OUTCOME OF PREGNANCY AND PERINATAL OUTCOME IN AMNIOTIC FLUID INDEX LESS THAN OR EQUAL TO 5 IN TERM LOW RISK PREGNANCY

Dissertation submitted in partial fulfillment of

M.S. DEGREE EXAMINATION

M.S.OBSTETRICS AND GYNAECOLOGY- BRANCH II

CHENGALPATTU MEDICAL COLLEGE,CHENGALPATTU

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

APRIL 2017

BONAFIDE CERTIFICATE

This is to certify that the dissertation titled**" OUTCOME OF PREGNANCY AND PERINATAL OUTCOME INAMNIOTIC FLUID INDEX LESS THAN OR EQUAL TO 5 IN TERM LOW RISK PREGNANCY"** is a bonafide work done by **DR. SURYA**in the CHENGALPATTU MEDICAL COLLEGE, during the academic year 2014-2017 submitted to the Tamilnadu Dr. M.G.R. Medical University in partial fulfillment of University regulation for M.S. Branch – II Obstetrics and Gynaecology degree examination of The TamilnaduDr.M.G.R Medical University to be held in April 2017

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DECLARATION BY THE CANDIDATE

I, Dr. S. SURYA solemnly declare that the dissertation titled**"OUTCOME OF PREGNANCY AND PERINATAL OUTCOME IN AMNIOTIC FLUID INDEX LESS THAN OR EQUAL TO 5 IN TERM LOW RISK PREGNANCY"** has been prepared by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award , degree, diploma to any other University board either in India or abroad.

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Designation	:	2nd Year MS O&G
Co-Investigators	:	1.Dr.S.Sampathkumari,MD(O&G) Asso.Prof.of O&G
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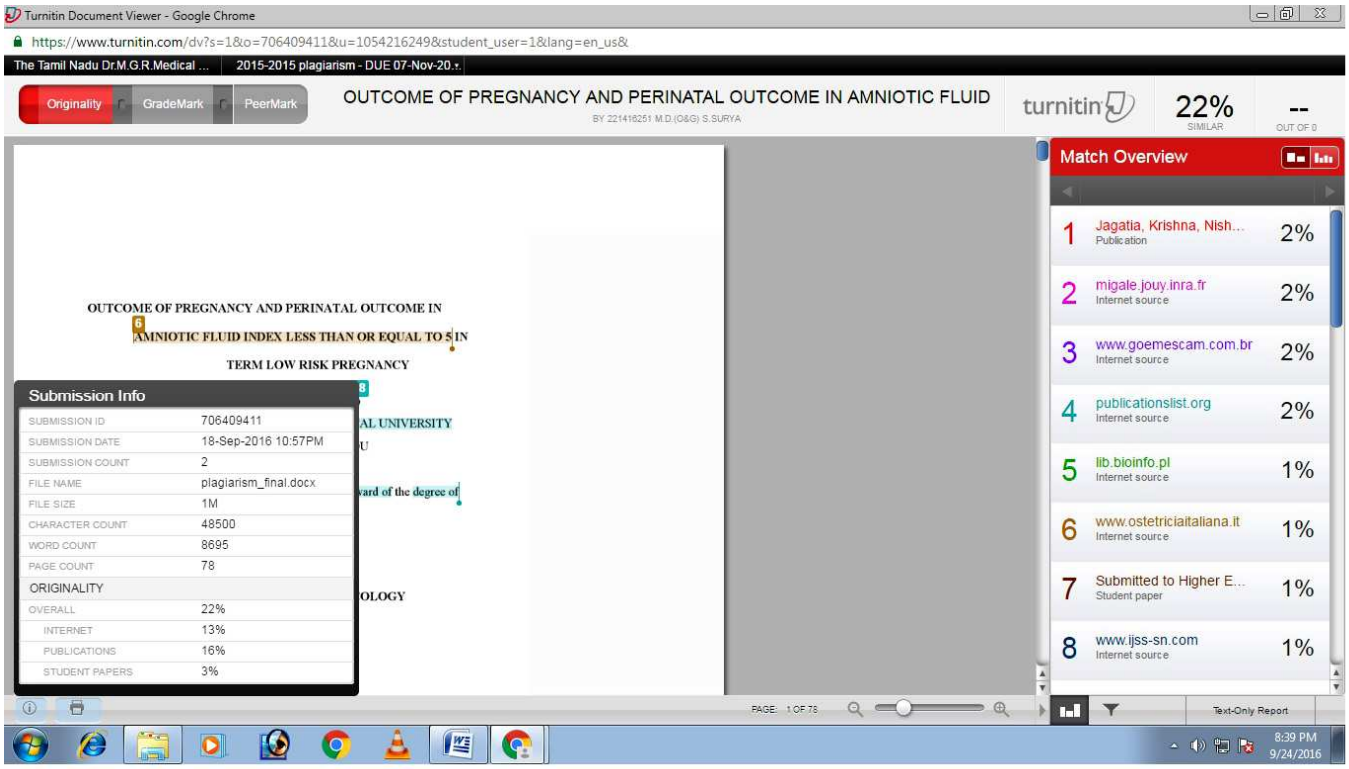
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I humbly submit this work to the Almighty who has given the health and ability to pass through all the difficulties in the compilation and proclamation of my dissertation.

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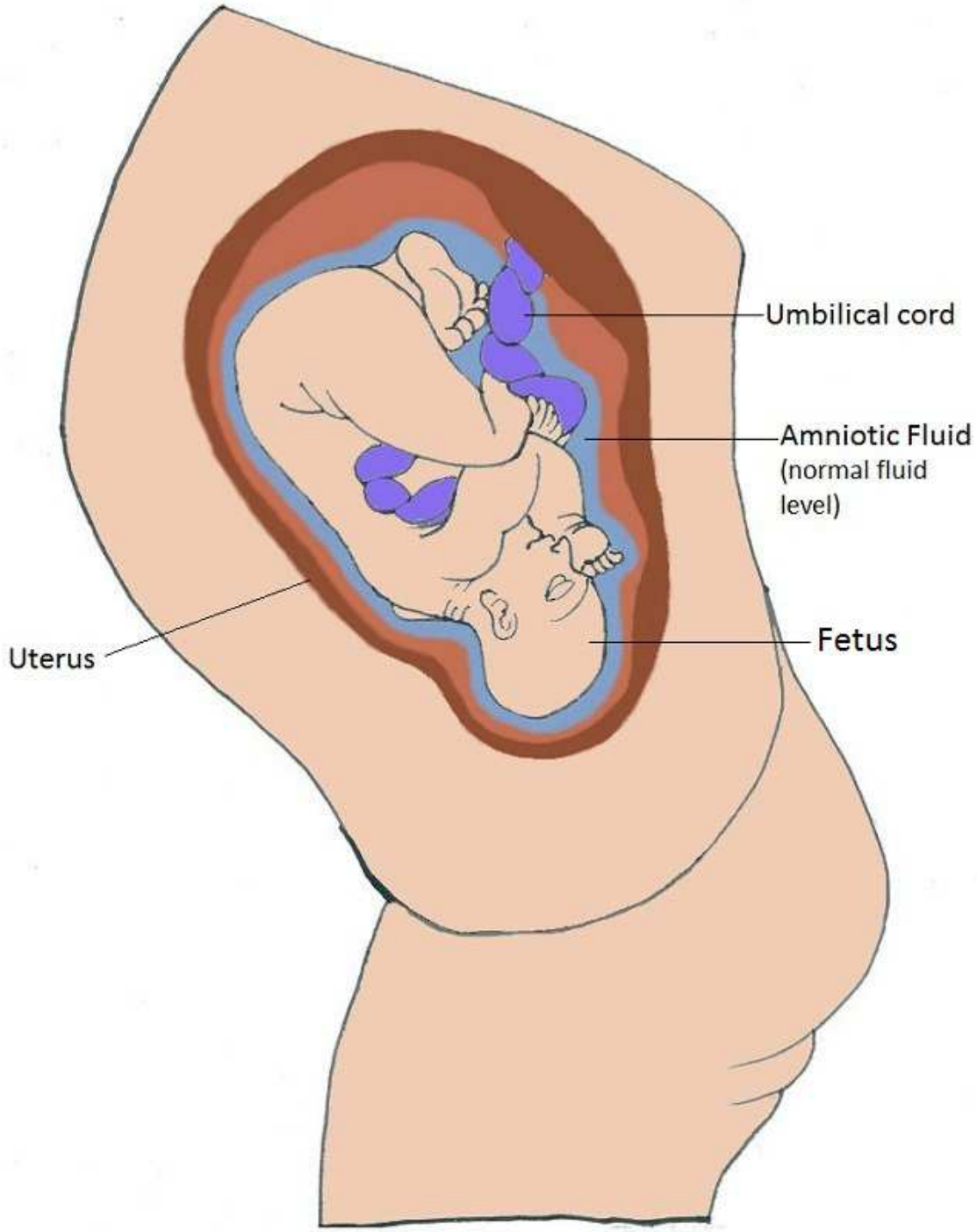
AMNIOTIC FLUID

Amniotic fluid is a clear, mildly yellowish coloured fluid contained in amniotic sac which is in circulation around the fetus. It has ennumerous functions which are important for the fetus and its development inutero[2].

- It gives a physical space for fetus to develop which is necessary for normal musculoskeletal development.
- It acts as shock absorber thereby protects the fetus from external and internal injury, and umbilical cord compression.
- It helps to prevent infection in the amniotic environment by its bacteriostatic property.
- It allows and helps in fetal swallowing – essential for the gastrointestinal tract development, and also supports in fetal breathing necessary for lung development.
- It maintains amniotic fluid pressure there by reducing the loss of Lung fluid which is an essential component for pulmonary development (Nicolini,1989)[1]

Amniotic Fluid



AMNIOTIC FLUID PRODUCTION

Before the embryo becomes evident, an amnionic space is recognized just before the time of implantation.

In the first and mid trimester, amniotic fluid is formed from both maternal and fetal and compartments. Substances freely cross the fetal skin and diffuses through the amniontrans membranous flow, across the fetal vessels on the placental surface as well-intramembranous flow.

Thus amniotic fluid present in early gestation is just a dialysate which is identical to maternal and fetal plasma, but its proteinconcentration is comparatively low with the maternal plasma.

Fetal urine production starts between 8 to 11 weeks but it is only by 20^{th} week that it become the major component. Water transport across the fetal skin continues until about 22 to 25 weeks, till fetal skin keratinization takes place, because it makes impermeable to further most diffusion.(1) At this point of time, the fetus solely contributes to the amniotic fluid volume and also its composition exclusively through urine. Urine can be observed in fetal bladder at 11 weeks transabdominally , between 3 to 9 weeks by transvaginally. Since fetal urine is hypotonic amounting to 80-140 mOsm/liter , it leads to hypotonic fluid 250-260 mOsm/liter at term containing increasing concentrations of uric acid, urea and creatinine.

Around term a fetus has the capacity to produce around 500 to 700 ml /day, but it begins to decline in urine production hourly after 40 weeks of gestation[15].

AMNIOTIC FLUID REGULATION

Amniotic fluid is regulated primarily by fetal swallowing and this has been observed as early in 16 weeks. The fluid gets absorbed through fetal gastrointestinal system and it either gets transferred to the maternal circulation or gets recycled back through the kidney.

Secondary factor that aids in removal of amniotic fluid is through the respiratory tract. As early as 11 weeks of gestation, fetal respiratory activity can be observed. As amniotic fluid is more hypotonic compared to fetal plasma,when amniotic fluid is expressed to the fetal alveolar capillary bed, it results in movement of water from amniotic cavity to the fetus.

It is also postulated that amniotic fluid is also removed by continuous flow via oncotic and hydrostatic forces. At the chorionic plate, fluid exchange takes place and leads to reabsorption of water upto 80 ml/day by fetus.

