 S	no	s n	o no	no	no	none	done
1 2	Muddebihal	vida 40 110	None	None	no	no 3	s no	o no	no	none	8	80 88.		2.9	done	done	No hypothyroidism	gms more than	8 s	no	S	o no		no	none	done
3	Bagewadi	vida 30 110	None		no	no 3	no	o no	no	none	4 11.	4 85.	1.55	1.61	done	done	No hypothyroidism	3000 gms 2500-3000	8 no			S	no	no	none	done
0 2	Sindagi	ida 30 110	None	None	no		e	o no		none	2 10.	4 74.	3.06	7 2.78	done	done	No hypothyroidism	gms 2500-3000			no n		ve	no	none	done
0 2	Talikote	ida 33 110	None PE without severe	None	no	S	s no	o no		none	7	7 85.	3.30	8	done	done	No hypothyroidism	gms 2500-3000			no n		S	no	none	done
8	Jamkandi	ida 39 s	features	None		no n				none	1 11.	7 78.	3.73	98 2.19	done	done	No hypothyroidism	gms more than			no n			no	MAS	done
3	Indi	vida 41 no	None	None	no		o no		no	none	6 10.	7	2.25	9	done not	done not	No hypothyroidism	3000 gms 1500-2000	7 no	no e	ve				none	done
9	Muddebihal	vida 32 110	eclampsia	None	no ye	S	e ye	no e		none	8	77 74.		2.32	done not	done	No hypothyroidism	gms 2000-2500	7 s	no	S	o no		no	none	done
6 2	Bijapur	vida 39 110	None PE with severe	None	S	по	s s	no	no	none	9.4	9 89.	2.04	0.25	done	done	No hypothyroidism	gms more than		no				no	none	done
4 2	Dandeli	vida 39 110	features	None medically	no	no x	S 110	o no		none	1	6 79.	2.21	1.2	done	done	No hypothyroidism	3000 gms 2500-3000			no n				none	done
4	Arakeri	ida 39 110	None PE with severe	managed	no		8	no no			8.8	6 86.	3.21	0.8 2.39	done	done	No hypothyroidism	gms 2000-2500	Ve	·	no n				none	done
1 2	Bagewadi	vida 39 110	features	None		no n				none	2	74.	2.21	4	done	done	No hypothyroidism	gms 1500-2000	o s	по	s II		no no		MAS	done
6	Bijapur	ida 35 no	None	None	no	s	o no	no	no	Ricketsial fever	7	3	2.19	1.1	done	done	No hypothyroidism	gms	7 no	no	$\begin{bmatrix} s \\ s \end{bmatrix}$ n	o no	no	no	none	done

$\begin{bmatrix} 2 \\ 8 \end{bmatrix}$	Harinal	multigrav ida 37	7 no	None	None	no	no r	o y	e no	no	none	12. 7	83. 6		0.92	not done	not done	No hypothyroidism	2500-3000 gms	7 n	o no	no	no r	no no	no	none	not done
2 5	Bijapur	multigrav ida 40	0 no	None	None	no	no r	o y	e no	o no	none	13	89. 7	1.54	1.14 7	not done	not done	No hypothyroidism	more than 3000 gms	7 n	o no	no	no r	no no	no	none	not done
2 5	Devaripparigi	primigra vida 40	0 no	None	None	no	no 3	re s n	o no	o no	none	10. 9	76	2.91	0.64	not done	not done	No hypothyroidism	2500-3000 gms	7 n	o no	no	no r	no no	no	none	not done
3 0	Bijapur	primigra vida 36	6 no	None	None	no	ye s	o n	o no	o no	Hellp syndrome	11. 5	88. 1	1.57	1.1	not done	not done	No hypothyroidism	1500-2000 gms	7 n	o no	ye s	no r	no no	no	none	not done
2 5	Talikote	multigrav ida 40	0 no	None	None	no	no l	re s n	o no	o no	none	10. 1	78. 9	2.15	1.2	not done	not done	No hypothyroidism	2500-3000 gms	7 n	o no	no	no r	no no	no	none	not done
2 6	Yadiyapur	multigrav ida 39	9 no	None	None	ye s	no 3	re y	e no	o no	none	9.7	87	1.23	1.2	not done	not done	No hypothyroidism	2500-3000 gms	0 n	o ye	no	ye s	no no	no	none	not done
2 8	Indi	multigrav ida 36	6 no	None	None	no	ye y	re s n	o no	no	none	5.4	53. 5	2.14	1.25	not done	not done	No hypothyroidism	2000-2500 gms	7 y	e no	ye s	no r	no no	no	none	not done
2 4	Tanda	multigrav ida 38	8 no	None	None	no	no i	re y	e s no	no	none	10. 4	70. 5		2.3	not done	not done	No hypothyroidism	2500-3000 gms	8 n	o no	no	no r	no no	no	none	not done
2 0	Bijapur	vida	0 no	None	None	no	no r	o n	o no	o no	none	13. 3	86. 6		1.31 9	not done	not done	No hypothyroidism	more than 3000 gms	7 n	o no	no	no r	no no	no	none	not done
2 5	Indi	vida	7 no	None	None	no	no r	o n	o no	o no	none	11	79		2.28 6	not done	not done	No hypothyroidism	2500-3000 gms	7 n	o no	no	no r	no no	no	none	not done
2 2	Chavabai	vida	б по	None	None	no	ye y	re s	o no	o no	none	9.5	80. 8	3.49	2.21	not done	not done	No hypothyroidism	1500-2000 gms	7 y	e no	ye s	no r	no no	no	none	not done
2 4	Jamkandi	102	1 no	None	None	no	no r	o n	o no	o no	none	12. 7	90. 3	2.22	2.1	not done	not done	No hypothyroidism	more than 3000 gms	7 n	o no	no	no r	no no	no	none	not done
2 0	Bijapur	vida	0 no	None	None	no	no 3	re s	o no	ye s	none	8	69. 3	3.42	1.42 9	not done	not done	No hypothyroidism	more than 3000 gms	7 n	o no	no	no r	no no	no	none	not done
2 4	Indi	vida	8 no	None	None	no	no r	o n	o no	o no	none	12. 9	82. 5	1.9	2.3	not done	not done	No hypothyroidism	2000-2500 gms	6 y	e no	ye s	no r	no no	no	none	not done
2 8	Indi	multigrav ida 36	6 no	None	None	no	ye y		o no	o no	none	10. 1	78. 1	2.34	2.6	not done	not done	No hypothyroidism	2500-3000 gms	7 n	o no	no	110	no s	no	none	not done
2 2	Budihal	iua	8 no	None	None	no	no r	o y	e s no	o no	none	13	94. 8	1.9	1.5	not done	not done	No hypothyroidism	2500-3000 gms	8 n	o no	no	no r	no no	no	none	not done
5	Indi	ıda	8 no	None	None	no	no r	o n	o no	o no	none	11. 7	72. 6	2.93	2.04	not done	not done	No hypothyroidism	2500-3000 gms	6 n	o no	no	no r	no no	no	none	not done
2 2	Bijapur	102	7 no	PE without severe features	None	no	no r	o y	e s no	o no	none	14. 2	70. 9	1.59	1.2	not done	not done	No hypothyroidism	2000-2500 gms	7 y	e no	ye s	no r	no no	no	none	not done
2 6	Bijapur	10a	9 no	PE with severe features	None	no	no r	o y	e s	o no	none	12. 2	82	1.72	1.3	not done	not done	No hypothyroidism	2500-3000 gms	7 n	o no	no	no r	no no	no	none	not done
2 7	Bujapur	10a	5 no	None	medically managed	no	ye s	o n	o no	o no	none	9.6	92	2.1	1.9	not done	not done	No hypothyroidism	2500-3000 gms	7 n	o no	no	no r	no no	no	none	not done
2 4	Bijapur	102	8 no	None	None	no	no 3	re s	o no	o no	none	10.	71. 4	2.85	1.4	not done	not done	No hypothyroidism	2000-2500 gms	7 y	e no	ye s	no r	no no	no	none	not done
2 3	Bijapur	102	7 no	None	None	no	no 3	re s	o no	o no	Hbsag and covid positive	10. 4	76. 9	1.95	2	not done	not done	No hypothyroidism	2000-2500 gms	6 y	nc	ye s	no r	no no	no	none	not done
2 2	Bijapur	ıda	8 no	None	medically managed	no	no r	o n	o no	o no	none	12. 1	89. 3	0.93	1.7	not done	not done	No hypothyroidism	2500-3000 gms	7 n	o no	no	no r	no no	no	none	not done
2 8	Bijapur	vida	7 no	None	None	no	no r	o n	o no	o no	none	12. 3	84		1.43	not done	not done	No hypothyroidism	more than 3000 gms	6 n	o no	no	no r	no no	no	none	not done
5	Solapur	102	8 no	None	None	no	no r	o n	o no	o no	none	11. 8	84. 8	1.16	1.21	not done	not done	No hypothyroidism	2000-2500 gms	7 y	e no	ye s	no r	no no	no	none	not done
5	Devaripparigi	10a	3 no	None	None	no	ye s	o n	o no	o no	Placenta previa	11. 2	78		1.3	not done	not done	No hypothyroidism	1500-2000 gms	7 n	o no	ye s	no r	no ye	no	none	not done
8	Bijapur	10a	6 no	None	None	no	ye y	e s	o no	o no	none	9.7	72. 7	1.93	1.24	not done	not done	No hypothyroidism	1500-2000 gms	7 n	o no	ye s	no r	no no	no	none	not done
3 1	Bijapur	ıda	1 no	None	None	no	ye s	o n	o no	no	none	11	91. 9	1.91	1.23	not done	not done	No hypothyroidism	1500-2000 gms	7 n	o no	ye s	no r	no no	no	none	not done