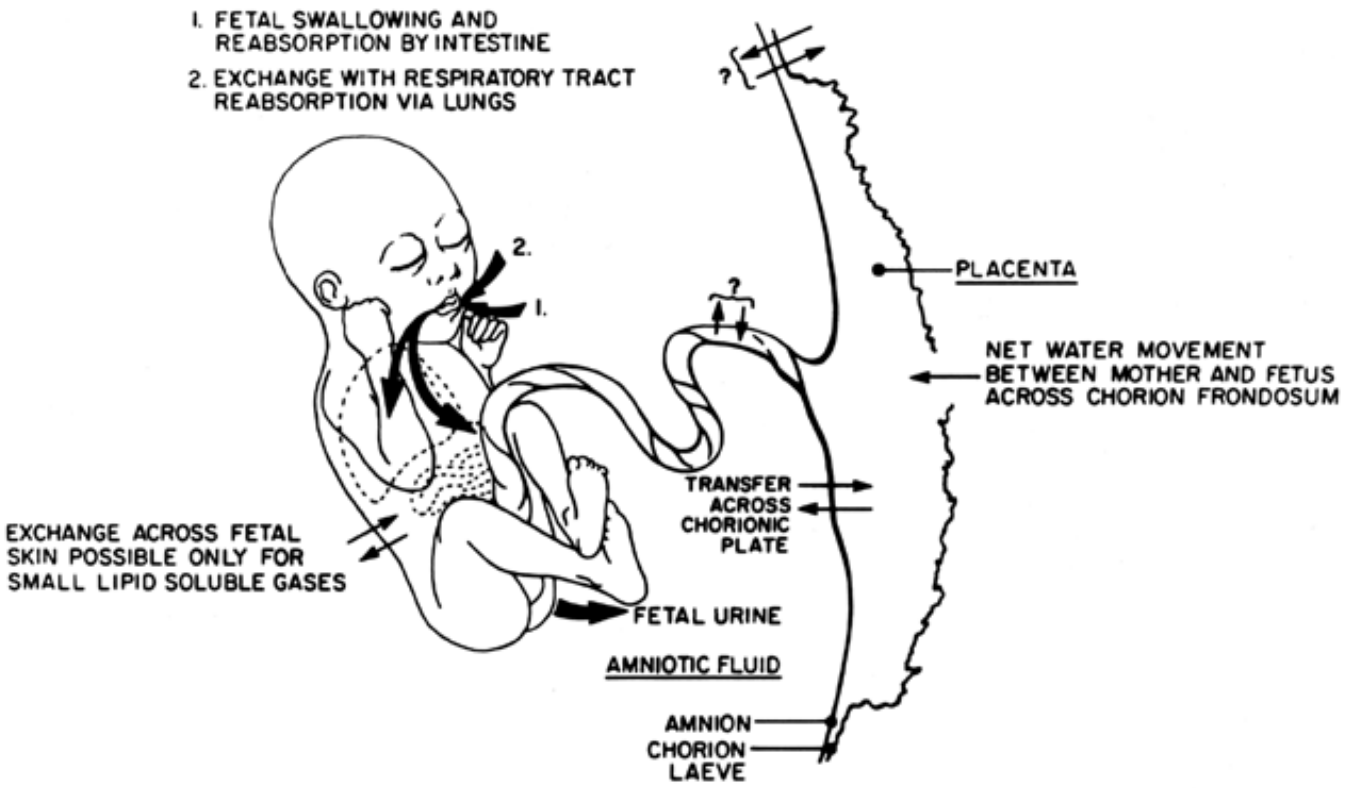


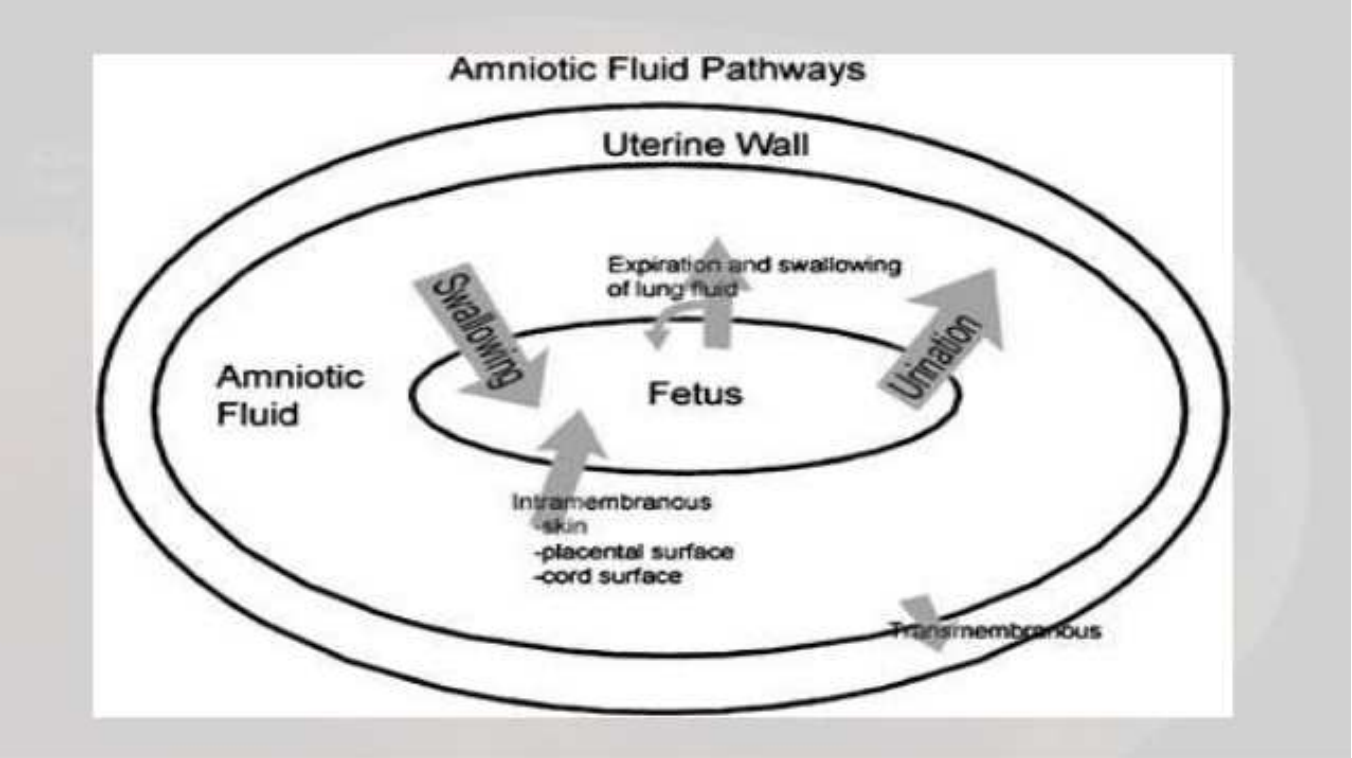
FIG 2: AMNIOTIC FLUID PRODUCTION AND REGULATION

Last but not the least, placenta itself plays a major role in amniotic fluid regulation The fetus capillary interface possessing a large surface area has the ability to magnify smaller osmolar gradient between the fetus and the mother, thereby leading to net water transfer in larger volumes.This level of water exchange maximally influences intravascular volume of the fetus and effectively affects renal blood flow and production of urine.

AMNIOTIC FLUID VOLUME REGULATION IN LATE PREGNANCY		
PATHWAY	**EFFECT ON VOLUME**	**APPROXIMATE DAILY VOLUME(ml)**
Fetal urination	Production	1000

Fetal swallowing	Resorption	750
Fetal lung fluid secretion	Production	350
Intramembranous flow	Resorption	400
Transmembranous flow	Resorption	Minimal

FIG 3: AMNIOTIC FLUID PATHWAYS



AMNIOTIC FLUID CHARACTERISTICS

PHYSICAL: It is a clear yellow fluid with a pH around 7.2 and specific gravity of 1.0069-1.008

CHEMICAL COMPOSITION : Changes with gestational age.

- Water constitutes 98 to 99% of amniotic fluid.

Amniotic fluid has a huge number of dissolved substances like:

- Urea
- Bile pigments
- Creatinine
- Glucose
- Fructose
- Albumin and globulin
- Renin
- Hormones like progesterone and oestrogen
- Lipids
- Enzymes
- Minerals like Na+, K+, Cl+
- Undissolved substances like fetal epithelial cells

In the second trimester of pregnancy, amniotic fluid has a decreased osmolarity and it is similar to dilute urine and substances from metabolites.

AMNIOTIC FLUID VOLUME (WATER)

During the period of embryogenesis, Amniotic fluid volume rises in a faster rate compared to the size of the embryo. Amniotic fluid is mainly produced from the

maternal plasma by its passage through the fetal membranes based on osmotic and hydrostatic forces.

In early fetal period, Amniotic fluid volume and the size of the fetus grows in a linear trend. It is evidenced that amniotic fluid volume begins to increase from 25 ml at 10 weeks of gestation to about 400 ml in 20 weeks. During this period, its composition is almost similar to the fetal plasma.

A bi directional diffusion takes place between the amniotic fluid and the fetus across the non keratinized fetal skin. During this period, amniotic fluid serves as an extension of extracellular compartment of the fetus as well as a physiologic buffer,

In the mid second and in third trimester

At 19 to 20 weeks of gestation, fetal skin begins to keratinize and the same usually gets completed by 25 weeks of conception.Internal homeostasis of Amniotic fluid volume is well balanced by inflow and outflow pathway.Inflow pathway includes fetal urine and lung fluid secretion. Outflow pathway includes swallowing and intermembranous absorption of amniotic fluid.

Amniotic fluid production is predominantly constituted by **fetal urine** – around 300 ml/kg of fetal weight/day to 600 - 1200 ml/day at term and **lung fluid production –** about 60-100 ml/kg fetal weight/day[16]. Efflux of lung secretions into AF is mainly contributed by fetal breathing movements. Also it is contributed by oral, tracheal and nasal secretions.

Amniotic fluid removal is primarily accomplished **by fetal swallowing-** about 200 to 250 ml/kg of fetal weight per day.

Also an **intra-membranous pathway** helps in transferring fluid and solutes- 200 to 500 ml/day from amniotic sac to entire fetal circulation crossing through the amniotic membrane[17,18].

Evidence from studies indicates that increased VEGF gene expression[19] in fetal membranes is related with high transfer of amniotic fluid into the fetal circulation. Also presence of aquaporin protein channels[20] in fetal membranes suggests that water channels are another potentiating regulator of water between amnion and placenta.

Trans membrane pathway affects amniotic volume only to a minimal extent .It allows direct exchange of substances across the fetal membranes to the maternal circulation accounting to 10 ml/day at term[16].

Studies reveal that Hormonal changes play a vital role in regulation of amniotic fluid. It has proposed that prolactin[21] present in the decidua exerts an effect on amniotic permeability. Many other hormone related mechanisms are under research.

Uterine perfusion helps in regulation of amniotic fluid. It has proposed that maternal dehydration inturn leads to increased production of vasopressin and arginine as well as increase in fetal plasma osmolality. Thereby this inturn leads to increase in osmolaltity of fetal urine[22].

Amniotic fluid volume, a perfectly regulated process is dependent on the respective gestational age,and it is maintained within a specific fixed range. The amniotic fluid volume peaks between 36 and 38 weeks of about 800-1000 ml and therefore declines to about 400 ml at 42 weeks.

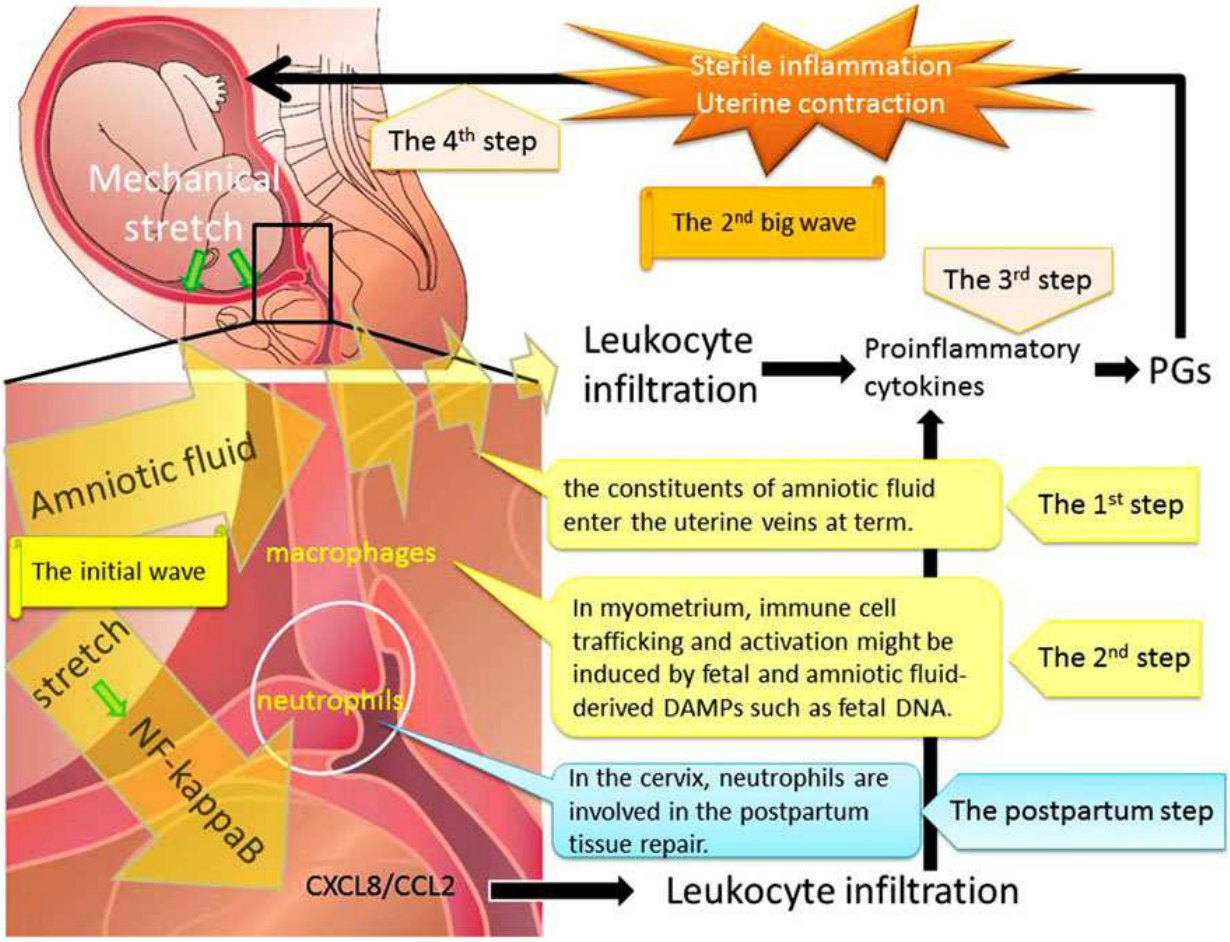


FIG 4: AMNIOTIC FLUID MECHANISM

AMNIOTIC FLUID: DISSOLVED SUBSTANCES

<u>NUTRIENTS</u>

Amniotic fluid constitutes carbohydrates, peptides and proteins, lactate, lipids, pyruvate, enzymes electrolytes and hormones.

Taurine is rich in amniotic fluid and it is the only amino acid present in larger quantity than its concentration in maternal and fetal blood. Whereas other amino acids are present in lower concentrations in amniotic fluid.

Glutamine in amniotic fluid plays a precursor role in nucleic acid synthesis for all the cells and also helps in proliferation of rapidly dividing cells especially intestinal mucosa cells.

Arginine plays a key role in development of both fetus and placenta. Arginine is usually hydrolyzed to ornithine, which in turn is converted to polyamines[23],spermine, and spermidine, putresine. These are the key regulators in placental angiogenesis, embryogenesis and trophoblast growth.

The role of swallowed **carbohydrates as well as lipids** in AF is less well defined. Fetal growth restriction reversal by infusion of nutrients into the amniotic environment has not yet demonstrated in studies.

GROWTH FACTORS

Human fetal intestinal cells have been cultured to study and demonstrated the trophic effects of amniotic fluid[24].Similar to the growth factors present in human milk, the growth factors that are present in amniotic fluid plays a vital role in fetus growth and development.

The trophic mediators in amniotic fluid include :

1.Epidermal Growth Factor(EGF)

2.Transforming growth factor beta-1(TGF-b1)

3.Granulocyte colony-stimulating factor (G-CSF)

4.Erythropoietin

Epidermal growth factor (EGF) – this factor peaks during the mid trimester. In fetal growth restriction, this factor is significantly reduced. Its entire function in human fetus is still under study and remains unknown yet.