8	Indi	vida	8 no	None	None	no	no r		o no	ye s	none	11. 9	80.	2.58	2.75	not done	not done	No hypothyroidism	2500-3000 gms	7 n	o no	no	no r	no no	no no	none	not done
2 8	Bijapur	lua	8 no	None	medically managed	no	no	re s n	o no	o no	none	7.4	71. 9	1.41	1.32	not done	not done	No hypothyroidism	2500-3000 gms	7 n	o no	no	no r	no no	no	none	not done
$\begin{bmatrix} 2 \\ 2 \end{bmatrix}$	Bijapur	ıda	5 no	None	None	no	S	o y	e s no	o no	Placenta previa	12. 5	88. 4	1.39	2.00	not done	not done	No hypothyroidism	2000-2500 gms	7 n	o no	S	no r		no no	none	not done
2 5	Bijapur	10a	0 no	PE with severe features	None	no		s n	o no	no	Hellp sundrome	7.8	79. 5	0.17	1.91	not done	not done	No hypothyroidism	1000-1500 gms	6 n	o no	S	no r	no ye	l no	none	not done
2 2	Muddebihal	vida	3 no	eclampsia	None	no	no	S		o no	none	9	92. 4	1.09	1.6	not done	not done	No hypothyroidism	2000-2500 gms	6 n	o no	S			no no	none	not done
2 1	Indi	lua	5 no	None	None	no	ye y	re s n	o no	no	AFLP	10.	78. 9	1.23	1.8	not done	not done	No hypothyroidism	2000-2500 gms	7 n	o no	ye s	no y	s no	no	none	not done
2 4	Bijapur	primigra vida 40	0 no	PE without severe features	None	no	no S	re s n	o no	no	none	10. 7	94. 3		1.7	not done	not done	No hypothyroidism	more than 3000 gms	7 n	o no	no	no r	no no	no	none	not done

3	D	multigrav 37 n					V	e			13.	78	8. 1.10	1.0	not	not		2500-3000								not
3	Bijapur	ida 37 III		None	no		0 S	no	no	none	4	5 69	5 1.18	1.3	done	done	No hypothyroidism	gms 2000-2500	7 no	no				no	none	done
8	Sindagi	primigra vida 37 no	None	None	no	no s	s no	o no	no	none	8.2	4	1.85	2	not done	not done	No hypothyroidism	gms	7 no	no no	S	ye s	no	no	none	not done
3 5	Bijapur	primigra vida 35 no	None	None	no	ye y	e no	o no	no	none	10. 9	84 5	5 3.1	2.2	not done	not done	No hypothyroidism	2000-2500 gms	7 no	no	ye s no	no	no	no	none	not done
2 9	Bijapur	multigrav ida 39 no	None None	None	no	no n	o no	o no	no	none	12. 8	79 8	3 2.33	1.4	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	no	no	no	none	not done
2 2	Bijapur	multigrav ida 40 no	None	None	no	no n	o no	o no	no	none	12. 6	83 9	3. 2.26	1.6	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	no	ye s	no	none	not done
1 9	Bijapur	primigra vida 40 no	None	None	no	no y	e no	o no	no	none	9.5	79 7	9. 1.62	2.79 7	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	no	no	no	none	not done
2 1	Bijapur	primigra vida 30 no	None	None	no	ye y	e no	o no	no	none	9.9	86		1.2	not done	not done	No hypothyroidism	1000-1500 gms	5 no	no	ye ye s	no	ye s	ye s	none	not done
2 2	Honalli	multigrav ida 34 no	None	None	no	ye s	o no	o no	no	none	13. 4	81 1	1. 5.58	2.26 5	not done	not done	No hypothyroidism	1500-2000 gms	7 no	no	ye no	ye s	no	no	none	not done
2 8	Bijapur	primigra vida 39 no	None	None	no	no n	o no	o no	no	none	11. 2	87	7 1.69	1.2	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no	no	no	none	not done
2 1	Devaripparigi	primigra vida 39 no	None	None	no	no y	e no	o no	no	none	8.7	85 4	5. 1 7.06	1.2	not done	not done	No hypothyroidism	2500-3000 gms	0 no	ye s	no s	no	no	no	none	not done
1 8	Bijapur	primigra vida 38 no	None	None	no	no n	o no	o no	no	none	12. 1	92		0.73	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	no	no	no	none	not done
2 0	Indi	multigrav ida 42 no	PE with severe features	None	no	ye s n	o no	o no	no	none	11	90 5		2.67 4	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	no	no	no	none	not done
2 4	Bijapur	multigrav ida 38 no	PE with severe features	None	no	no n	o s	no	no	none	12. 3	90 6		1.08	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no	no	no	none	not done
2 2	Bijapur	multigrav ida 40 no	None	None	no	no n	o y	no	no	none	12. 5	88 8		1.23	not done	not done	No hypothyroidism	more than 3000 gms	8 no	no	no no	no	no	no	none	not done
3 0	Bijapur	primigra vida 36 no	None	None	no	ye s	o no	o no	ye s	none	11. 2	80	0. 2.99	0.62	not done	not done	No hypothyroidism	2000-2500 gms	7 no	no	ye s no	no	no	no	none	not done
2 6	Indi	multigrav ida 41 no	None	None	no	no y	e no	o no	no	none	10. 6	8.2	.2 3.49	2.36 7	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no	no	no	RDS	not done
2 9	Chadchan	multigrav ida 39 no	None	None	no	no y	e ne	o no	no	none	10	64 3		1.1	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no	no	no	none	not done
2 8	Indi	multigrav ida 37 no	PE without severe features	None	no		e no	ye s	no	none	10. 9	83 4	3. 1.96	1.44 1	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no	no	no	none	not done
2 5	Bijapur	multigrav ida 31 no	None	None	no	ye s n	o y	no	ye s	none	11. 6	88	8. 1.79	1.22	not done	not done	No hypothyroidism	1500-2000 gms	7 no	no	ye s no	no	no	no	none	not done
2 8	Bijapur	multigrav ida 39 no	None	None	no	no n	o y	no	ye s	none	11. 2	93 7	3. 3.19	2.29 7	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no no	no no	no no	no	no	none	not done
2 2	Bijapur	primigra vida 38 no	None	None	no	no n	o no	o no	no	none	14. 1	95 6		2.03	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	no	no	no	none	not done
2 3	Sangoli	multigrav ida 41 no	None	None	no	no n	o no	o no	no	none	12. 2	89 2		2.16	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	no	no	no	RDS	not done
2 2	Sindagi	multigrav ida 38 no	eclampsia	None	no	no y	e ye	no	no	none	10. 9	71 6	1 / 1 /	1.6	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no	no	no	none	not done
2 2	Gulbarga	primigra vida 40 no	None	None	no	no n	o y	no	ye s	none	13. 9	80	0. 1.35	1.7	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	no	no	no	none	not done
2 0	Honawad	primigra vida 40 no	PE without severe features	None	no	no n	o ne	o no	no	none	11. 6	86	6. 1 1.94	2.3	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no	no	no	none	not done
2 0	Chadchan	primigra vida 40 no		None	no	no y	e ne	o no	no	none	9.2	68		2.11	not done	not done	No hypothyroidism	2000-2500 gms	7 ye	no	ye s no	no no	no	no	none	not done
3 2	Bijapur	multigrav ida 38 no	None	None	no	no n	o ne	o no	no	none	11. 9	72	2. 1.43	1.57	not done	not done	No hypothyroidism	more than 3000 gms	7 no	ye s	no no	no	no	no	HIE 2	not done
2 4	Shiraguppi	primigra vida 36 no	PE with severe features	None	no	ye s n	o ne	o no	ye s	none	12.	81		1.9	not done	not done	No hypothyroidism	2000-2500 gms	7 ye	no	ye s no	no no	no	no	none	not done
2 8	Sindagi	multigrav 38 no		medically managed	no	no y	e ne	o no	no	none	9.4	67		1.80	not done	not done	No hypothyroidism	1000-1500 gms	0 ye	ye s	ye ye	no	no	no	none	not done
3 3	Bijapur	multigrav ida 36 no	PE without severe features	None	no	ye s n	o yo	no	no	none	11.	90		1.33	not done	not done	No hypothyroidism	1500-2000 gms	7 no	o no	ve	o no	no	no	none	not done
2 5	Indi	primigra vida 40 no		None	no	no n	o no	o no	no	none	14.	94	4. 1.77	2.3	not done	not done	No hypothyroidism	2000-2500 gms	6 no	no	no no	o no	no	no	none	not done
2	Bijapur	primigra vida 38 no	None	None	no	HO 1	e ye	110	no	none	9.6	70	1 1 3 /	1.7	not done	not done	No hypothyroidism	2500-3000 gms	7 no	o no	no no	o no	no	no	none	not done
2 5	Indi	multigrav ida 32 no	None	None	no				no	Rh negative status	10. 8	62		0.28	not done	not done	No hypothyroidism	1000-1500 gms	7 no	no no	ye no	no	no	no	none	not done
2	Indi	primigra vida 39 no	None	None		no n				none	11.	79 7		2.1	not done	not done	No hypothyroidism	more than 3000 gms	7 no	o no	no no	o no	no	no	none	not done
2	Sindagi	primigra vida 40 no	PE with severe features	None	no	no n	o ne	o no	no	none	11	74	4 1.04	1.8	not done	not done	No hypothyroidism	more than 3000 gms	7 no	o no	no no	o no	no	no	none	not done
1		7100	10414103												GOIL	GOIL		2000 51115								GOILE

$\begin{bmatrix} 2 \\ 0 \end{bmatrix}$	Bijapur	primigra vida	40 no	PE with severe features	None	no	no no	no	no	no	none	12.	90. 6	2.47	2.79	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	ye	no	no	none	not done
2 3	Jamkandi	neimiara	39 no	PE with severe features	None	no	no no	no	no	no	none	12.	87. 7	2.19	2.1	not done	not done	No hypothyroidism	2000-2500 gms	7 ye	no	ye no	no	no	no	none	not done
2 2	Bijapur	multigray	40 no	None	None	no	no no	no	no	no	none	12. 6	89. 1	2.77	1.6	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	no	no	no	none	not done
2 0	Solapur	primiaro	40 no	gestational hypertension	None	no	no ye	no	ye s	no	none	10.	82. 1	3.27	2.74	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no	no	no	none	not done
3 2	Indi	multigray	37 no	None	None	no	no s	no	no	no	none	9.1	68. 6	4.63	2.4	not done	not done	No hypothyroidism	2000-2500 gms	7 ye	no	ye s no	no	no	no	none	not done
2 0	Nidoni	primigra vida	40 no	PE with severe features	None	no	no no	no	no	no	none	11. 7	85. 8	2.09	0.7	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no	no	no	none	not done
2 4	Sindagi	primigra vida	37 no	None	None	no	no no	no	no	no	none	13. 5	82. 9	1.75	2.7	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no	no	no	none	not done
2 3	Indi	multigrav ida	29 no	None	None	ye s	ye ye s s	no	no	no	none	7.3	64. 8	1.76	1.7	not done	not done	No hypothyroidism	1000-1500 gms	4 no	no	ye ye s s	no	ye s	ye s	none	not done
2 8	Tikota	multigrav ida	37 no	None	None	no	no no	ye s	no	no	none	14. 2	90. 2	1.75	2.46	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	no	no	no	none	not done
2 0	Muddebihal	multigrav ida	38 no	None	None	no	no no	no	no	no	none	11. 6	76. 9	2.87	1.4	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no	no	no	none	not done
2	Muddebihal	vida	38 no	None	None	no	no no	no	no	no	none	12. 2	85. 1	2.38	1.46	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	no	no	no	none	not done
2 3	Hadagali	10a	37 no	None	None	no	no ye	_	no	no	none	9.8	88. 7	3.01	1.7	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	no	no	no	none	not done
2 5	Jainapur	ıaa	35 no	PE with severe features	None	no	ye s no	no		ye s	none	12. 2	78. 6	1.83	2.2	not done	not done	No hypothyroidism	1000-1500 gms	7 ye s	no	ye s no	no	no	no	none	not done
2 8	Bijapur	lua	38 no	None	None	no	no no	S	ye s	no	none	11	83.	3.25	2.5	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no	no	no	none	not done
2 1 2	Nandihal	ıda	40 no	None	None	no	S	no	no	ye s	none	8.7	88. 8	2.49	2.73	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no	no	no	none	not done
1	Bomanjogi	vida	38 no	PE with severe features	None	no	no s	no	no	no	none	9.6	67.	2.72	2.1	not done	not done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no	no	no	none	not done
5	Mavinakonda	ıda	38 no	None	None	no	no s	no		ye s	none	10.	89. 8	2.1	2.4	done	done	No hypothyroidism	more than 3000 gms	7 no	no	no no	no	no	no	none	not done
0 2	Hipparagi	102	38 no	None	None	no	no no	ye s	no	no	Gestational thrombocytopenia	12.	8.1	0.74	1.8	not done	not done	No hypothyroidism	2500-3000 gms	7 no	no	no no	no	no	no	none	not done
4 2	Bijapur	primigra vida	0 no	None	None	no			no	no	none	12.	83.	3.29	2.1	done	not done	No hypothyroidism	2000-2500 gms 2000-2500	7 s	no	ye s no	no	no	no	none	not done
5	Muddebihal	1da multigrayy	36 no	None	both	no	ye ye s s	ye s	no	no	Placenta previa	6.1	69. 7 68.	3.49	7	not done	not done	No hypothyroidism	gms 2000-2500	7 no	no	s no	no	no	no	none	not done
3	Chadchan	1da multigray	33 no	None	None	no	ye s no	s	no	no	none	11	89.	1.12	3	not done not	not done not	No hypothyroidism	gms 2000-2500	7 no	no	ye no	no	no	no	none	not done not
$\begin{bmatrix} 2 \\ 0 \\ 2 \end{bmatrix}$	Ningadalli	ida	38 no	None	None	no	no no	no	no	S	none	12.	6 77.	2.08	2.1	done	done	No hypothyroidism	gms more than	$\begin{array}{ c c c c c c c c c c c c c c c c c c c$	no	s no	no	no	no	none	done
8	Bagewadi	ida	39 no	None	None	no	no no	no	no	no	none	4 11.	5 82.	2.39	2.35	done	done	No hypothyroidism	3000 gms 2500-3000	7 no	no	no no	no	no	no	none	done
3	Talikote	vida	38 no	eclampsia	None	no	no no	no	no	no	none Gestational	14.	82. 82.	2.32	1.2	done	done	No hypothyroidism	gms more than						no	none	done
0	Bijapur	vida	39 no	None	None		no no			no ye	thrombocytopenia	2	5 75.	1.05	2.9	done	done	No hypothyroidism	3000 gms 2000-2500	7 no		no no				none	done
0	Sindagi	ida	37 no	None	None	no	S Ve	S	по	S	none	8	7 89.	3.2	2.1	done	done	No hypothyroidism	gms more than	7 s	no	S IIO			no	none	done
8	Jamkandi	ida	37 no	None	None	no	S	S		no	none	5	8	1.8	2.2	done	done not	No hypothyroidism	3000 gms 2500-3000		no					none	done
3 2	Bijapur	ida	40 no 39 no	None	None	no	S	no			none	10.	75 83	3.91 2.37	0.25	done not	done not	No hypothyroidism	gms more than			no no			no	none	done not
5	Indi	ida multigrav	39 no	None	None None	no ye	S		no		none	7 12.	92.	3.06	2.86	done not	done not	No hypothyroidism	3000 gms 2000-2500	_		no no				none	done not
8 2	Bijapur Indi	multigrav	39 no	None	None	S	no no		no		none	5 10.	95.	1.97	2.4	done not	done not	No hypothyroidism	gms 2500-3000		no	no no				none	done not
8 2		nultigray	40 no	None	None		VE				none	9	8 62.			done not	done not	No hypothyroidism No hypothyroidism	gms 2500-3000			no no			no		done not
2 9	Bijapur Bijapur	ida primigra	39 no	None	None		no s		no		none	14.	3 94.	1.53		done 0.4	done 0.4	Overt	gms 2500-3000			no no				none	done not
9	Hosapet, Sangoli	primigra	38 no	PE with severe	None		no no			ye	Premature rupture of	3 11.	3 85.	3.61		0.4	13.2	hypothyroidism Overt	gms 2500-3000			no no		VA		none	13.2
2	Marakatinahalli,	vida multigrav	41 no	features None	None		no no			s no	membranes Acute gastroenteritis	11.	90	1.99		0.5	4	hypothyroidism Overt	gms more than			no no		S	no	none	13.4
8	Bagewadi	ida	71 110	TAOHE	TAOHE	Ш	110 110	, 110	ш	110	Acute gastrochterius	1	70	1.77	5.55	0.5		hypothyroidism	3000 gms	/ 110	110	110 110	110	ш	110		13.4

2 5	Vijayapura	multigrav 35 ye ida s	None	None	no	ye n	o no	no	no	none	12.	87. 7	2.42	12.1	0.4	0.8	Overt hypothyroidism	2000-2500 gms	7 no	no	no	no n	no no	no	Meningitis	1.8
1 9	Bagalkot	primigra vida 36 no	None	None	no	ye s n	o no	o no	no	none	14.	92. 6	3.1	6.17	0.45	0.9	Overt hypothyroidism	1000-1500 gms	7 ye	no	ye s	no n	no no	no	RDS	2.52
3 2	Vijayapura	multigrav 36 ye ida	PE without severe features	None	no	ye s n	o yo	no	no	none	11.	74. 8	2.6	7.82	0.6	1.2	Overt hypothyroidism	2500-3000 gms	7 nc	o no	no	no 3	ye s no	no	none	1.3