Transforming growth factor beta-1 (TGF-b1) – present in the amniotic fluid mainly during the last trimester. This factor plays a role in inducing terminal differentiation of the intestinal epithelial cells and also in accelerating the healing process of intestinal wounds by inducing cell migration. It also stimulates production of IgA[(25)].

The entire neonatal gut comprises of Insulin-like growth factor 1 (IGF-I) and IGF-II receptors[(26)].

Granulocyte colony-stimulating factor (G-CSF) :studies have come out with extensive growth of intestinal cells in sucking mice, when this factor was administered enterally.

Erythropoietin: Is present in amniotic fluid, mature milk as well as in colostrum. The function of swallowed erythropoietin in human fetus is not yet clearly understood. Its concentration in amniotic fluid is related proportionally with increased umbilical cord blood erythropoietin concentration, and hence increased amniotic fluid erythropoietin is indicated as a marker for chronic hypoxic status of the fetus[27].

IMMUNE FACTORS

Many substances that represent the immune system has been found in amniotic fluid and vernix. They have also been found to posess antimicrobial property.

They include:

1. The human beta defensin
2. alpha defensin (HNP1-3)
3. Lactoferrin (LF)

The human beta defensin – represent a major portion in the family of natural antimicrobials in vertebrates. Human beta defensins 1-4 are present at mucosal surface. In addition, they have chemo attractant properties and they have also found to have interaction between the adaptive and innate immune system. It functions as a chemokine which is involved in parturition.

Few other antimicrobial agents like **alpha defensin (HNP1-3),** whose concentrations have been increased in the amniotic fluid in conditions like preterm labour,

chorioamnionitis and premature rupture of membranes are probably because of its release from neutrophils.

Lactoferrin (LF), a glycoprotein which has two sites for binding for ferric iron appears in human amniotic fluid as early as 20 weeks of gestation. Its concentration rises as with gestation. It is found to be secreted in the amniotic fluid by amniotic cells and neutrophils.

Lactoferrin possess both bacteriostatic as well as bacteriocidal activity. It exerts its bacteriostatic action by sequestrating iron thereby making it unavailable to aid in growth of microbes. Its bactericidal action is exhibited by binding over the outer membranes of the bacteria and triggering the release of liposaccharides. Lactoferrin releases a microbicidal cationic peptide by the action of enzymes at acid pH called Lactoferricin. Lactoferricin exhibits microbicidal action against protozoa , virus and fungi. With the onset of labour, lactoferrin levels decreases.

Other high potent antimicrobial agents exhibiting broad spectrum activity against viruses, bacteria, fungi and protozoa present in amniotic fluid are calprotectin, secretary leukocyte protease inhibitor, psoriasin, and cathelicidin[28,29,30]. Certain polyamines present in Amniotic fluid possess a cationic charge and has a role in both antimicrobial and nutritive value.

Cellular innate immune system: Mononuclear phagocytes (ie., monocytes,histiocytes, and macrophages) in the amniotic fluid are found to be limited in normal pregnancies,whereas they are increased in fetuses with multi neural tube defects.It

is still uncertain that these macrophages are present in order to prevent infection due to disruption of fetal skin or behave as scavenger cells to clean the neural debris .

Neutrophils when present in amniotic fluid, indicates the possibility of infectious foci in the amniotic fluid as these are not normally present in the healthy fetuses.

FETAL CELLS:

Amniocentesis is a popular invasive diagnostic technique performed prenatally for diagnosing choromosomal and genetic abnormalities. In this procedure, amniocytes and the fetal cells that are shed from the skin, gut and genitourinary system along with biochemical substances are removed and analyzed.

PARTICULATE MATTER:

In the third trimester, the term ‘echogenic amniotic fluid' represents the evidence of vernix caseosa or meconium. Pulmonary surfactant produced by the type 2 alveolar cells induces a ‘roll up’ phenomenon where it detaches the vernix caseosa from the surface of fetal skin. Certain congenital anomalies have been found to be associated with presence of particulate matter in amniotic fluid. These include

fetal acrania

harlequin icthyosis

epidermolysis bullosa fetalis.

STEM CELLS:

Amniotic fluid constitutes different cell types derived from the developing fetus. It also includes the cells that are potent to get differentiated to adipose, bone, muscle and neurons.

Thus stem cells derived from Amniotic fluid are pluripotent and are found to possess the ability to differentiate into a huge number of lineages including those that are present in all three embryonic germ layers[12,13].

Amniotic fluid stem cells are said to have all biophysical characteristics of both adult and embryonic stem cells Therefore they are advantageous over both adult and embryonic stem cells:

1. Easily accessible
2. Replicates rapidly in culture as they double every 36 hrs
3. Does not require support from other feeder cells which will cause contamination
4. Does not form tumours in vivo

Amniotic fluid stem cells also possess therapeutic properties[14] and behave as precursors to a wide range of differentiated cell types. It has been evident from studies that amniotic fluid stem cells can be engaged in repairing damaged tissues from conditions like cartilage damage, spinal cord injuries, stroke and diabetes.

OLIGOHYDRAMNIOS

According to Literature, incidence of oligohydramnios are varied from 0.5% to > 5%, depending on the definition of oligohydramnios and study population. As development of lung and limbs require adequate amniotic fluid, oligohydramnios possess a negative impact on that development.

Oligohydramnios is found to be associated with

- pulmonary hypoplasia
- postural deformity
- fetal distress
- perinatal morbidity and death.

DEFINITION:

Oligohydramnios is defined as amniotic fluid volume less than expected for gestational age. Amniotic fluid volume measuring less than 500 ml at 32 to 36 weeks of gestation is oligohydramnios.

Amniotic fluid volume depends mainly on the gestational age, therefore the best definition could be the one that is less than fifth percentile.

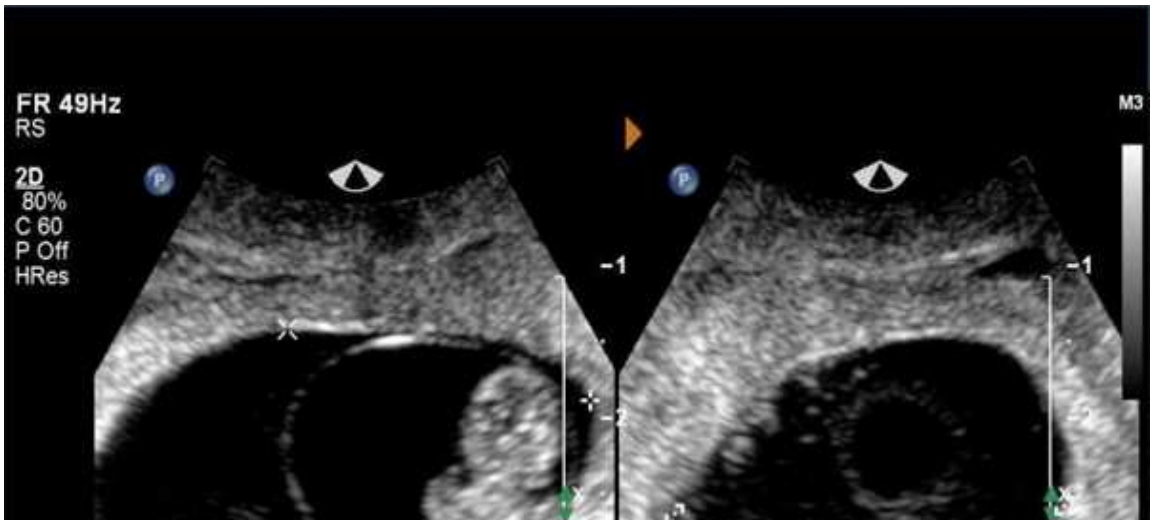
Oligohydramnios is defined by USG as an amniotic fluid index 5 cm or less or Singe deepest pocket (SDP) of amniotic fluid value less than 2 cm is oligohydramnios.

ETIOLOGY:

The leading causes of oligohydramnios varies according to the trimester.

FIRST TRIMESTER: The etiology of oligohydramnios in the first trimester is ill understood. It is diagnosed when the difference between the mean sac diameter and the CRL is less than 5 mm. At this gestation, the fluid is contributed almost entirely by diffusion through the placental surface of the membranes. While this has been seen as a sign of poor prognosis, strong evidence is not available in large studies. These pregnancies should be followed up to the second trimester before any clinical decision can be made. Severe oligohydramnios/anhydramnios in the first trimester, carries a uniformly unfavorable diagnosis.

FIG 5: AMNIOTIC FLUID IN USG IN FIRST TRIMESTER



SECOND TRIMESTER: In the 2nd trimester, fetal urine contributes to the amniotic fluid volume significantly. Renal abnormalities hence form an important etiology. The causes can be sub classified as due to fetal, placental and maternal causes.

a) Placenta and Membranes: Utero placental insufficiency which causes early onset IUGR can result in oligohydramnios. The severity and onset depends on the degree of dysfunction and the fetal adaptation. Preterm prelabour rupture of membranes(PPROM) contribute to one third of the cases according to one study.

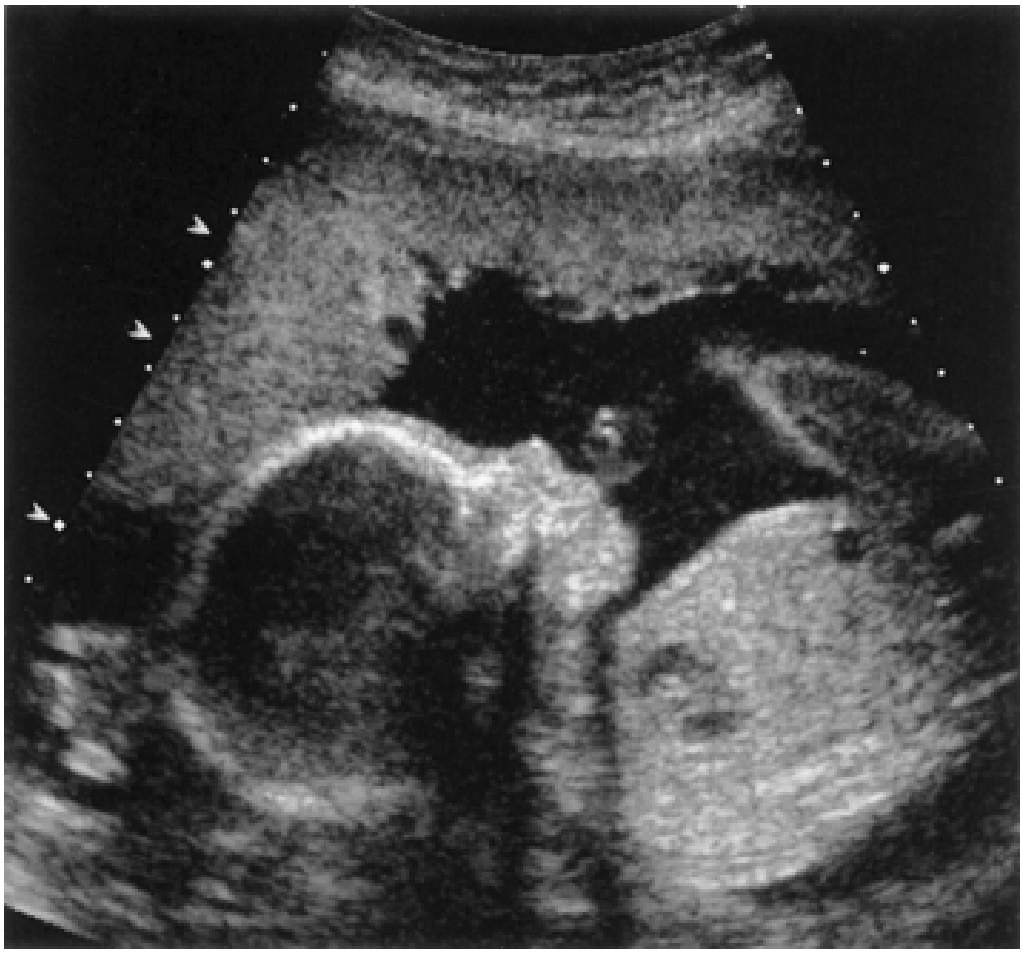
b) Fetal : The commonest problem being bilateral renal agenesis which presents as anhydramnios as early as 15-16 weeks. In conditions of unilateral renal agenesis, liquor volume is maintained normally. Problems occur when there is an insult to the solitary functioning kidney which may be in the form of cystic renal dysplasia or pelviureteric junction obstruction. Problems of lower urinary

tract namely, bladder outlet obstruction due to posterior urethral valve and urethral atresia can cause oligo/anhydramnios.

The timing of onset may vary. Aneuploidy accounts for 8% of the cases. Multisystem anomalies account for the rest. Diagnostic procedures like amniocentesis or therapeutic procedures for multiple pregnancies involving fetoscopy can also cause oligohydramnios due to PPROM

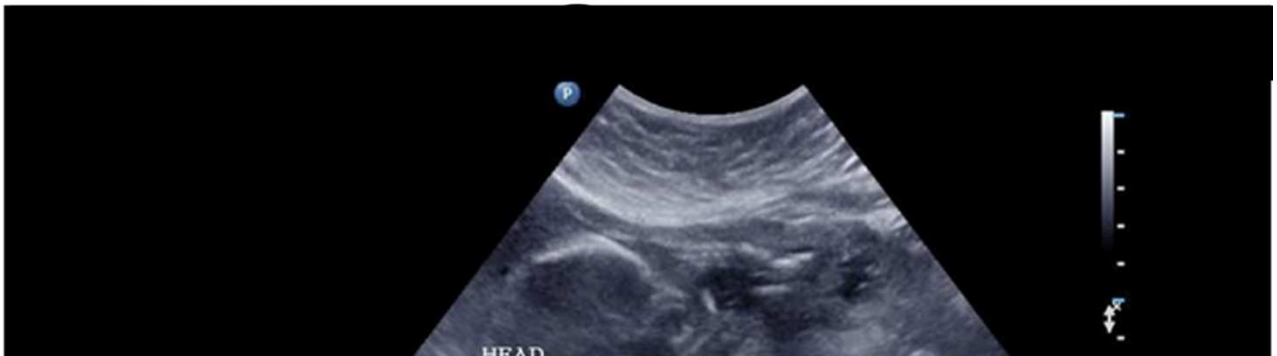
c) Maternal : Maternal systemic disorders including chronic hypertension, nephropathy and connective tissue disorders can result in reduced liquor volume, secondary to placental insufficiency and IUGR. Maternal drug intake mainly, NSAID's and anti hypertensives (Angiotensin receptor blockers – ARB's) have been proved to cause oligohydramnios or even anhydramnios due to renal dysfunction.