3	Vijayapura	multigrav ida 39 ye s	None	None	no	no n	o no	o no	no	PROM	12	82	2.56	4.29	0.5	1	Overt hypothyroidism	more than 3000 gms	8 no	o no	no	no 3	ye s no	no	none	3.254
2 8	Bijapur	primigra vida 35 s	imminent eclampsia	None	no	no y	e s no	no	no	none	10. 9	81. 7	2.84	2.11	0.6	21.2	Overt hypothyroidism	2000-2500 gms	7 nc	no	ye s	no n	no no	no	none	12
2 6	Bijapur	multigrav ida 31 no		None	no	ye y	e no	no	no	Rh negative status	9.3	72. 8	3.35	6.9	0.6	20.8	Overt hypothyroidism	1500-2000 gms	6 no	no	ye s	no n	no no	no	none	3.3
2 6	Indi	multigrav ida 33 no	PE without severe features	None	no	ye y	e s no	no	no	none	8.8	57. 6	3.47	3.10	0.23	0.8	Overt hypothyroidism	1000-1500 gms	7 no	no	ye s	no n	no no	no	MAS	8.6
2 5	Bijapur	multigrav ida 36 no	None	None	no	ye y	e s no	no	no	none	9.4	97. 8	2.82	6.72	0.7	0.6	Overt hypothyroidism	2500-3000 gms	7 no	no	no	no n	no no	no	none	6.8
2 9	Bijapur	multigrav do s	None	None	no	no n	o no	no	no	none	12	85	216	2.08	0.6	25.1	Overt hypothyroidism	2500-3000 gms	7 no	no	no	no 3	ye s no	no	none	7.8
5	Talikote	multigrav ida 38 ye s	PE with severe features	medically managed	no	no y	e no	o no	no	none	10	97. 7	226	4.06 7	0.6	0.8	Overt hypothyroidism	more than 3000 gms	8 no	no	no	no n	no no	no	none	7.2
2 8	Muddebihal	multigrav de ida 39 ye s	None	None	no	no n	o s	no	no	none	11. 8	87. 1	286	5.68 1	0.5	0.8	Overt hypothyroidism	2500-3000 gms	7 no	no	no	no n	no no	no	none	10.2
2 5	Bijapur	multigrav ida 36 s	None	None	no	no y	e ye	no	no	Polyhydromnios	10. 5	84. 8	175	1.71 9	0.5	140.3	Overt hypothyroidism	more than 3000 gms	7 no	no	no	no 3	ye s no	no	none	11.2
2 2	Babaleshwara	primigra vida 37 no	gestational hypertension	None	no	no y	e no	no	no	none	8.5	91. 5	147	4.1	0.8	0.3	Overt hypothyroidism	more than 3000 gms	8 no	no	no	no n	no no	no	none	9.2
2	Vijayapura	multigrav ida 37 no	PE without severe features	medically managed	no	no y	e s no	no	no	none	9.2	75. 2	93	7.96 4	0.6	0.8	Overt hypothyroidism	2500-3000 gms	8 no	no	no	no n	no no	no	none	8.2
5	Bijapur	multigrav dida 31 no	imminent eclampsia	None	ye s	ye y	e ye	no	no	none	7.7	93. 1	1.82	4.56 4	0.56	122	Overt hypothyroidism	2000-2500 gms	0 nc	ye s		ye s	no no	no	none	10.4
2 0	Bijapur	primigra vida 37 no	gestational hypertension	medically managed	no	no y	e s no	no	no	none	10	82. 2	2.88	5.17	0.3	26	Overt hypothyroidism	2500-3000 gms	7 no	no	no	no 1	ye s no	no	none	13
3 0	Bijapur	multigrav ida 38 ye s	gestational hypertension	None	no	no n	o s	no	no	none	11. 7	91. 9	2.24	3.2	0.6	14	Overt hypothyroidism	2500-3000 gms	7 no	no	no	no n	no no	no	none	12.2
1 8	Bijapur	primigra vida 39 no	None	None	no		e no	no	no	none	10. 2	94. 8	2.6	4.20	0.8	0.4	Overt hypothyroidism	2500-3000 gms	7 no	no	no	no 3	ye s no	no	none	8.2
2 4	Bijapur	multigrav do no	None	None	no	no n	o no	no	no	none	11. 8	80. 4	2.37	5.43 6	0.8	0.2	Overt hypothyroidism	2500-3000 gms	7 no	no	no	no n	no no	no	none	14.6
1 8	Indi	primigra vida 39 no	None	None	no	no n	o no	no	no	none	13. 2	86. 3	2.54	10.4 29	0.8	0.12	Overt hypothyroidism	2500-3000 gms	7 no	no	no	no 3	ye s no	no	none	10
2 4	Bijapur	multigrav de ida 41 ye s	None	None	ye s	no y	e no	no	no	Polyhydromnios	10	82. 2	2.13	5.43 6	0.24	13	Overt hypothyroidism	more than 3000 gms	5 no	no	no	ye s	no no	no	HIE	16
3 1	Indi	primigra vida 37 no	PE with severe features	None	no		e no	no	ye s	none	10. 4	84. 8	1.57	5.20 9	0.8	0.45	Overt hypothyroidism	more than 3000 gms	8 no	no	no	no n	no no	no	none	16
2 1	Bagewadi	multigrav ida 39 no	PE with severe features	None	no	no n	o no	no	no	none	11. 6	89	2.2	6.58	0.6	1.2	Overt hypothyroidism	2500-3000 gms	8 no	no	no	no n	no no	no	none	10.9
3 4	Bagewadi	multigrav ida 39 no	None	None	no	no n	o no	no	no	none	12	82. 4	2.03	5.4	0.5	16	Overt hypothyroidism	more than 3000 gms	7 no	no	no	no n	no no	no	none	8.6
2 4	Bijapur	multigrav do no ida	None	None	no	no y	e s no	no	no	none	8.3	63. 5	1.72	3.49 6	0.77	0.5	Overt hypothyroidism	2500-3000 gms	0 nc	ye s	ye s	ye s	no no	no	none	8.8
2 0	Indi	primigra vida 41 no	None	None	no	no n	o no	no	no	none	12. 5	82. 3	2.45	6.4	0.21	1.5	Overt hypothyroidism	2000-2500 gms	7 no	no	S	no n		no	none	7.6
3 2	Kalburgi	primigra vida 40 ye s	PE without severe features	None	no	no y	e s no	no	no	AFLP	9.9	87	1.53	7.2	0.5	120.5	Overt hypothyroidism	more than 3000 gms	0 nc	ye s	no	ye s	no no	no	none	9.7
3 3	Muddebihal	multigrav do s	None	None	no	no n	o s	no	no	none	11. 2	92. 7	2.91	4.56 4	0.6	143	Overt hypothyroidism	2500-3000 gms	7 no	no	no	no 3	ye s no	no	none	8.6
2 2	Muddebihal	multigrav ida 37 ye s	imminent eclampsia	None	no	no n	o no	no	no	none	11. 8	91. 8	2.83	4.6	0.8	0.34	Overt hypothyroidism	2500-3000 gms	7 no	no	no	no	ye s no	no	none	8.2
3 0	Tikota	multigrav ida 36 s	PE with severe features	None	no	ye y	e s no	no	no	none	7.8	87. 2	1.83	3.9	0.5	7.8	Overt hypothyroidism	1500-2000 gms	$\begin{array}{ c c c c c c c c c c c c c c c c c c c$	no	ye s	no n	no no	no	none	18.7
2 4	Indi	primigra vida 40 no	None	None	no	no n	o no	no	no	none	12. 1	92	3.43	3.04	0.6	0.8	Overt hypothyroidism	2500-3000 gms	7 no	no	no	no n	no no	no	none	5.6
3 0	Tikota	multigrav ida 36 s	PE with severe features	None	no	ye y	e s no	no	no	none	7.8	_	1.83	00	0.5	7.8	Overt hypothyroidism	1500-2000 gms	8 ye	no	ye s	no n	no no	no	none	9.8
2 1	Gulbarga	primigra vida 37 no	eclampsia	None	no	no y	e s no	no	no	none	10. 4	90. 8	3.35	4.86 7	0.4	92	Overt hypothyroidism	2500-3000 gms	5 no	no	no	ye s	no no	no	Birth asphyxia	8.4
2 1	Sindagi	primigra vida 40 no	gestational hypertension	None	no	no n	o no	no	no	none	11. 4	95	3.16	4.09	0.2	33.4	Overt hypothyroidism	more than 3000 gms	7 ye	no	no	no 3	ye s no	no	none	9.7
3 0	Bagewadi	primigra vida 34 no	None	None	no	no n	o no	no	no	Gestational thrombocytopenia	13. 4	88. 6	1.00	3.8	0.9	69.7	Overt hypothyroidism	1500-2000 gms	7 no	no	ye s	no n	no no	no	none	12.1

3 4	Hipparigi	multigrav 38 ye ida	PE with severe features	medically managed	no	110	e ye	e no	no	none	9.2	70. 8	1.83	3.63	0.5	10.8	Overt hypothyroidism	2500-3000 gms	0 n	o ye	no	ye s	no no	no	none	9.7
3 0	Nidagund	primigra vida 40 s	PE without severe features	None	no	v	e	o no	no	none	10. 7	93. 2	2.15	7.66	0.41	0.8	Overt hypothyroidism	2500-3000 gms	7 n	o no	no	no 1	no no	no	none	12.1
2 3	Athani	primigra vida 40 s	None	medically managed	no	no y	e no	o no	ye s	none	9.5	68	2.19	4.24	0.6	0.8	Overt hypothyroidism	2000-2500 gms	7 y	e no	ye s	no i	no no	no	none	18.1
2 0	Muddebihal	primigra vida 37 no	PE with severe features	None	no		e no	o no	ve	none	8.9	79. 9	2.69	5.82	0.1	0.6	Overt hypothyroidism	more than 3000 gms	7 n	o no	no	no 1	no no	no	none	4
2 8	Muddebihal	multigrav ida 39 no	DE with savara	None	no	no n	o no	o no	no	none	11.	80. 2	2.73	3.81	0.1	0.8	Overt hypothyroidism	more than 3000 gms	7 n	o no	no	no	no no	no	none	12.1