FIG 6: AMNIOTIC FLUID IN SECOND TRIMESTER



THIRD TRIMESTER : Oligohydramnios in third trimester, is predominantly due to PROM. Fetal growth restriction can result in oligohydramnios as detailed above. Many cases of third trimester oligohydramnios are due to idiopathic causes. Feldman et al in a study in 2009, determined that oligohydramnios is more common in summer months due to reduced maternal hydration**.**

FIG 7: AMNIOTIC FLUID IN THIRD TRIMESTER



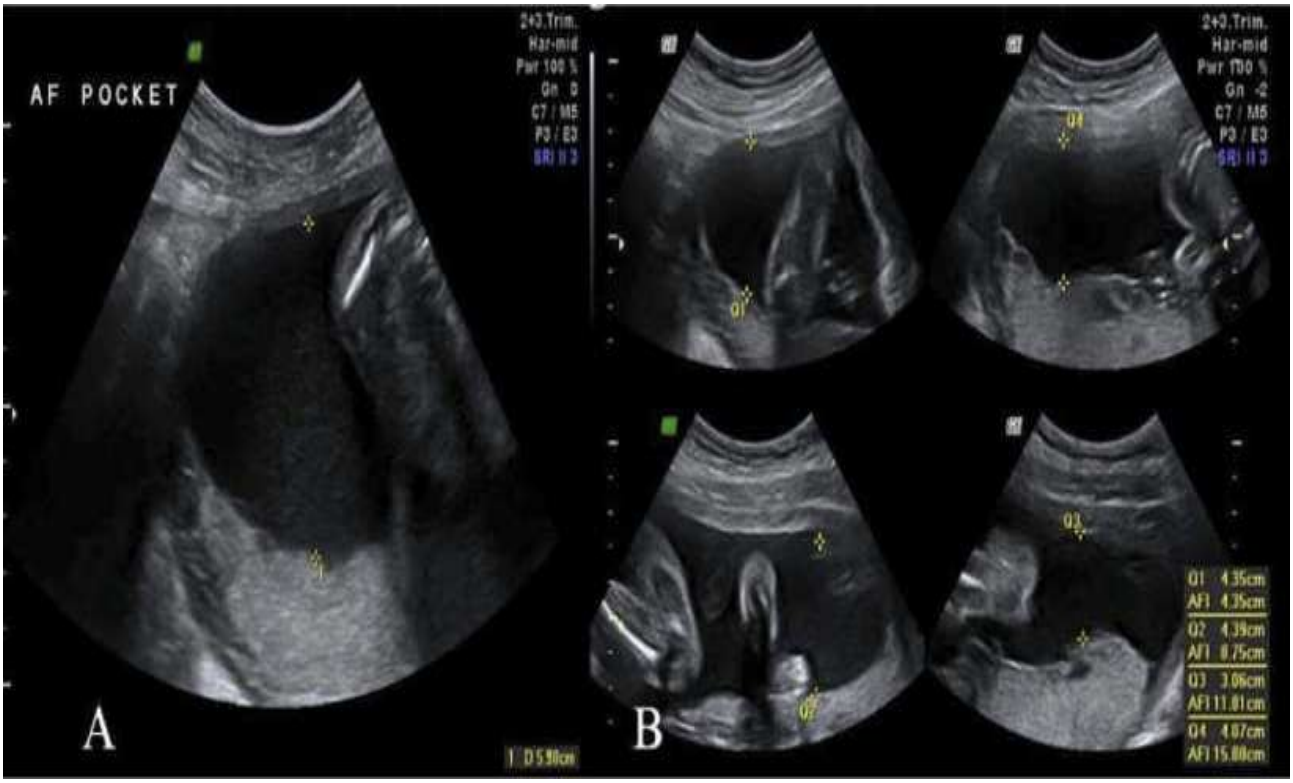
DIAGNOSIS:

There are no specifc symptoms. Some of the pointers may be H/O leaking per vaginum, post term pregnancy, preeclampsia, drugs and less perception of fetal movements. On clinical examination the uterus may be small for date ie. Smaller symphysio fundal height and feels full of fetus because of scanty amniotic fluid. There can be malpresentations and IUGR.

ASSESSMENT OF AMNIOTIC FLUID BY USG

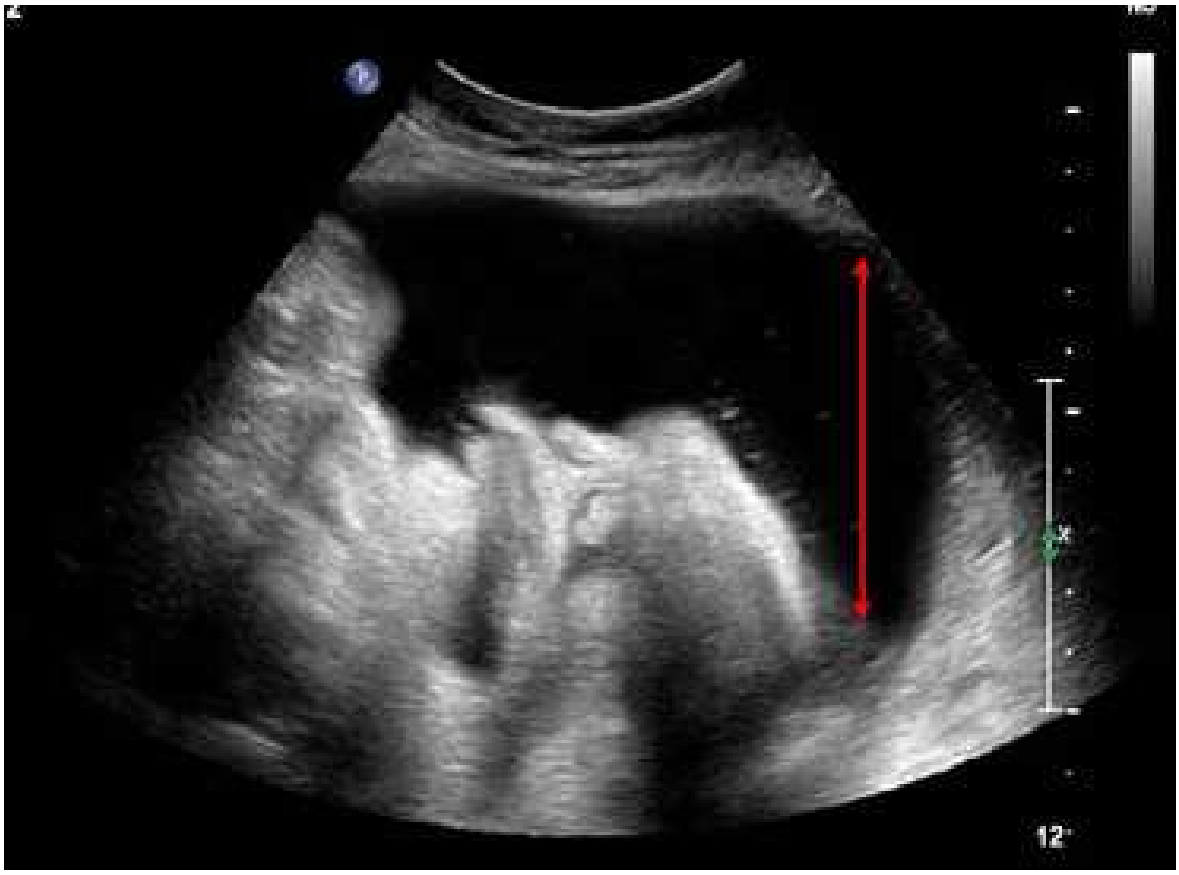
Ultrasound measurement of amniotic fluid can be assessed either as subjective assessment or as a semi quantitative method. Semiquantitative methods include single maximal vertical pocket (SVP), the two diameter pocket technique and the amniotic fluid index (AFI)[3,4].

FIG 8: ASSESSMENT OF AMNIOTIC FLUID



SINGLE DEEPEST VERTICAL POOL: In the Single Vertical Pool method, the deepest vertical pool is identified and then the transducer is placed perpendicular to the contour of the uterus. The maximum vertical diameter of the amniotic pool devoid of cord and fetal parts is measured. It is important that at this level, the horizontal component of the pocket should be more than 1 cm.

FIG 9:SINGLE DEEPEST VERTICAL POOL IN USG



The interpretation of the SVP is as follows[3]:

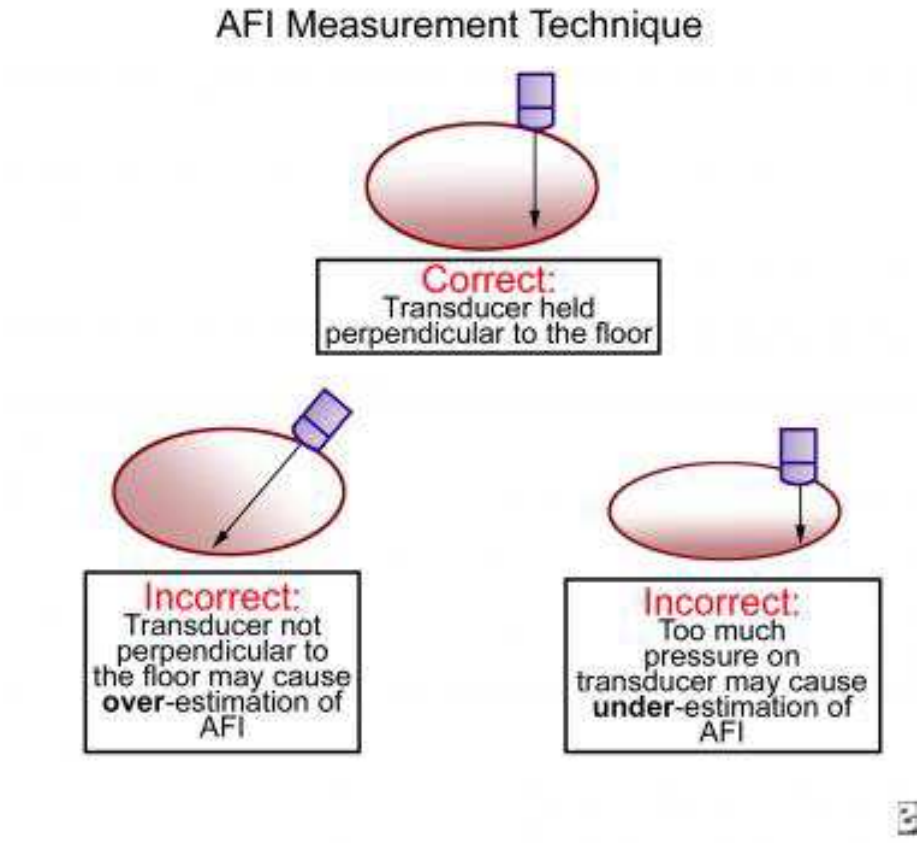
- Oligohydramnios - depth <2 cm
- Normal- depth ≥ 2 cm and < 8 cm
- Polyhydramnios- depth ≥8 cm

AMNIOTIC FLUID INDEX(AFI) : AFI is a 4 quadrant method wherein the uterus is divided into 4 quadrants by a horizontal and vertical line intersecting at the umbilicus. The single deepest vertical pool is measured in each quadrant and the sum of all the four quadrants in cms. This technique is used in late second trimester and in the

third trimester and not used in early first and early second trimester as the fundal height does not cross the umbilicus. The interpretation of AFI is as follows[3]

- Oligohydramnios: AFI≤ 5 cm
- Normal: AFI >8 cm and < 20 cm
- Polyhydramnios: AFI ≥24 cm

An AFI of 5.1 – 8 cm may be considered as Borderline Oligohydramnios[4] and AFI of 21-23 cm may be considered Borderline Polyhydramnios.

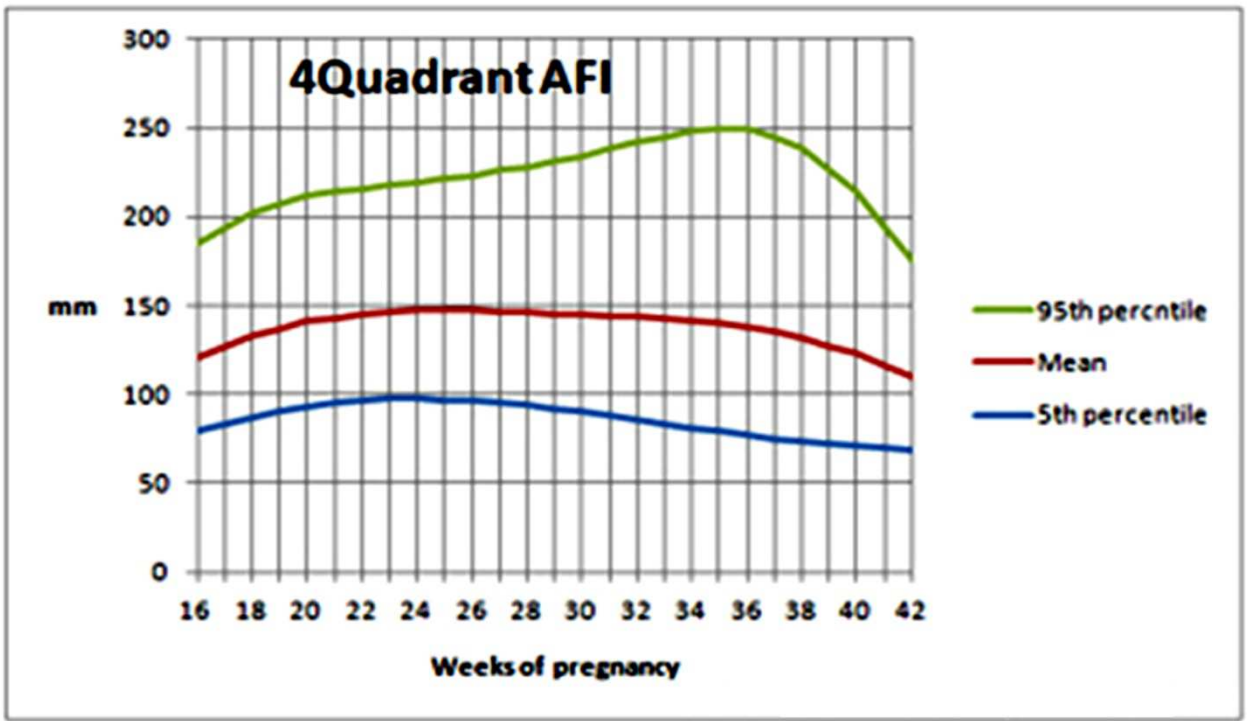


TWO DIAMETER POCKET TECHNIQUE: This technique involves measuring and multiplying the vertical and horizontal diameter of a pocket which is devoid of cord or fetal parts. The interpretation of this is as follows[5]:

- Oligohydramnios: 0 to 15 cm
- Normal: 15.1 to 50 cm
- Polyhydramnios: greater than 50 cm

There is no agreement in the obstetric literature as to which method of sonographic measurement of amniotic fluid volume is best. The investigators concluded that the minimal improvement in accuracy offered by another ultrasound measurements was not sufficient to warrant replacement of the AFI[6].