2 7	Kondaguli	primigra vida 37 no	DE with savons	None	no	no n	o no	o no	no	none	11	89. 1	3.1	5.00	1	0.8	Overt hypothyroidism	2000-2500 gms	7 y	no	ye s	no 1	no no	no	none	9.8
2 8	Sindagi	multigrav ida 35 no		None	no	ye s	o yo	no	no	Partial Hellp	14. 34	81. 7	1.11	11.3 59	0.6	17	Overt hypothyroidism	1500-2000 gms	7 n	o no	ye s	no 1	no no	no	none	9.3
2 2	Bagalkot	primigra ye vida 39 s	eclampsia	None	no	no n	o no	o no	no	none	12. 2	89. 8	2.52	3.37	0.3	0.9	Overt hypothyroidism	more than 3000 gms	7 n	o no	no	no 1	no no	no	none	8.9
2 9	Belgavi	primigra vida 31 ye s	None	None	no	ye s	o no	o no	no	Covid positive	13. 4	81. 7	1.43	4.3	0.12	0.8	Overt hypothyroidism	1000-1500 gms	0 n	o ye	ye s	ye s	no no	no	none	14.6
2 2	Bijapur	multigrav ida 33 ye	None	None	no	ye s	o no	o no	no	none	11. 9	83. 9	2.96	4.5	0.3	65	Overt hypothyroidism	2000-2500 gms	6 n	o no	ye s	no 1	no no	ye s	Because of cardiac arrest	11.1
3 2	Bijapur	primigra vida 33 ye s	None	None	no	ye s	o no	no	no	none	11. 6	88. 4	4.37	2.3	0.4	0.8	Overt hypothyroidism	1500-2000 gms	7 n	o no	ye s	no 1	no no	no	none	5.6
3 2	Bijapur	multigrav de dida 39 ye s	None	None	no	no n	o no	o no	no	none	12. 1	36. 5	2.72	2.36	0.3	0.8	Overt hypothyroidism	2500-3000 gms	7 n	o no	no	no l	no no	no	none	4.6
3 0	Chadchan	multigrav do no	PE with severe features	None	no	no y	e ye s s	no	no	none	9.7	97. 3	2.52	3.98 5	0.4	1.2	Overt hypothyroidism	more than 3000 gms	8 n	o no	no	no	no no	no	none	9.6
2 6	Indi	multigrav ida 37 no	None	None	no	no y	e ye	no	no	Rheumatic heart disease	8.5	68. 4	3.1	6.38 1	0.5	0.8	Overt hypothyroidism	more than 3000 gms	7 n	o no	no	no l	no no	no	none	10.9
3 2	Budagali	multigrav dida 38 ye s	None	None	no	no n	o no	o no	no	none	12. 4	83. 8	1.87	3.11 6	0.4	239.3	Overt hypothyroidism	2000-2500 gms	$7 \begin{vmatrix} y_1 \\ s \end{vmatrix}$	no	ye s	no l	no no	no	none	12.67 8
2 8	Indi	primigra vida 38 no	None	None	no	no y	e no	o no	no	none	10. 7	95. 3	2.6	3.46	0.4	2.1	Overt hypothyroidism	more than 3000 gms	7 n	o no	no	no	no no	no	none	11.26
3 2	Bijapur	multigrav ida 37 ye s	None	None	no	no n	o s	no	no	none	11. 6	89. 8	1.44	19.6 63	0.6	74.2	Overt hypothyroidism	2500-3000 gms	7 n	o no	no	no !	no no	no	none	14
2 3	Indi	multigrav ida 37 ye s	None	None	no	no n	o no	no	no	none	12. 3	86. 6	1./8	17.2 3	0.5	302	Overt hypothyroidism	2500-3000 gms	7 n	o no	no	no	no no	no	none	13
2	Bijapur	primigra vida 40 ye s	None	None	ye s	no y	e ye s s	no	no	none	6.1	117 .6	0.36	10.1 21	0.25	0.8	Overt hypothyroidism	1500-2000 gms	7 y	e no	ye s	no 1	no no	no	none	11
2 1	Indi	primigra vida 38 no	None	None	no	no y	e s	no	no	none	9.6	70. 9	1.32	5.15	0.4	0.8	Overt hypothyroidism	2500-3000 gms	7 n	o no	no	no	no no	no	none	12
2 4	Indi	multigrav ida 39 no	None	None	no	no n	o no	o no	no	none	13. 6	78. 9	1.98	4.15 1	0.4	0.8	Overt hypothyroidism	more than 3000 gms	7 n	o no	no	no 1	no no	no	none	7
$\begin{bmatrix} 2 \\ 0 \end{bmatrix}$	Nagthan	primigra vida 38 ye s	None	None	no	no n	o no	no	no	none	11. 6	84. 8	2.18	15.6	0.5	0.8	Overt hypothyroidism	2500-3000 gms	7 n	o no	no	no	no no	no	none	14
0	Bijapur	primigra vida 36 no	None	None	ye s	ye y	e s	o no	no	none	6.8	66. 1	1.38	4.41 2	0.3	0.8	Overt hypothyroidism	2000-2500 gms	7 n	o no	no	no 1	no no	no	Birth asphyxia	4.6
7	Indi	multigrav ida 39 no	PE without severe features	None	no		e no	o no	no	none	10	85. 5	2.8	4.69	0.6	1.9	Overt hypothyroidism	more than 3000 gms	7 n	o no	no	no	no no	no	none	0.37
3 0	Belagavi	multigrav da no	None	None	no	no 3	e s no	o no	no	none	7.9	85. 1	1.2	4.00	0.4	1.3	Overt hypothyroidism	more than 3000 gms	7 n	o no	no	no ·	ye s no	no	none	6.4
$\begin{bmatrix} 2 \\ 0 \end{bmatrix}$	Indi	multigrav ida 35 no	None	None	no	S	e s no	o no	no	none	10.	93. 5	210	11.4 5	0.6	1	Overt hypothyroidism	1500-2000 gms	5 y	e no	ye s	ye s	no no	ye s	none	5.4
2 2	Sangoli	multigrav ida 36 no	None	None	no	ye y	e s	o no	no	none	10.	85. 4	2.77	7.64	0.6	1.8	Overt hypothyroidism	2000-2500 gms	7 n	o no	ye s	no 1	no no	no	none	0.5
2 8	Bijapur	multigrav ida 38 no	None	None	no	no y	e no	o no	no	none	10.	86	247	5.07	0.4	92.6	Overt hypothyroidism	2500-3000 gms	7 n	o no	no	no i	no no	no	none	8.7
5	Gulbarga	multigrav ida 37 no	None	None	no	no n	o no	no	no	none	12. 6	79		1.60 4	0.5	31.6	Overt hypothyroidism	2500-3000 gms	7 n	o no	no	no I	no no	no	none	2.147
1	Athani	multigrav ida 36 no	None	None	no	no n		no	no	none	10	81. 6	2.97	3.16	0.5	0.8	Overt hypothyroidism	2500-3000 gms	7 n	o no	no	no	no no	no	none	1.367
5	Gulbarga	multigrav ida 39 no		None	no	no J	e no	no	no	none	10. 5	78. 3	3.11	3.08	0.5	0.8	Overt hypothyroidism	2500-3000 gms	7 n				no no		none	9.8
3 2	Indi	primigra vida 32 s	None	None	no	S			no	none	14	75. 7	2.6	3.44	0.29	112.4	Overt hypothyroidism	1000-1500 gms	5 n	o no	Ü	~	no no	Ü	none	3.5
9	Indi	primigra vida 38 no	None	None	no	ye s	o no	no	no	none	11.	89	2.2	8.4	0.23	0.8	Overt hypothyroidism	2500-3000 gms	7 n	o no	8	S	no no	8	none	11.2
3	Koppal	multigrav ida 37 no	None	None	no	S	o no	no	no	none	12.	79		3.3	0.45	0.8	Overt hypothyroidism	2500-3000 gms	7 n	o no	S	S	no no	S	none	21
3 2	Muddebihal	multigrav ida 37 no	None	None	no	$\begin{vmatrix} ye \\ s \end{vmatrix}$ n	o no	no	no	none	12	90. 1	1.98	6.9	0.6	0.8	Overt hypothyroidism	.3000 gms	7 n	o no	ye s	ye s	no no	ye s	none	13.8

3	CI 11	primigra	20	N.	N		ye .					13.	89.	1.77	4.5	0.42	1.1	Overt	2500-3000			ye	ye		ye		140
1	Chadchan	vida	38 no		None	no	S	10	no n	o no	none	4	6	1.77	4.5	0.43	1.1	hypothyroidism	gms	7 no	o no	S	S	no 1	no s	none	14.2
2 8	sindagi	multigrav ida	39 no	o None	None	no	S	10	no n	o no	none	14.	78. 6	2.16	3.45	0.35	0.9	Overt hypothyroidism	2500-3000 gms	7 no	o no	S	S	no 1	no ye	none	12.6
2 7	Bijapur	primigra vida	41 no	None None	None	no	ye s	10	no n	o no	none	11.	89. 2	3.4	3.1	0.33	1.2	Overt hypothyroidism	2500-3000 gms	8 no	o no	ye s	ye s	no 1	no ye s	none	9.8
2 5	Indi	multigrav ida	40 no	None None	None	no	ye s	10	no n	o no	none	12	85. 4	4.12	11.2	0.67	0.8	Overt hypothyroidism	2500-3000 gms	6 no	o no	ye s	ye s	no l	no ye s	none	12.2
2 9	Devaripparagi	primigra vida	40 no	None None	None	no	ye s	10 I	no n	o no	none	13. 21	88. 3	3.21	5.7	0.5	0.8	Overt hypothyroidism	>3000 gms	6 no	o no	ye s	ye s	no 1	no ye	none	15.6
3	Chadchan	primigra vida	38 no	None None	None	no	ye s	10 I	no n	o no	none	12. 23	79. 4	2.66	8.9	0.54	0.7	Overt hypothyroidism	2500-3000 gms	7 no	o no	ye s	ye s	no	no ye s	none	12.6
3 0	Bagalkot	primigra vida	37 no	None None	None	no	ye s	10	no n	o no	none	10. 9	88. 6	2.7	6.7	0.24	0.8	Overt hypothyroidism	2500-3000 gms	7 no	o no	ye s	ye s	no l	no ye	none	10.4
2 0	Indi	multigrav ida	39 no	None	None	no	ye s	10	no n	o no	None	11.	75. 7	2.22	3.43	0.3	0.8	Overt hypothyroidism	2500-3000 gms	7 no	o no	ye s	ye s	no	no ye s	none	13.2
3 2	Bagalkoy	multigrav ida	38 yo		None	no	no 1	10	no n	o ye s	None	12. 5	84. 1	3.01	3.24	0.93	4	Subclinical hypothyroidism	more than 3000 gms	7 no	o no	no	no	no I	no no	none	1.4
2 2	Vijayapura	primigra vida	37 no	None	None	no	no l	10	no n	o ye s	None	11	81. 5	1.56	12.8 8	1.3	2.1	Subclinical hypothyroidism	1000-1500 gms	$\left \begin{array}{c c} 6 & y_0 \\ s \end{array} \right $	e no	ye s	no	no i	no no	none	1.4
3 2	Badami tq, Bagalkot	multigrav ida	38 y	INOILE	None	no	no 1	10 I	no n	o ye s	None	12. 5	84. 1	3.01	3.24	0.93	4	Subclinical hypothyroidism	more than 3000 gms	7 no	o no	no	no	no I	no no	none	1.4
2 8	Vijayapura	multigrav ida	37 no	None None	None	no	no 1	no	ye s n	o no	None	12. 8	77. 6	2.88	3.21	0.8	1.2	Subclinical hypothyroidism	2500-3000 gms	7 no	o no	o no	no	no I	no no	none	1.8
2 0	Sindagi, vijayapura	multigrav ida	42 no	None None	None	no	no ·	ye s	ye s n	o no	None	9.6	68. 7	3.73	4.3	0.7	0.8	Subclinical hypothyroidism	2000-2500 gms	7 s	nc	ye s	no	no 1	no s	hypoglycemic convulsions	1.9
2 3	Vijayapura	primigra vida	38 no	imminent eclampsia	None	no	no 1	10	no n	o no	None	12. 6	89	1.92	4.32	1	0.8	Subclinical hypothyroidism	2000-2500 gms	0 90	,	-	ye s	no 1	no no	none	9.2
2 6	Talikote	multigrav ida	38 no	None None	None	no	no 1	10	ye s n	o no	None	11	79	2.19	3.59 5	0.9	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	o no	no	ye s	no 1	no no	RDS	1.6
2 0	Indi	primigra vida	36 no	PE without severe features	None	no	no 1	10	no n	o ye s	None	12. 6	95. 3	2.84	3.2	0.8	0.8	Subclinical hypothyroidism	1000-1500 gms	5 yo	e no	ye s	ye s	no 1	no no	none	1.6
3	Vijayapura	multigrav ida	30 no	PE with severe features	None	ye s	ye s	ye s	no n	o no	None	7.5	67. 1	2.05	3.15	0.8	7.6	Subclinical hypothyroidism	1000-1500 gms	7 no	o no	ye s	no	no 1	no ye	none	9.7
2 7	Bonnanjogi	primigra vida	40 ne	o None	None	no	no :	ye s	ye s n	o no	None	10. 5	70. 6	3.78	4.62	0.9	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	o no	o no	no	no	no no	none	7.8
2 0	Maralabhavi	multigrav ida	38 no	None None	None	no	no 1	10	no n	o no	None	12. 5	81. 8	3.4	5.6	1.2	1	Subclinical hypothyroidism	more than 3000 gms	5 no	o no	no	ye s	no i	no no	none	2.1
2 3	Tikundi	primigra vida	36 no	o None	None	no	no 3	ye s	no n	o ye	None	8.4	67. 3	4.06	4.44 9	1.5	0.8	Subclinical hypothyroidism	1500-2000 gms	7 no	o no	ye s	no	no i	no no	none	3.4
1 9	Indi	primigra vida	38 no	None None	None	no		ye s	no n	o no	Hiv positive	10. 1	70. 5	2.86	3.36	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	o no	no	no	no I	no no	none	4.2
3	Bijapur	multigrav ida	37 no	o None	None	no	no 1	10 I	no n	o no	none	13	90. 7	2.72	3.69	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	o no	o no	no	no I	no no	none	14.2
2 4	Bijapur	multigrav ida	38 no	None None	None	no	no :	ye s	ye s n	o no	Rh negative status	7.2	57. 6	3.39	3.47 6	0.8	0.8	Subclinical hypothyroidism	2000-2500 gms	7 y	nc	ye s	no	no I	no no	none	4.6
2 4	Sindagi	primigra vida	40 ne	imminent eclampsia	None	no	no 1	10	no n	o ye	None	12. 4	90. 7	2.58	3.7	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	o no	o no	no	no 1	no no	none	6.8
2 5	Bagewadi	multigrav ida	39 no	o None	None	no	no ?	ye j	ye s n	o ye	Gestational thrombocytopenia	8.6	59. 6	1.5	3.17	0.8	0.8	Subclinical hypothyroidism	2000-2500 gms	8 y	e no	ye s	no	no 1	no no	none	6.7
2 3	Bijapur	primigra vida	29 no	imminent eclampsia	None	no	no 1	10	no n	o no	None	12. 7	96. 6	2.61	5.16	0.8	0.8	Subclinical hypothyroidism	1000-1500 gms	4 no	o no	ye s	ye s	no 1	no ye	none	12.4
2 6	Muddebihal	multigrav ida	40 no	o None	None	no	no 1	10	no n	o ye	None	11.	107	351	3.4	0.9	3.3	Subclinical hypothyroidism	more than 3000 gms	7 no	o no	o no	no	no 1	no no	none	4.6
2 5	Indi	primigra vida	30 no	o None	None	no	ye s	10	no n	o ye	None	12.	92. 5	2.89	6.6	0.8	0.8	Subclinical hypothyroidism	1000-1500 gms	6 no	o no	ye s	no	no	ye s no	none	7.8
2 3	Sindagi	primigra vida	38 no	PE with severe features	None	no	no :	ye s	no n	o no	None	9.5	78. 2	1.1	3.86	0.8	0.8	Subclinical hypothyroidism	2000-2500 gms	7 no	o no	o no	no	no 1	no no	none	19
2	Indi	multigrav ida	39 no		None	ye s	ye :	ye s	no n	o no	None	10	89	2.8	4.3	0.9	0.8	Subclinical hypothyroidism	2000-2500 gms	7 no	o no	o no	no	no 1	no no	none	5.6
2 2	Indi	multigrav ida	40	e gestational hypertension	None	no	no 1	10	ye n	o no	None	13.	86	2	4.6	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	o no	o no	no	no 1	no no	none	7.8
2 9	Ronihal	multigrav ida	29 yo	e PE with severe	None	no	ye s	10	no n	o no	None	12.	77. 6		3.39	0.7	1.01	Subclinical hypothyroidism	500-1000gms	4 no	o no	ye s	ye s	no 1	no ye	none	8.8
2 5	Bijapur	man lti amarı	39 no		None	no	no 1	10	no n	o no	None	12.	86.		6.14	0.9	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	o no			no 1	no no	none	11.3
2 5	Koppal	14 !	37 no	o None	None	no	no 1	10	ye n	o no	none	12.	18. 8	3.52	3.72	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	o no	o no	no	ye s	no no	none	11.5
1 9	Indi	primigra vida	39 no	o None	None	no	no 1			o no		13.	92.	2.76	3.61	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	o no	o no	no		no no	none	4.6
7		viua										5	U		7			nypomyroidisiii	gilis								

2 5	Bijapur	multigrav ida	39	no	PE without severe features	medically managed	no no	no no no no	None	12.	88. 5	2.18	6.14	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no n	medically managed	11.4
2 5	Gangawati	multigrav ida	37	no	None	None	no no	no ye no ye	None	12.	18. 8	3.52	3.72	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no n	o none	7.8
2	Bijapur	multigrav ida	39	no	None	None	no no	ye ye no no	None	8.8	76. 8	3.27	3.19	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no n	o none	9.8
3	Bijapur	multigrav ida	38	no	None	none	no no	no ye no no	None	12.	81.	2.32	3.2	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no n	none none	8.3
2	Bijapur	primigra vida	40	no	None	None	no no	ye no no no	None	9.5	74. 8	2.75	5.4	0.9	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no n	o none	4.6
2 2	Athani	primigra vida	38	no	eclampsia	None	no no	ye no no no	None	10.	83.	2.87	3.2	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	8 no	no	no n	o no	no n	o none	7.3
2 4	Indi	multigrav ida	39	no	None	None	no no	no no no no	None	12.	90	2.46	6.58	0.8	0.8	Subclinical hypothyroidism	more than 3000 gms	7 no	no	no n	o no	no n	o none	8.3
2 4	Bagalkot	multigrav ida	29	no	PE with severe features	None	no no	ye no no no	Hypovolemic shock	3.6	69. 9	1.3	13.8 41	0.7	0.8	Subclinical hypothyroidism	1000-1500 gms	4 no	ye s		e no	no n	none none	14.6
2 6	Bijapur	multigrav ida	29	no	eclampsia	None	no no	no no no no	none	12. 5	83. 6	1.4	3.53 7	0.8	0.8	Subclinical hypothyroidism	500-1000gms	4 no	no	ye y	e no	ye y	none	12.7
3 4	Indi	multigrav ida	40	no	None	None	no no	ye no no no	None	8.7	73. 6	2.92	4.66 8	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no n	o none	9.8
2 5	Bijapur	multigrav ida	40	ye s	None	both	no no	no ye no no	None	11	86. 4	32.6	3.2	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no n	both	8.6
3 4	Bijapur	multigrav ida	37	ye s	None	None	no no	no ye no no	None	12	89. 8	2.34	4.6	0.9	122	Subclinical hypothyroidism	2000-2500 gms	7 ye s	ye s	ye s	o no	no n	none none	18.4