The upper limit of normal amniotic fluid index is defined a s a value of AFI ≥ 25 cm and it can be used for all gestational age and also can be utilized as gestational-age specific threshold. Neither method is shown superior to other method[7].



COMPLICATIONS:

FETAL

- Prematurity
- Abortion
- IUFD
- Potters syndrome- characteristic facies, limb deformities and pulmonary hypoplasia
- Deformities- CTEV, contractures, amputation
- Malpresentations
- Cord compression
- Fetal distress
- Meconium stained amniotic fluid- Meconium aspiration syndrome
- Low APGAR
- Amnion nodosum

MATERNAL:

- Prolonged labour- dystocia and uterine inertia
- Increased operative interference
- Increased morbidity

NEWER CONCEPTS IN MANAGEMENT OF OLIGOHYDRAMNIOS

In conditions like fetal obstructive uropathy, where oligohydramnios is commonly encountered , as a treatment procedure Vesico-amniotic shunts are found to be quite effective in treating low amniotic fluid levels[11]. But its effectiveness in maintaining pulmonary and renal functions is questionable yet.

L-arginine , a precursor of vasopressin is a potent treatment option in the treatment of oligohydramnios[8)A].

Newer drugs:

Herbal extract of Salvia miltiorrhiza in its purified form have been found to improve the amniotic fluid volume in preterm through increasing uteroplacental perfusion and circulation. A Chinese medicine, Salvia miltiorrhiza is found to be effective in the treatment of oligohydramnios[9].

The drug hemodialysate(solcoseryl) effectiveness on liquor volume in intra uterine growth restriction (IUGR) babies and its outcome in form of APGAR score and health status studies concludes that Solcoseryl remains as a drug of choice in pregnancy with IUGR, after 28 weeks of gestation[10].

MANAGEMENT

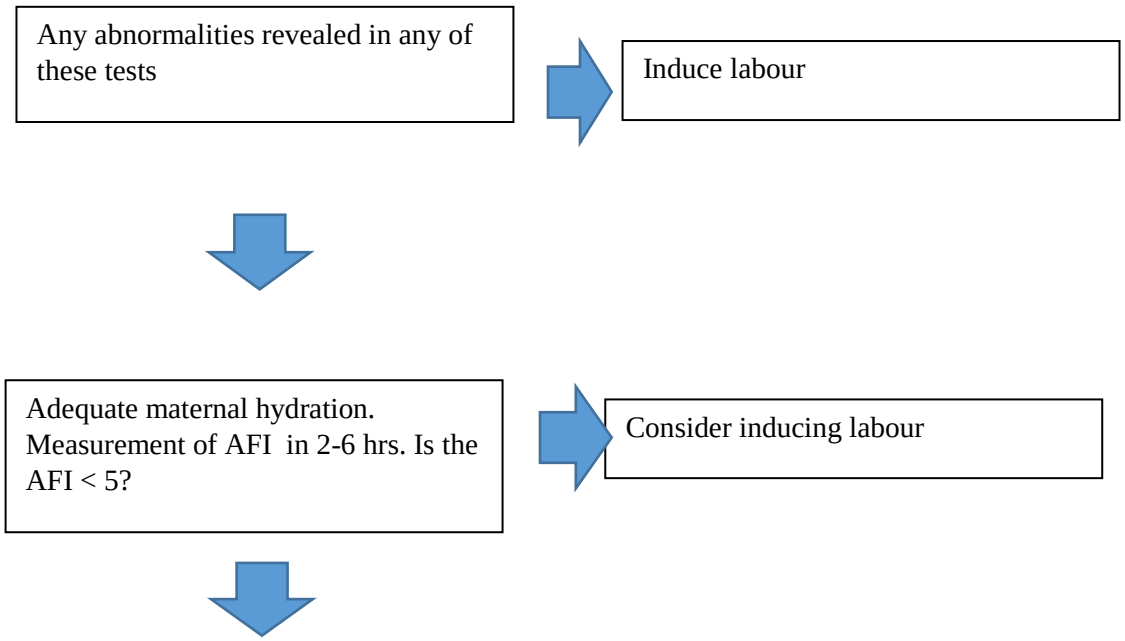
Depends upon

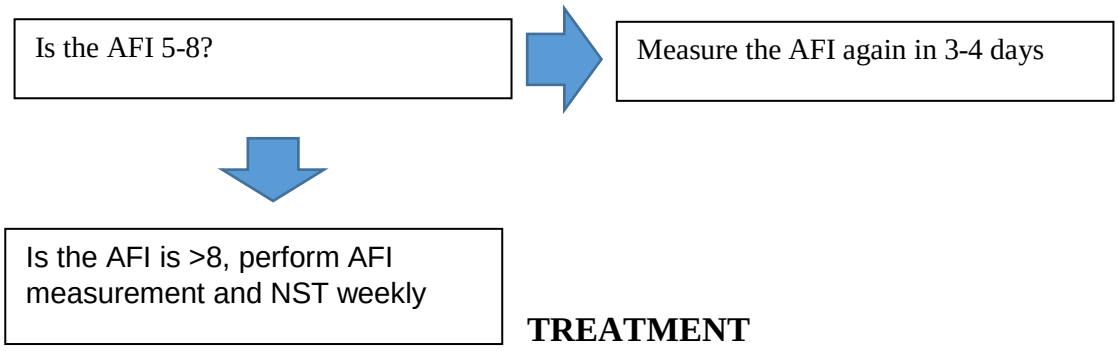
- Aetiology
- Gestational age
- Severity
- Fetal status and well being

Assessment of the pregnant woman with oligohydramnios at term

A woman with gestational age in between 37-41 weeks with AFI <=5 cm

1. Look for evidence of ruptured membranes.
2. Correct her dates.
3. Order non stress test (NST) to evaluate fetal well being.
4. Determine presence of hypertensive disorders, diabetes and conditions leading to uteroplacental insufficiency.
5. Order USG for IUGR and fetal congenital anomalies.





TREATMENT

- Improve Hydration- plenty of oral fluids
- Adequate rest
- Hydration – Oral or IV hypotonic fluid (2 litres/ day)
  - Temporary improvement
  - Betterment during labour, just prior to ECV and USG
- Serial Ultrasound – to monitor growth,BPP, AFI
- LSCS or Induction of labour depends upon
  - Lung maturity status
  - Lethal anomalies
  - Severe IUGR
  - Severe oligohydramnios

AIMS AND OBJECTIVES

To determine Obstetric outcome in term low risk pregnancy with AFI less than or equal to 5.

To assess whether antepartum oligohydramnios is associated with adverse perinatal outcome.

REVIEW OF LITERATURE

Oligohydramnios is a frequent complication of pregnancy that is found to be associated with a bad perinatal outcome. Etiologies include premature rupture of membranes (PROM),congenital anomalies, intrauterine growth restriction (IUGR), post datism, drugs, abruptio placentae, twinning abnormality, severe maternal illness and idiopathic oligohydramnios.

Prolonged oligohydramnios leads to pulmonary hypoplasia and fetal compression syndrome. Perinatal morbidity and mortality are significantly increased in pregnancies with oligohydramnios. Ultrasound finding of oligohydramnios should remind the clinician to evaluate the patient for other significant illness.

Further a thorough fetal anatomic study highlighting on the genitourinary tract and looking for free amniotic bands should be done with ultrasound. Karyotyping plays an important role.

Once diagnosed, oligohydramnios, should further lead to intensive fetal surveillance including ultrasound evaluation. The role of amnioinfusion as a support to continuous fetal monitoring in labour to improve neonatal outcome appears beneficial in selected study series.

David G. Weismiller[31]; Said the two most common uses of transcervical amnioinfusion are dilution of thick meconium fluid during labour and treating decelerations. In 1983, Hill-LM[32]; et al concluded that oligohydramnios remains as an important factor for intrauterine growth restriction and has been studied in populations prone to have IUGR. They studied the incidence in obstetric population. During 16-months of study period, USG scans were done in 1,408 patient. Severe oligohydramnios were found in six patients (0.43%). Two of them found to have congenital malformations, 4 were found to have severe IUGR. A subgroup of 317 patients who were scanned within 2 weeks of confinement was then reviewed. Although none of the patients fitted the proper criteria of oligohydramnios (absence of a pocket of amniotic fluid 2 cm or less in largest diameter), three of six small-for-gestational age infants, had a subjective decrease in amniotic fluid volume for their respective gestational age. By changing the criteria of oligohydramnios to a subjective decrease in amniotic fluid volume, the sensitivity of ultrasonic marker was increased by 50% and the specificity by 100%. They found out that the semiquantitative assessment of amniotic fluid volume can be used to effectively screen for IUGR, regardless of gestational age, at risk for perinatal morbidity and mortality.

Eighty pregnancies encountered by oligohydramnios forms the basis of a retrospective study done by [33]Shenker-L et al. Forty patients had PROM ; of which 24 had good outcome. Twelve of 14 fetuses with oligohydramnios and IUGR had good survival rate. None of nine fetuses with renal anomalies were alive. None of the twin with twin to twin transfusion syndrome and oligohydramnios lived. Six pregnancies with premature separation of placenta and with oligohydramnios were identified; all of them resulted in fetal or neonatal death during the (10) second trimester.

William-K[34], said amniotic fluid measurement plays a very important role in assessing fetal well being because it has an advantage of being non invasive. Uterine cavity being not regular is difficult and complicated to find indirect techniques.

The three ultrasound methods include

1)assessment of the subject

2) maximum vertical pocket depth

3) amniotic fluid index (AFI).

All three techniques aid in predicting peripartum mortality and morbidity by measuring amniotic fluid volume.

Amniotic fluid volume (AFV) remains a good predictor of perinatal outcome when used in combination with fetal biophysical profile to determine perinatal outcome.

Youssef AA, et al[35]; quoted semiquantitative Amniotic fluid volume determination is one of the component of fetal biophysical profile . TO determine

reduced AFV they performed BPP for 174 fetuses with in one week of confinement. Two techniques was used,one by measuring single vertical pocket depth (oligohydramnios <1cm) and 4 quadrant AFI (AFI <5cm). AFV, studied by each technique were associated with fetal outcome measures (meconium stained amniotic fluid, fetal distress, IUGR, lower Apgar score and perinatal morbidity).

The AFI remains highly sensitive in predicting mortality (87.5%) and also other measures of perinatal morbidity: fetal distress amounting to 86.6%, meconium stained fluid to 63.6%, low 5-minute Apgar score to 88.8% and IUGR with 79.4%. The sensitivity of AFI <1cm was 72.2%, 66.6%, 75% and 55.8%, respectively for the respective measures.

Their study data determines that qualitative measurement of AFV using AFI remains better to that of single vertical pocket in fetal biophysical surveillance. In 1989, sixty-two cases of olighydramnios diagnosed by ultrasound between 13-28 weeks (12) gestations were reviewed by Three Moore-TR et [a(36)l]; skilled ultrasonographers rated oligohydramnios into grades of mild, moderate, severe, or nil. Inter observer views were excellent. The overall incidence of pulmonary hypoplasia was 33% and perinatal mortality were 43%. One-third found to have fatal congenital anomalies. The severity of adverse outcome corelated severely with severe most degree of oligohydramnios; with only 11 % in mild and moderate group and 88% with severe oligohydramnios or anhydramnios underwent adverse outcomes. The presence of urogenital tract anomaly was associated with the severe degree of oligohydramnios and remained fatal .

Pulmonary hypoplasia was found evident in 6% of the moderate group and 60% in the severe group.

They came to a conclusion that subjective grading in oligohydramnios by skilled observers remained both predictive and reliable of perinatal outcome. The evidence of severe oligohydramnios in second trimester was strongly predictive of adverse fetal outcome as it necessitates a thorough search for etiology and prompt intervention. Moderate grades of decreased amniotic fluid may be managed with optimistic attitude.

In1986 Sivit CJ; et al[37] reviewed ultrasonograms of all patients with oligohydramnios between 16 and 30 weeks of gestation over a period of 4 years to understand:- 1- Whether sonographically identified fetal anomalies were present. 2- When these anomalies been present, how could this information be used in managing maternal and fetal outcome. Cases of fetal demise and ruptured membranes were excluded from the study.

Sixteen patients with severe oligohydramnios was identified. On postmortem examination, nine were found to have urinary tract anomalies, one with evidence of chronic intrauterine infection, and four with no anomalies. Only two neonates survived: one had no anomaly while the other had posterior urtheral valve. These findings concluded that second trimester oligohydramnios had poor prognosis and were often related with anomalies of urinary tract. Sonography helps in the clinical management of those patients.

Outcome of structural pathology was studied in 170 chromosomally abnormal fetus by Wladimiroff JW[38], referral for those before 20 wks gestation was based only fetal structural pathology (92%); after 20 wks patients referral was based on structural pathology combined with small for gestational age polyhydramnios/oligohydramnios.An abnormal AFV were present in 59 out of 170 (34.5%) chromosomally affected pregnancy i.e. polyhydramnios in 28 and oligohydramnios in 31 cases with adverse fetal outcome.