2 3	Bilagi	primigra vida	40	no	None	None	no no	no no no no	None	12. 4	100 .4	2.69	3.04	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	6 no	no	no n	o ye	no n	none none	8.9
1 9	Bijapur	primigra vida	38	no	None	None	no no	ye no no ye	None	10.	64. 9	1.75	4.34	0.6	0.8	Subclinical hypothyroidism	1500-2000 gms	$0 \begin{vmatrix} ye \\ s \end{vmatrix}$	•		e no	no n	none none	13.6
2 5	pagewadi	multigrav ida	35	ye s	None	None	no ye	110 110 110	None	11. 9	93. 5	2.5	4.34	0.5	0.8	Subclinical hypothyroidism	2000-2500 gms	7 no	no	ye s	o no	no n	none none	8.9
2 5	Bijapur	multigrav ida	34	no	PE with severe features	None	no ye	no no no ye	None	12. 9	92. 3	2.49	5.66 9	0.9	0.8	Subclinical hypothyroidism	1500-2000 gms	7 no	no	ye s	o no	no n	none none	13.6
2 4	Bijapur	multigrav ida	34	ye s	PE with severe features	None	no s	110 110	None	8.9	79. 3	1.43	8.3	1.6	147	Subclinical hypothyroidism	2000-2500 gms	7 no	no	ye s	o no	no n	o none	18.7
2 4	Bijapur	multigrav ida	35	no	None	None	no no	ye no no no	None	9.8	71. 1	3.5	4.34	0.8	0.9	Subclinical hypothyroidism	more than 3000 gms	7 no	no	no n	o no	no n	none	9.6
2 2	Bijapur	primigra vida	35	no	PE with severe features	None	no se	no no no no	None	11. 1	85. 8	2.72	8.65 6	0.9	0.8	Subclinical hypothyroidism	2000-2500 gms	7 no	no	ye s	o no	no n	o Pneumonia	9.7
2 4	Bijapur	multigrav ida	36	no	PE with severe features	None	no ye		Dengue	8.7	87. 3	0.15	6.5	2.5	94.8	Subclinical hypothyroidism	2000-2500 gms	7 no	no	ye s	o s	no n	none	9.6
2 5	Bijapur	primigra vida	34	no	eclampsia	None	no ye	no no no no	Rh negative	12. 2	82	1.91	7.27 7	1.46	14.4	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no n	none	16.3
$\begin{bmatrix} 2 \\ 0 \end{bmatrix}$ M	Iuddebihal	primigra vida	33	no	PE with severe features	None	no ye	no no no ye	None	11. 7	78. 6	2.87	7.5	0.8	0.8	Subclinical hypothyroidism	1500-2000 gms	6 no	no	ye s	o no	no n	none	21
2 2	Sangli	multigrav ida	36	no	None	None	no ye		Polyhydromnios	10. 8	84. 8	2.74	3.28	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no n	none	11.2
2 6	Bijapur	multigrav ida	38	no	None	None	no no	ye ye no no	None	7.4	78. 9	0.85	5.6	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no n	none none	6.5
3	Bijapur	primigra vida	37	no	chronic hypertension	None	no no	no no ye s	None	11. 1	92. 3	1.78	3.4	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no n	none none	7.9
2 5	Jamkandi	primigra vida	40	no	PE with severe features	None	no no	no no no ye	None	12. 7	90. 4	1.64	4.01 8	0.8	0.8	Subclinical hypothyroidism	2000-2500 gms	6 no	no	ye s	o no	no n	RDS	9.8
O	Bijapur	primigra vida	39	no	None	None	no no	ye no no no	None	8.8	62. 3	3.94	4.33	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no n	none none	7.6
2 6	Sindagi	multigrav ida	38	no	None	None	no no	ye no no no	None	7.5	64. 1	2.81	3.84	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no n	none none	7.3
3	Bijapur	multigrav ida	40	no	None	None	no no	ye no no no	None	9.8	77. 3	1.95	3.22 9	0.8	0.8	Subclinical hypothyroidism	more than 3000 gms	7 no	no	no n	o no	no n	o none	7.6
$\begin{bmatrix} 2 \\ 5 \end{bmatrix}$ M	Iuddebihal	multigrav ida	38	no	PE with severe features	None	ye s no	ye no no no	None	5.2	104	1.23	20.4 18	0.9	0.8	Subclinical hypothyroidism	1500-2000 gms	6 ge	no	ye s	o no	no n	o MAS	9.23
8	Gulbarga	multigrav ida	32	no	eclampsia	None	no no	no ye no no	None	16. 6	94. 6	0.99	4.3	1.8	0.8	Subclinical hypothyroidism	1000-1500 gms	5 ye s	no	ye y	e no	ye y	none	8.7
2	Bagewadi	multigrav ida	40	no	None	None	no no	no no no no	None	11.		2.32	5.61 4	0.8	0.8	Subclinical hypothyroidism	more than 3000 gms	8 no	no	no n	o ye	IIO II	none	9.9
9	varipparigi	multigrav ida	31	ye s	None	None	no s	no no no no		12. 3	91. 4	2.2	4.3	1.3	0.8	Subclinical hypothyroidism	1000-1500 gms	6 no	no	ye s	o no	no n	none none	7.1
2 2	Bijapur	multigrav ida	33	no		medically managed	no ye		Gestational thrombocytopenia	5.5	112 .9	0.41	6.51 9	1.3	0.8	Subclinical hypothyroidism	1500-2000 gms	7 no	no	ye s	o no	no n	medically managed	10.2
$\begin{bmatrix} 2 \\ 1 \end{bmatrix}$	Jamkandi	primigra vida	38	no	PE with severe features	None	no no	no no no no	Hellp syndrome	11.	92. 1	0.62	3.2	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no n	o no	no n	none	4.2

$\begin{bmatrix} 2 \\ 2 \end{bmatrix}$	Jamkandi	multigrav de	None	None	no	no r	o no	o no	no	none	13. 5	82. 9	2.68	3.20	1.2	112	Subclinical hypothyroidism	more than 3000 gms	7 no	no	no	no n	o no	no	none	4.3
2 1	Bijapur	multigrav ida 39 no	None	None	no	no r	o no	o no	no	None	12. 1	87. 4	2.66	6.29	0.8	0.8	Subclinical hypothyroidism	more than 3000 gms	7 no	no	no	no n	o no	no	none	9.6
2 8	Indi	multigrav ida 38 no	PE without severe features	None	no	no r	o y	e no	no	None	13. 8	90	2.78	3.15	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no	no n	o no	no	none	8.7
2 2	Indi	multigrav ida 40 no	None	None	no	no r	o no	o no	no	None	12. 7	84. 3	2.41	4.89	1.2	0.6	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no	no n	o no	no	none	11.2
2 4	Bijapur	multigrav ida 41 no	None	None	no	no 2	e ye	e no	no	Gestational thrombocytopenia	10. 7	80. 4	0.44	5.09	0.9	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no	no n	o no	no	none	8.12
2 4	Bijapur	multigrav ida 37 no	None	None	no	no r	o no	o no	no	none	12. 6	84. 3	1.72	3.6	0.9	0.8	Subclinical hypothyroidism	2000-2500 gms	7 ye s	no	ye s	no n	o no	no	none	8.3
1 9	Devaripparigi	primigra vida 39 no	eclampsia	medically managed	no	no 3	e no	o no	no	None	9.3	90. 2	1.21	3.38	1.4	2.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no	no n	o no	no	Cleft lip	9.7
2 5	Athani	multigrav ida 31 no	PE with severe features	None	ye s	ye y	e no	o no	no	None	9.2	79. 1	4.98	3.74	1.2	0.8	Subclinical hypothyroidism	1000-1500 gms	0 no	ye s	ye s	ye s	o no	no	none	11.4
2 5	Indi	multigrav ida 39 ye s	PE without severe features	None	no	no r	o $\frac{y_0}{s}$	e no	no	None	14. 9	78. 1	0.96	4.01	0.7	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no	no 3	re s no	no	none	10.8
2 0	Bijapur	primigra vida 40 no	None	None	no	no r	o no	o no	no	None	12. 3	91. 1	3.05	7.44 6	0.9	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no	no n	o no	no	none	8.6
6	Indi	multigrav ida 39 ye s	PE without severe features	None	no	no r	o y	e no	no	None	12. 7	82	2.09	4.3	1	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no	no n	o no	no	none	9.9
2	Kalburgi	primigra vida 38 s	None	None	no	no r	o no	o no	no	None	12. 1	92. 1	3.2	5.02	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no	no n	o no	no	none	4.2
2 9	Muddebihal	primigra vida 36 no	None	medically managed	no	ye y	e no	o no	no	None	10. 4	80	1.48	10.3 93	1.2	0.8	Subclinical hypothyroidism	2000-2500 gms	$\begin{array}{ c c c c c c c c c c c c c c c c c c c$	no	ye s	no n	o no	no	medically managed	9.6
2 9	Bijapur	multigrav ida 30 ye s	None	None	no	ye s	o ne	o no	no	None	11. 8	93. 6	1.37	3.72	0.8	0.8	Subclinical hypothyroidism	1000-1500 gms	6 no	no	ye s	no n	o no	no	none	14.3