Mercer-LJ; Brown LG[39] diagnosed 34 cases of oligohydramnios in 2nd trimester in ultrasound scan. 9 of of those pregnancies was associated with fetal anomalies like atrioventricular dissociation (two), Potter syndrome (three), multiple anomalies (three) and congenital absence of thyroid (one). There was ten unknown stillbirths, only one fetal demise due to abruptio placentae, eight with perinatal morbidity after preterm labor and/or abruptio placentae, and six were live- born full term infants. Although oligohydramnios in the 2nd trimester were found to be associated with high perinatal morbidity, this finding was not associated with poor outcome universally.

In 1997,eighteen cases with bladder outlet obstruction was diagnosed [40] at Queen Mother's Hospital for a 6 yr period. Commonest cause found to be was posterior urtheral valve observed in 11 cases (61%) oligohydramnios was present in 78%of patients with mortality of (67%) mainly due to pulmonary hypoplasia. The six survived infants (33%) underwent early postnatal surgery. Two of these were found to develop chronic renal failure on further follow up.

Levine-D; et al[41]; said preterm fetuses with breech presentation had more incidence of dolichocephaly than with the ones who had cephalic presentation and it was more common in oligohydramnios with longer duration. 10 out of 12 living dolichocephalic fetuses with cephalic presentation developed respiratory distress syndrome compared with 31 of 73 (42%) in normocephalic fetuses. They concluded that dolichocephaly was found to be associated with oligohydramnios of prolonged duration .In fetuses with preterm rupture of membranes, it is associated with respiratory distress syndrome.

In 1984 Barss-VA[42]; et al reported 12 cases of severe oligohydramnios in second trimester. The outcome of those pregnancies were poor all throughout, with no survivors in the entire series. Of them, Four patients had therapeutic abortions, one of them underwent spontaneous labor at 22 weeks, and seven of them continued till viability. Of these, around five patients were found to have severe renal anomalies that were not compatible with life .Two infants died near or immediately after, birth due to severe intrauterine growth retardation (IUGR), one had a triploid karyotype. Review of the literature showed a similar adverse outcome with severe oligohydramnios in the 2nd trimester.

Wolff-F et al[43] studied complications in oligohydramnios in past medical records, Of all 5,210 of deliveries between 1987 and 1990, 181 cases with oligohydramnios were studied. The diagnosis was by USG and was based on the method described by Plat and Mannings: Amniotic fluid pockets of <1cm were defined as oligohydramnios.All of 181 meet the above definition, that resulted in an incidence 3.5% pregnancies with decreased

amniotic fluid. They reviewed the records of both fetus and the mother, the delivery and its perinatal complications and the follow up of newborn. In more than 60% fetal asphyxia, breech presentation and other abnormalities during delivery lead to Cesarean section and vaginal operative delivery by application of forceps or vaccum extraction. In 30 cases, oligohydramnios were found to be caused by PROM. In the remaining cases, diagnosis was based on fetal and maternal complication such as abruptio placentae (n=10, multiple gestation (n=7),), fetal growth retardation , and malformations or chromosomal anomalies. Eighty-nine newborns was referred to a children's hospital after delivery. Potter's syndrome was the most anticipated diagnosed malformation (n=20). The decreased amniotic fluid was further more followed by a high rate of respiratory complications such as hypoplasia of the neonatal lung (n=21) and Respiratory Distress Syndrome.

In conclusion, their study highlighted that oligohydramnios should be considered as marker of high risk pregnancy followed by other complications of fetus and newborn .The incidence of perinatal mortality in their study was 7.2% and this rate was found to be 10 fold higher compared to total number of the deliveries in their department.

Golan –A; et al[44] diagnosed 145 cases of oligohydramnios in 2nd &3rd trimester by ultrasonogram out of 25,000 obstetric patients amounting to 0.58%. In that group, common complications encountered are induced hypertension in 22.1% and bleeding in the 2nd trimester in 4.1%. There was a high incidence of meconium-stained amniotic fluid in 29.1% fetal distress in 7.9% and premature separation of placenta in 4.2% IUGR

(24.5%) . Asphyxia during labour was encountered in (11.5%) and many other perinatal problems in (23.5%). Cesarean section were performed in 35.2% of those pregnancies, 17% of cases presented by breech. IUFD were in (5.5%) of those pregnancies. The overall perinatal mortality amounted to (16%) and the corrected perinatal mortality amounted to 10.7%. The overall fetal malformations was in 11% & that fatal malformations were in 4.8%. The urinary system (4.1%) and skeletal (7.6%) were the systems affected. Oligohydramnios was associated with increased rate of fetal morbidity and mortality, thus termination must be decided in case of fetal distress.

In 1990 Lin-CC; et al[45] studied 147 cases of suspected IUGR based on ultrasonographic abdominal circumference below 10th percentile, 56 were confirmed as IUGR infants. Eight of 316 control fetuses with abdominal circumference above the 10th percentile figured out to be IUGR infants at birth. The incidence of oligohydramnios was stunningly different among the other three groups: 29% for IUGR group, 9% for non-IUGR group and 0.6% for the control group (P less than 0.001). When the same criteria of abdominal circumference below the 10th percentile & the presence of oligohydramnios were combined together, the positive predictive value seemed to be doubled (from 38.1 to 66.7%; P less than 0,01). Finally there was no significant difference between fetal growth retardation with or without oligohydramnios, in respect to maternal high risk factors or the fetal outcome. They confirmed that in fetal growth retardation, the occurrence of oligohydramnios during the 3rd trimester of pregnancy was not strongly associated with adverse fetal outcome.

In study done by Conway-DL[46]; et al women who underwent labor induction for isolated oligohydramnios between 37 to 41 weeks of gestation were counter matched by gestational age and parity to women with normal AFI who presented in spontaneous labor. Pregnancies complicated by hypertension, diabetes, fetal anomalies, or suspected IUGR were excluded. The primary outcome variable was mode of delivery. Secondary outcomes examined were presence of meconium, acidosis, low Apgar score and NICU admission. A total of 183 women underwent induction for isolated oligohydramnios. When compared to the control group, neonatal outcome had no difference in the group induced for oligohydramnios. However the women who were induced had significantly more Cesarean section deliveries (15.8% vs. 6.6%, P<0.01, odds ratio 2.7). They concluded that isolated oligohydramnios in otherwise normal term pregnancy might not be a marker for fetal compromise; an induction of labour might not be warranted in most cases.

Hsich TT et al[47] ; investigated the perinatal outcome of patients with oligohydramnios (AFI <5cm) but excluding PROM and congenital fetal malformations, data from (245) singlet pregnancies were thoroughly investigated and compared with normal AFVs (5<AFI <24 cm n=27, 261).High incidences of primigravida, premature separation of the placenta, past history with IUFD, past history of preterm, post term pregnancy and advanced maternal age were noticed to occur with oligohydramnios. Pregnancies complicated by severely diminished AFV identified antenataly by ultrasonogram were more frequently associated with adverse perinatal outcomes such as

preterm delivery, low/ very low birth weight, low Apgar scores, IUFD, SGA newborn, meconium stained amniotic fluid, C/S delivery and neonatal death. Indomethacin is a non-steroidal anti-inflammatory agent widely used in the treatment of premature contractions. Indomethacin-induced oligohydramnios is a well documented side effect, and is strongly recommended as an indication for treatment discontinuation.

Shen-O, et al[48] presented a case of prolonged anhydramnios, secondary to indomethacin therapy with no apparent ill effect on the fetus or neonate. [49] Los-FJ et al stated early 2nd -trimester oligohydramnios were associated with normal levels of maternal alpha-feto protein (MSAFP) in 9 out of twenty six cases (35%) of congenital malformations of fetal urine tract that resulted in anuria was found to present in 9 cases; of which 7 being normal MSAFP level, were measured . Whereas, normal (MSAFP) level, was present in 2 out of 17 cases with no fetal malformations. These suggests that the fetal urine contributes the source of elevated AFP in the maternal compartment in the early 2nd –trimester with oligohydramnios. This is supported by lack of relation-ship between concentration of MSAFP with concanavalin A, created from AF pool (AFP), and its presence of fetal diuresis . 3 of 26 women had early recurrent risk of this condition.

Sarno-AP[50]; et al studied 200 term gravidas who presented in the latent phase of labor with vertex- presenting fetuses. An intrapartum AFI <5cm was associated with significant increase in Cesarean section in view of fetal distress and an Apgar score of <7 at one minute and abnormal fetal heart rate , the majority (71.4%) of the patient with

an intrapartum AFI <5cm had ruptured membranes on their admission; but, there was no profound significant difference found in outcome when they were compared to patients with intact membranes and oligohydramnios. Variable decelerations on admission were associated with oligohydramnios in (43.8%) of the patients. An AFI <5cm in their intrapartum period is a major risk for perinatal morbidity & abnormal fetal heart rate patterns in labor, and ruptured membranes in early labor are risk factor for oligohydramnios.

Sadovsky-Y[51], et al tested the hypothesis that patients who have a large cord-containing AF pocket has a lower risk for adverse perinatal outcome than women with small cord –containing AF pocket . Gravidas with an antepartum AFI not > 5 cm were thoroughly studied prospectively. The vertical diameter of the single largest cord-containing pocket, which was excluded from the calculations of the AFI, was measured. Women of ruptured membranes, multiple gestation, or fetal anomalies were excluded. Results of 51 women with gestational age of 35- 43 weeks were analyzed. Among 35 who had a cord –containing pocket of no > 5cm, 8(23%) had fetal distress demanding operative delivery and eight neonates had cord arterial PH below 7.2. None of these complications occurred in the16 women who had cord-containing pocket more than 5 cm (P <0.05). The mean AFI (2.9 versus 2.8 cm) was not significantly different between the groups. Conclusions: Among women with a low AFI, cord – containing pocket above 5cm identified a subset of women at lower perinatal risk compared with those with smaller cord-containing pocket.

Van-Reempts P[52]; et al studied outcome of neonates after very prolonged premature rupture of membranes (VPPROM) in 3 categories of neonates who were born before 34 to 52 weeks of gestation: group (1) VPPROM with oligohydramnios (n=14);, group (2) VPPROM without oligohydramnios-(n=28); and group (3), the comparison group without VPPROM (n=39). Mortality in the group (1) (2 of 28) was similar to that in group (2) (6 of 39) and was lower than that in group (2) with 5 of 14. Lung hypoplasia and the limb deformities does not occur frequent in group (1) than in group (3) (2of 28 and 0 of 28 versus 3 of 39 and 1 of 39 respectively) but occurred more frequently only in group (2) (5 of 14 and 4 of 14). All deaths in group (1) and (2) were because of lung hypoplasia. There were no significant difference between groups for asphyxia, (air leaks, respiratory distress syndrome, intracranial bleeding or bronchopulmonary dysplasia. Neonatal infection were more frequent in group (1) (4 of 14, 28.6%) and group (2) (7 of 28, 25%) when compared with group (3) (2 of 39 = 5%). Within groups (1) & (2) rupture of membranes were not very prolonged in neonates with infection (median, 9.7 days) compared with the neonates without infection (with a median, 9.6 days). They concluded that VPPROM is not complicated by oligohydramnios, mortality from lung hypoplasia and limb deformities were not found more frequent than in control (29) neonates of similar gestational age.

Bastide –A[53]; et al ; defined severe oligohydramnios where the largest pocket of amniotic fluid measures <1cm in its vertical dimension as determined by the USG, was studied in 113 patients in a population of 15, 431 referred as high risk patients (0.7%).

In all cases, intervention was done unless there were a recognized structural anomaly or extreme prematurity. Overall perinatal mortality was 132.7/1000, and the incidence in major anomaly was 13.3%. The corrected perinatal mortality rate was 17.7/1000 with intervention, a rate that was insignificant from that the rate observed in entire population. These findings were interpreted to emphasize that severe oligohydramnios in structurally normal (30) fetus remains an indication for deliver.

Van-Dongen-PW[(54)]; et al did a study retrospectively to detect the frequency of lethal lung hypoplasia in 48 cases with prolonged rupture of membranes for > 7 days before 34 weeks' gestation. Fourteen infants died amounting to 29%, but only four deaths in 8.3% was died due to lung hypoplasia. Three infants with lung hypoplasia had their membranes ruptured before 20 weeks gestation and showed presence of a persistent oligohydramnios, one with rupture at 26 weeks. The duration of rupture of membranes nor the gestational age at the time of rupture showed no influence in the occurrence of lung hypoplasia.

Schrimmer-DB[(55)]; et al studied prophylactic amnioinfusion as one of the treatment of oligohydramnios in a randomized study with a sample of 305 patients with oligohydramnios in labour, 175 patients underwent amnioinfusion and the remainder served as controls. Amniotic fluid was titered to AFI >10 cm in treatment group. Patients who received amnioinfusion had significantly less operative interference for fetal distress (P =0.0001) and few C/S (P =0.0001). Umbilical artery PH was also increased (P = 0.0001). Rates of amnionitis and endometritis were insignificant in infused patients and

controls.Though the hospital stay was markedly decreased (P= 0.002) in the treatment group, their data had support of earlier reports that amnioinfusion serves as technique for reducing intrapartum morbidity for both fetus and the mother (32).

Strong TH[56], et al performed amnioinfusion in a randomized trial of sixty women in latent phase with oligohydramnios by AFI < 5cm. All fetuses were at 37 weeks gestation, having a normal baseline fetal heart rate variability and no clinically significant fetal heart rate decelerations. The amnioinfusion group (n=30) were maintained at AFI level > 8.0 cm through the labor .In the group who received amnioinfusion, passage of meconium was exhibited in lower rates(p=0.04), operative delivery for fetal distress (P=0.002)and end stage bradycardia (P=0.05) occurred. Significant high umbilical arterial blood PH values was noticed in infusion group (P=0.02). They came to a conclusion that prophylactic intrapartum amnioinfusion serves as a valuable technique in reduction of morbidity.

MATERIALS AND METHODS

Present study was conducted at Chengalpattu Medical College in Department of Obstetrics and Gynaecology over period from 2015 to 2016. 200 patients in third trimester attending our hospital with evidence of Oligohydramnios were selected after satisfying inclusion and exclusion criteria and studied prospectively.

INCLUSION CRITERIA:

1) AFI less than or equal to 5.

2) Single live intrauterine gestation with cephalic presentation.

3) 37 completed weeks of gestation.

4) Intact membranes.

EXCLUSION CRITERIA:

1) Gestational age less than 37 completed weeks.

2) AFI more than 5.

3)Post term.

4)Associated fetal malformations.

5) Malpresentation and multiple gestation.

6))Ruptured membranes Malpresentation and multiple gestation.

7)High risk pregnancy

a)Placental insufficiency

- Hypertension
- Preeclampsia
- Diabetes
- Chronic renal disease
- Hypovolemia
- Connective tissue disorders

b)Abruption

c)Prostaglandin synthestase inhibitors therapy.

d)Angiotensin converting enzyme inhibitors therapy.

8)Uterine scar due to Previous LSCS,myomectomy,hysterotomy.

Study was conducted in order to observe outcome of labour in the form of maternal and perinatal outcome. After collecting a detailed history, complete examination was done. All required investigations performed with respect to patients condition. Oligohydramnios is confirmed by measuring Amniotic Fluid Index by USG. Routine management in form of rest, oral and intravenous hydration, left lateral position and control of etiological factor was done if present. Fetal surveillance was done by means of modified Biophysical profile and USG . Decision of delivery by induction or elective or emergency LSCS was done as indicated Some patients who were already in labour were

allowed to go in spontaneous labour. Cases were than thoroughly studied to observe maternal and perinatal outcome.

OBSERVATION AND RESULTS

A total of 200 women attending to our institution got admitted and studied in the study period.

Table 1 : AGE DISTRIBUTION

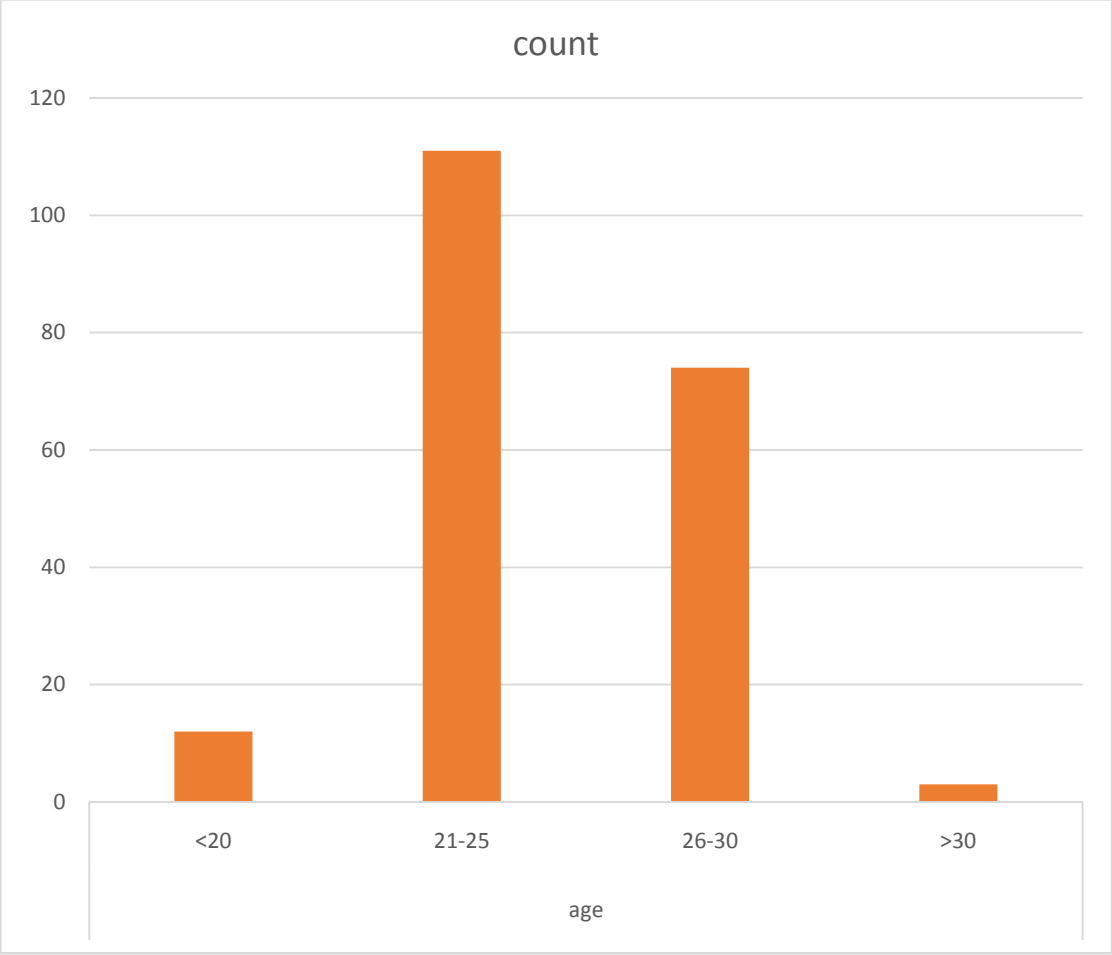


Table 2 : MATERNAL OUTCOME OF LABOUR WITH RESPECT TO AGE

		age group				Total
		<20	>30	21-25	26-30	
MOD	Count	5	1	37	29	72
	% within age group	41.7%	33.3%	33.3%	39.2%	36.0%
LSCS	Count	7	2	74	45	128
	% within age group	58.3%	66.7%	66.7%	60.8%	64.0%
Total	Count	12	3	111	74	200

% within age group	**100.0%**	**100.0%**	**100.0%**	**100.0%**	**100.0%**

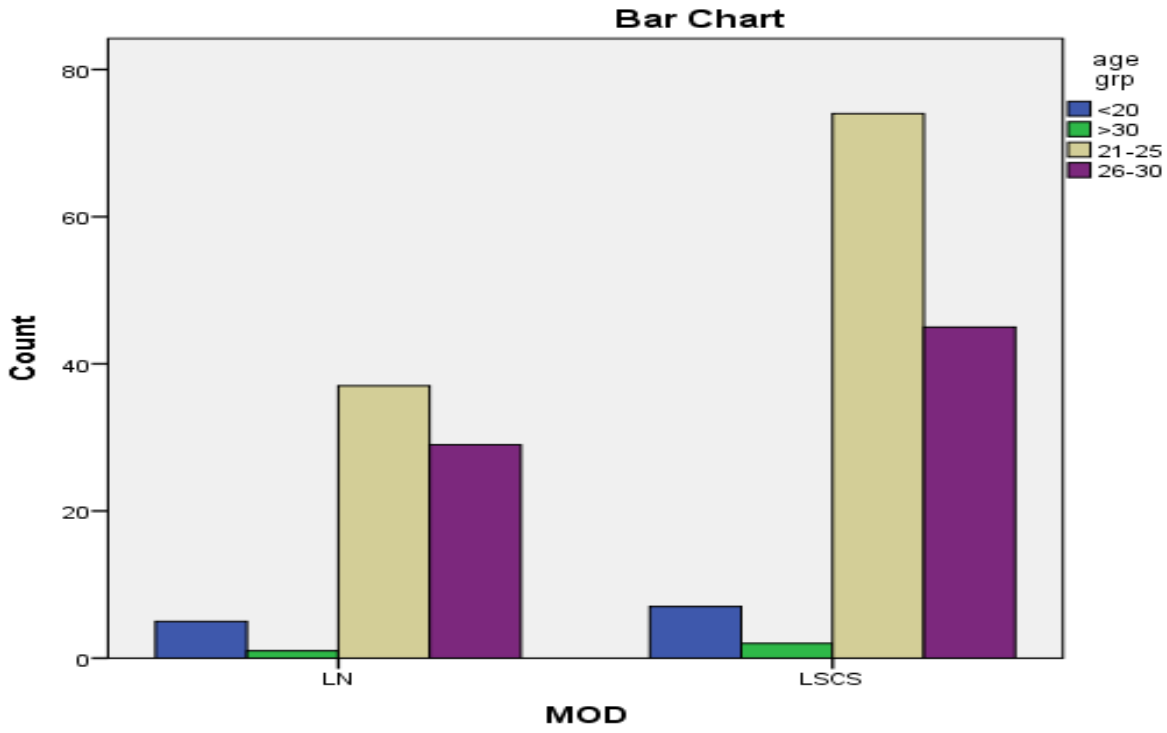
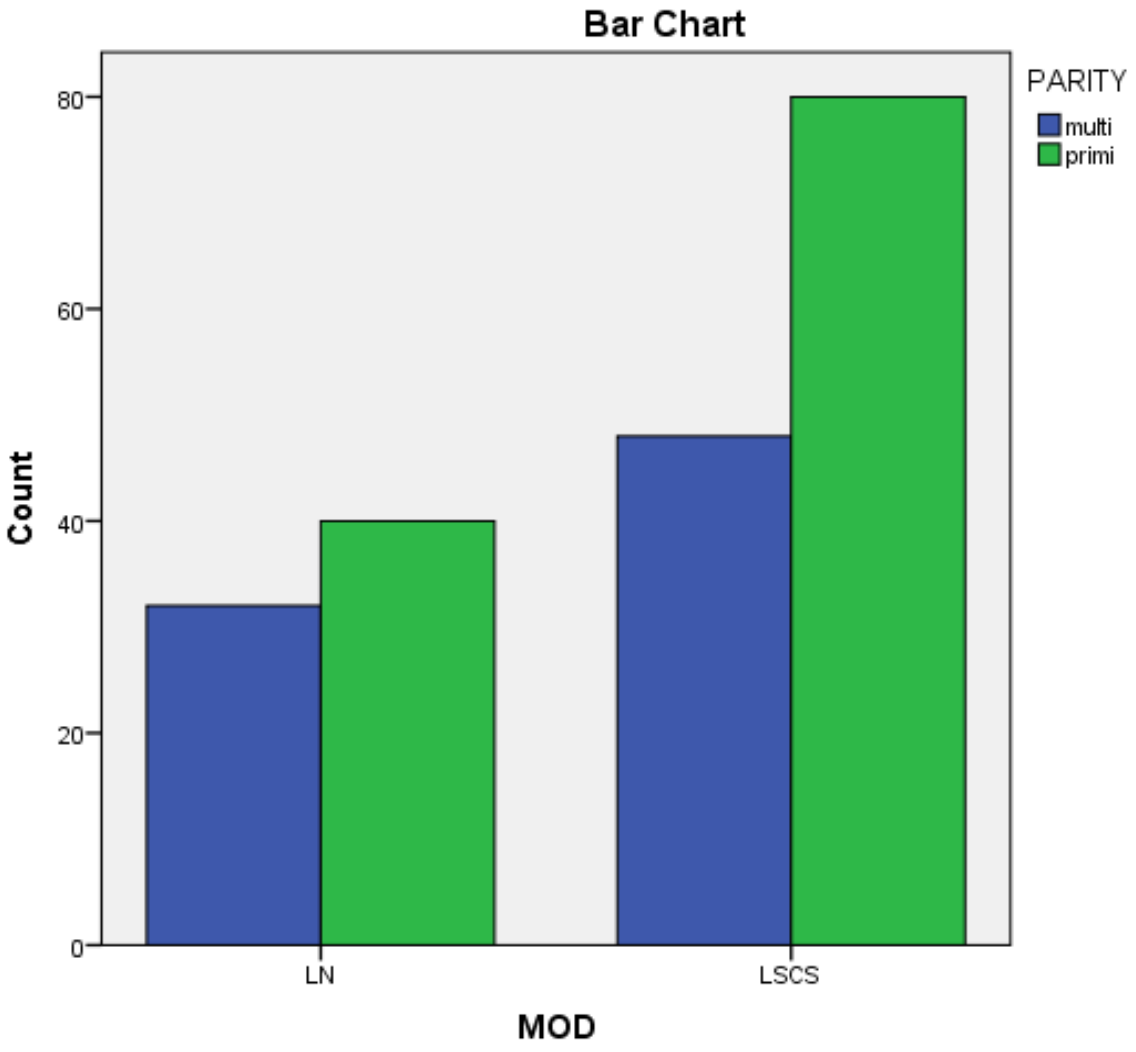


Table 1and 2 shows the distribution of study population according to age. Maximum patients belonged to 21-25 years. Rate of cesarean was found to be highest in the age group of 21-25 years .

Table 3 : PARITY DISTRIBUTION AND MATERNAL OUTCOME OF LABOUR

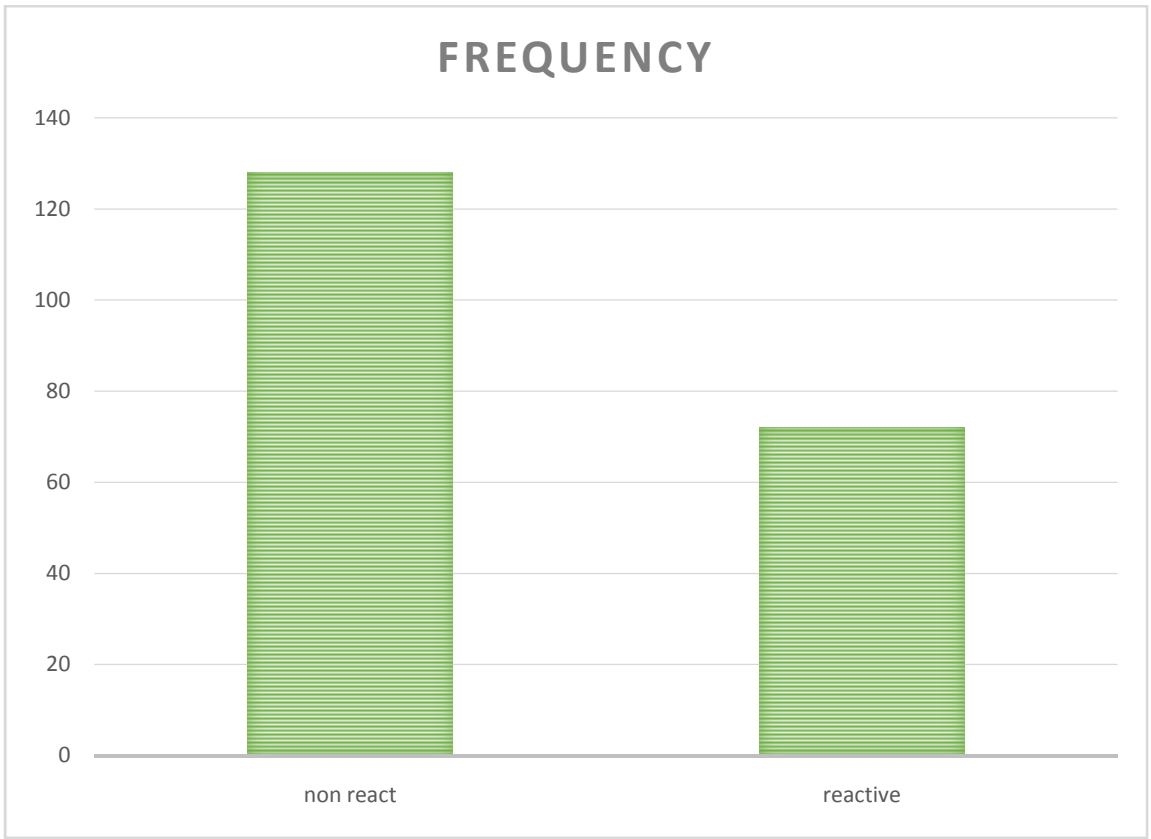
Out of 200 patients, 120 were primi and 80 were multiparous women. Operative delivery were more in primi (66.7%) compared to multipara (60 %).