2 0	Bijapur	multigrav ida 39 no	None	None	no	no r	o no	o no	no	None	12. 2	87. 1	1.51	4.68	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms			no	no 3	re s no		none	5.6
2 0	Inchageri	primigra vida 31 no	eclampsia	None	no	S	o ne	o no	no	None	12.	74. 6	2.94	4.10	0.9	141.6	Subclinical hypothyroidism		3 no	no	ye s	ye s	o ye	ye s	none	10.2
2 3	Hebbal	multigrav ida 36 no	None	None	no	s	e no	o no	no	None	10. 9	88. 7	2.18	4.32	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no	no n	o no	no	none	9.9
0	Talikote	primigra vida 36 no	eclampsia	None	no	S	o ne	o no	no	None	12. 9	85. 1	3.72	5.92 7	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	6 no	no	no	no n	o no	no	none	11.2
2 1	Bijapur	primigra vida 32 no	None	None	no	S	o no	o no	no	None	12. 2	80. 5	1.83	3.2	0.8	0.8	Subclinical hypothyroidism	1500-2000 gms	7 no	no	ye s	no n	o no	no	none	7.2
3 4	Bijapur	multigrav ida 35 ye s	None	None	no	ye y	e no	o no	no	None	10. 6	78. 9	3.04	3.45	2.1	0.9	Subclinical hypothyroidism	2000-2500 gms	7 no	no	S	no n	o no	no	none	5.7
3 1	Bijapur	multigrav ida 35 ye s	None	None	no	no r	o no	o no	no	None	11.	98. 5	1.53	4.2	2.3	91	Subclinical hypothyroidism	2000-2500 gms	7 no	no	ye s	no n		no	none	9.4
7	Bijapur	primigra vida 38 s	None	None	no	no S	e s	o no	no	None	10. 6	80.	5.05	2.40	1.2	0.9	Subclinical hypothyroidism	2500-3000 gms	7 no	no	no	no 3	no no	no	none	8.8
2 2	Bijapur	primigra vida 41 no	None	None	no	no r	o no	o no	no	None	12. 5	91.	2.71	4.42	0.8	0.8	Subclinical hypothyroidism	3000 gms		no	no	no n	o no	no	none	7.2
5	Indi	multigrav 39 ye s	None	None	no	no r	o no	o no	no	None	14. 2	81.	3.05	3.02	0.8	0.8	Subclinical hypothyroidism	2000-2500 gms	7 ye s	no	ye s	no n	o no	no	none	13.6
3 0	Bijapur	multigrav da 36 ye s	None	None	no	S	o s	no	no	Ерперѕу	11. 5	90.	2.17	2.58 5 4.98	0.8	0.8	Subclinical hypothyroidism Subclinical	2500-3000 gms	7 no	no	no	no n	o no	no	none	9.7
2 2 2	Bijapur	multigrav ida 33 no	None PE with severe	None	no	S		o no	s	none	11. 5	87. 6 86.	3.13	4.98 4 3.32	0.8	0.8	hypothyroidism Subclinical	1500-2000 gms 1500-2000	7 no	no	S	no n	o no	no	none	8.6
6 2	Indi	primigra vida 33 no	features	None	ye s	s	e no	o no	no	none	10. 6 12.	3 89.	0.32	9 4.12	0.8	0.8	hypothyroidism Subclinical	gms 2000-2500	7 no	no	ye s ve	no n	o no	no	none	8.6
5 2	Afzalpur	multigrav do no multigrav 37 no		None	no	no r	o s	no	no no	None	3 11.	7 80.	2.45	4.12 9 4.04	0.8	0.8	hypothyroidism Subclinical	gms 2000-2500	6 s	no	S	no n	o no	no	none	14
8	Bijapur	ida 3/ 110		None	no	no r	o no	o no	s ye	None	7 12.	7 91.	2.92	1 90.9	0.8	1.3	hypothyroidism Subclinical	gms more than	7 s	no	s	no n	o no	no	none	13.4
4	Huvinhipparigi	ida 39 s	None	None	no	no r	o ne	o no	no no	None	12. 4 12.	3 81.	2.59	1 3.04	0.8	3.11	hypothyroidism Subclinical	3000 gms 2500-3000	7 no	no	no	no 3	no no	no	none	15.53
2	Bagewadi	vida 37 110	-			no r	V	e		None	9	5	3.66	3	1	0.8	hypothyroidism Subclinical	gms more than			no			no	none	10
6 2	Devaripparigi	ida 38 s	None PE without severe	None		no r	S	3		None	1 1 13.	87 90.	2.41	9.59	1	0.8	hypothyroidism Subclinical	3000 gms 2500-3000			no				RDS	13
8	Indi	ida 38 110	features	None		no r	S		no	None	8	6 85.	2.78	3.1 4.51	1.2	0.8	hypothyroidism Subclinical	gms 2500-3000			no				none	9
2 5 2	Sindagi	vida 39 110		None		no r				None	3 11.	7 82.	2.92	8 5.75	1.6	0.8	hypothyroidism Subclinical	gms 1500-2000	7 no		no ve				none	13.7
$\begin{bmatrix} 2 \\ 0 \end{bmatrix}$	Muddebihal	vida 38 no	eclampsia	None	no	no r	o no	ono	no	None	9	8	1.44	5	0.8	0.8	hypothyroidism	gms	$\begin{array}{ c c c c c c c c c c c c c c c c c c c$	no	ye s	no n	o no	no	RDS	7.9

$\begin{bmatrix} 2 \\ 0 \end{bmatrix}$	Indi	primigra vida	37 no	None	None	no	no S	re n	o n	o no	None	9.8	65. 3	3.32	4.73 8	0.8	0.8	Subclinical hypothyroidism	1500-2000 gms	7	ye n	no :	ye no	ye s	no	no	none	16.1
2 1	Indi	multigrav ida	42 no	PE without severe features	None	no	no n	o y	e s n	o no	None	12. 2	85. 1	1.95	4.9	0.8	0.8	Subclinical hypothyroidism	2500-3000 gms	7	no n	10	no no	no	no	no	none	6.5
2 2	Bijapur	multigrav ida	38 no	PE with severe features	none	no	no n	o n	o n	o no	None	11. 1	92	1.97	4.2	0.8	0.2	Subclinical hypothyroidism	2500-3000 gms	7	no n	no l	no no	no	no	no	none	12.2
2 2	Bagewadi	primigra vida	37 no	none	none	no	no n	o n	o n	o no	None	10. 9	89. 2	1.56	3.6	0.8	0.2	Subclinical hypothyroidism	2500-3000 gms	7	no n	10	no no	no	no	no	none	12.2
2 4	Jamkandi	multigrav ida	39 no	none	none	no	no n	o n	o n	o no	None	11. 2	90	2.31	3.1	0.8	0.2	Subclinical hypothyroidism	2500-3000 gms	7	no n	no l	no no	no	no	no	none	12.2
2 1	Indi	multigrav ida	38 no	none	none	no	no n	o n	o n	o no	None	11. 3	88. 7	1.88	3.6	0.8	0.2	Subclinical hypothyroidism	2500-3000 gms	7	no n	no l	no no	no	no	no	none	12.2
3 4	Bijapur	primigra vida	40 no	none	none	no	no n	o n	o n	o no	None	11. 1	89. 2	2.8	4.5	0.8	0.2	Subclinical hypothyroidism	2500-3000 gms	7	no n	no l	no no	no	no	no	none	12.2
3 0	Devaripparagi	primigra vida	39 ye s	none	none	no	no n	o n	o n	o no	None	11	89. 6	2.99	5.8	0.8	0.2	Subclinical hypothyroidism	2500-3000 gms	7	no n	no l	no no	no	no	no	none	12.2
2 2	Bagewadi	multigrav ida	39 no	none	none	no	no n	o n	o n	o no	None	12. 4	90. 5	3.22	3.23	0.8	0.2	Subclinical hypothyroidism	2500-3000 gms	7	no n	no l	no no	no	no	no	none	12.2
2 1	Bijapur	multigrav ida	41 ye	gestational hypertension	none	no	no n	o n	o n	o no	None	12. 6	91	3.45	3.8	0.8	0.2	Subclinical hypothyroidism	2500-3000 gms	7	no n	10	no no	no	no	no	none	12.2
2 3	Huvinhipparigi	multigrav ida	40 no	none	none	no	no n	o n	o n	o no	None	12. 2	88. 6	2.36	3.98	0.8	0.2	Subclinical hypothyroidism	2500-3000 gms	7	no n	10	no no	no	no	no	none	12.2
2 5	Bijapur	primigra vida	$\begin{array}{ c c c c }\hline 37 & ye \\ s & \end{array}$	none	none	no	no 3	re s n	o n	o no	None	10. 2	88	1.75	4.1	0.8	0.2	Subclinical hypothyroidism	2500-3000 gms	7	no n	10	no no	no	no	no	none	12.2
2 9	Sindagi	primigra vida	37 no	none	none	no		re s n	o n	o no	None	10. 8	92	1.89	5.1	0.8	0.2	Subclinical hypothyroidism	2500-3000 gms	7	no n	10	no no	no	no	no	none	12.2
2 5	Indi	multigrav ida	39 no	None	None	no	no 3	re s	o n	o no	none	10. 7	83	2.37	0.25	not done	not done	No hypothyroidism	more than 3000 gms	8	no n	10	no no	no	no	no	none	not done

Thesis ORIGINALITY REPORT INTERNET SOURCES STUDENT PAPERS SIMILARITY INDEX **PUBLICATIONS** PRIMARY SOURCES "Handbook of Growth and Growth Monitoring in Health and Disease", Springer Nature, 2012 Publication www.liebertpub.com Internet Source Aruna M Biradar, Rajasri G Yaliwal, Shreedevi 3 S Kori, Sangamesh S Mathapati, Shobha S Shiragur, Subhashchandra R Mudanur. "Randomised Control Trial of 3 IU Intravenous Oxytocin Bolus With 7 IU Oxytocin Infusion Versus 10 IU Intramuscular Oxytocin in the Third Stage of Labour in the Prevention of Postpartum Hemorrhage", International Journal of Women's Health and Reproduction Sciences, 2020 Publication repository-tnmgrmu.ac.in Internet Source