PARITY	LABOUR NATURAL	LSCS	TOTAL
PRIMI	40 (33.3%)	80 (66.7%)	120
MULTI	32 (40%)	48 (60%)	80

Table 4: CTG

CTG	NUMBER	PERCENTAGE
REACTIVE	72	36
NON REACTIVE	128	64

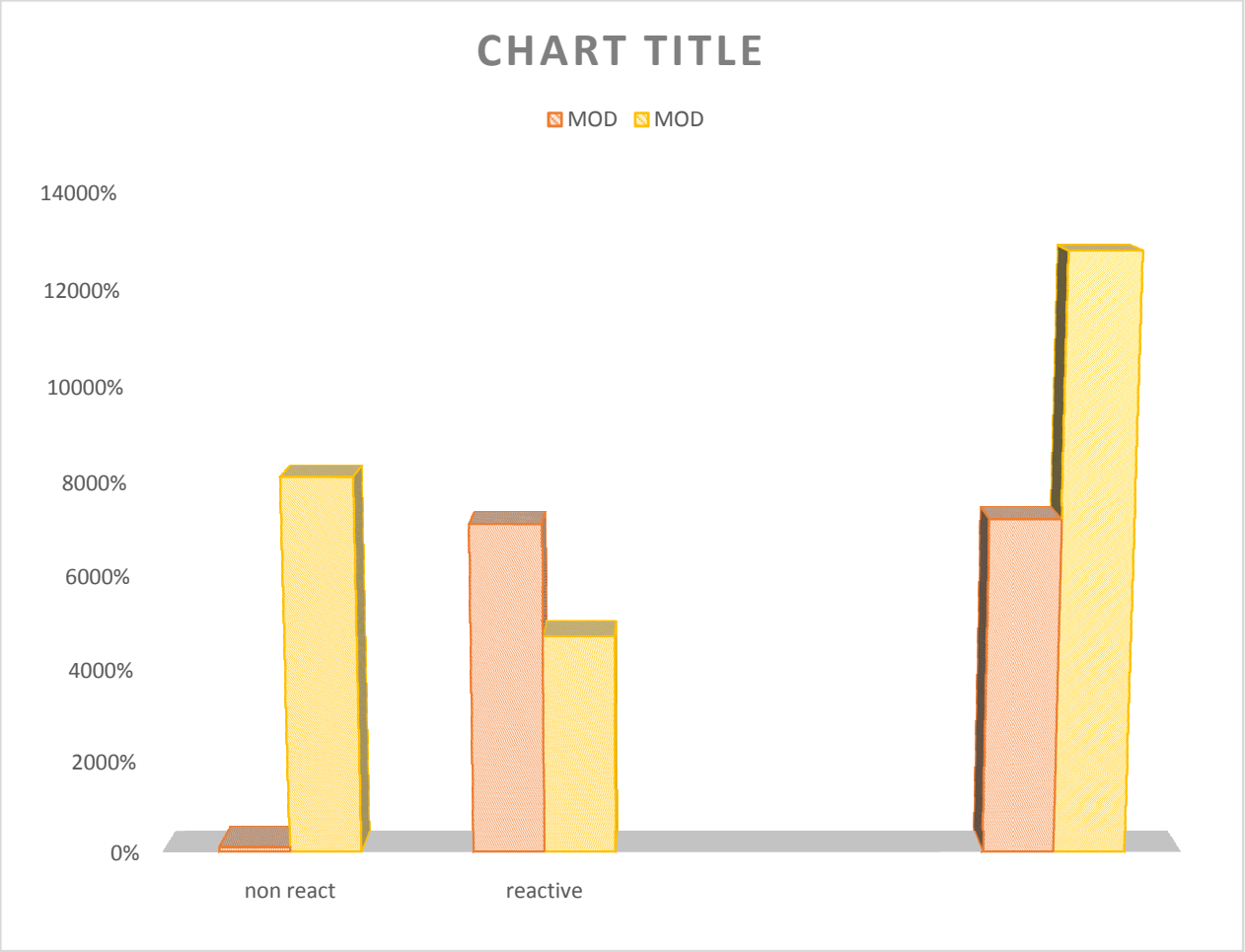


On observation, 128 (64%) out of 200 patients had non reactive CTG and only 72(36%) had reactive CTG. Operative morbidity were more in non reactive CTG group.

Table 5: MODE OF DELIVERY WITH RESPECT TO CTG

		MOD	
		LN	LSCS
REACTIVE	non react	1	81
	reactive	71	47
Total		72	128

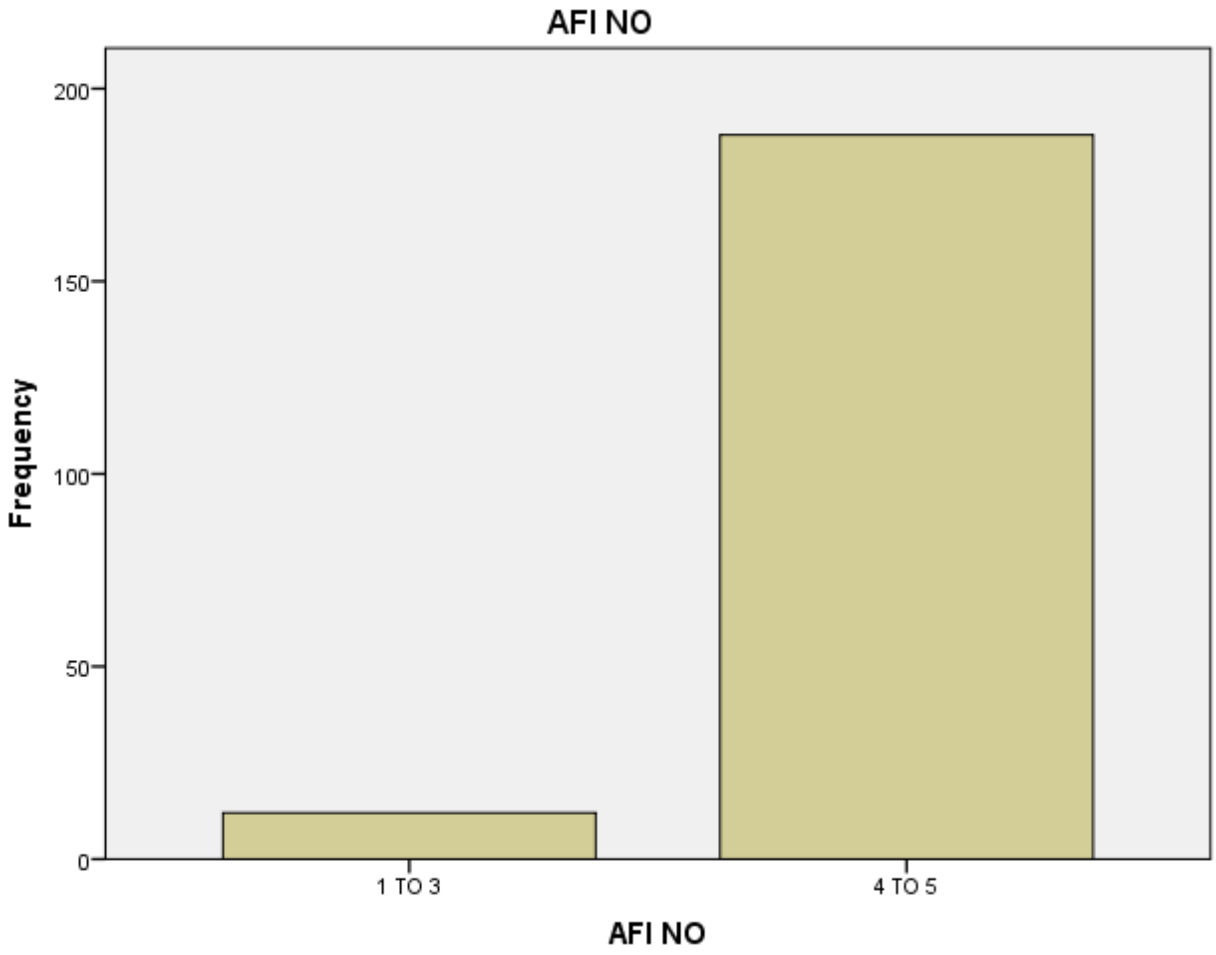
P value :0.001



This table shows that the rate of cesarean section was higher among non reactive CTG group . Its p value is (0.001) and it is statistically significant.

Table 6: DISTRIBUTION BY AFI(USG)

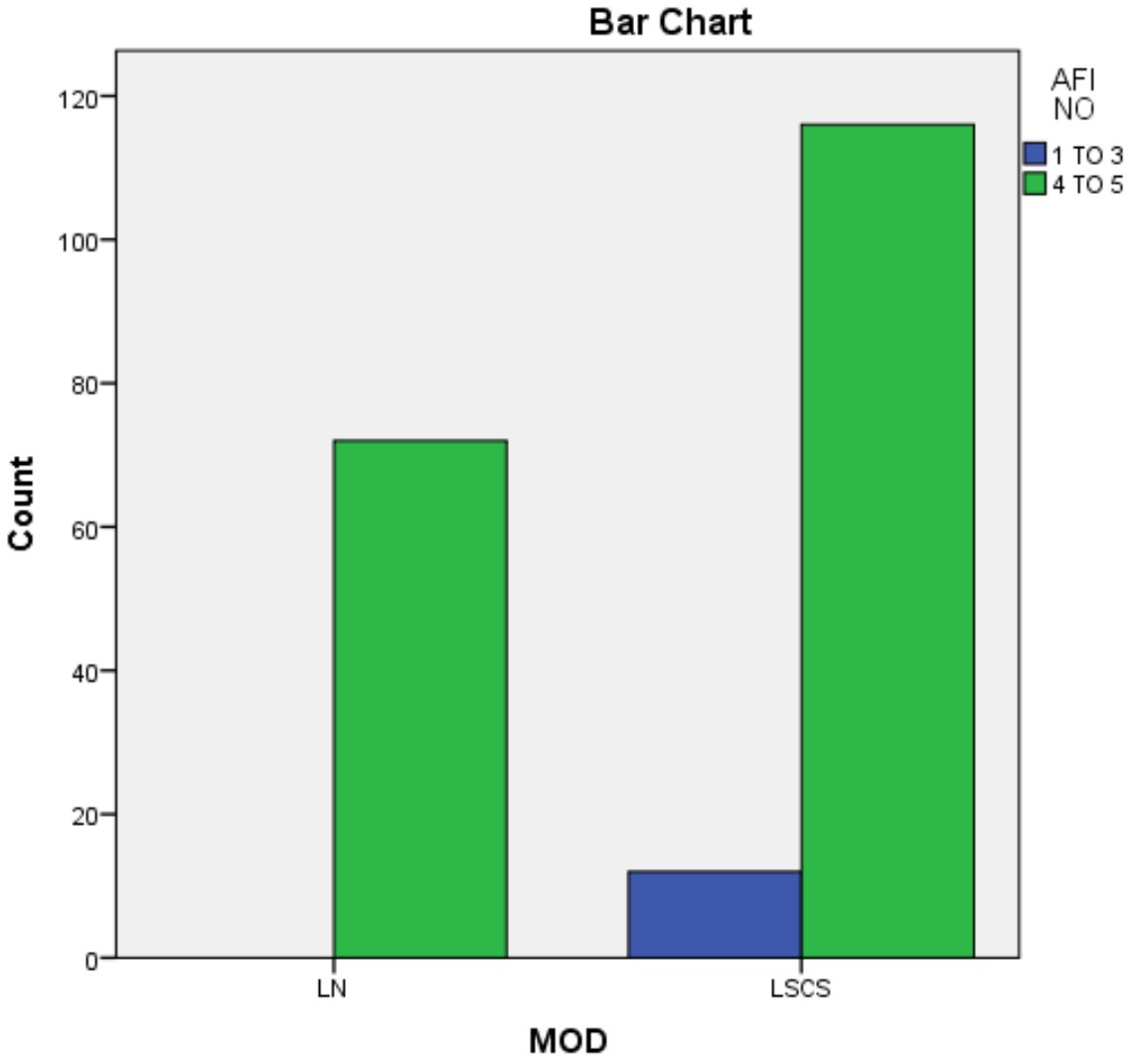
AFI	FREQUENCY	PERCENT
1 TO 3	12	6
4 TO 5	188	94
TOTAL	**100**	



This table shows that 94 % of the study population had an Amniotic fluid index of 4 to 5 cm and 6 % with AFI 1 to 3 cm.

Table 7: MODE OF DELIVERY WITH RESPECT TO AFI

			AFI NO		Total
			1 TO 3	4 TO 5	
MOD	LN	Count	0	72	72
		% within MOD	0.0%	100.0%	100.0%
		% within AFI NO	0.0%	38.3%	36.0%
	LSCS	Count	12	116	128
		% within MOD	9.4%	90.6%	100.0%
		% within AFI NO	100.0%	61.7%	64.0%
Total		Count	12	188	200
		% within MOD	6.0%	94.0%	100.0%
		% within AFI NO	100.0%	100.0%	100.0%



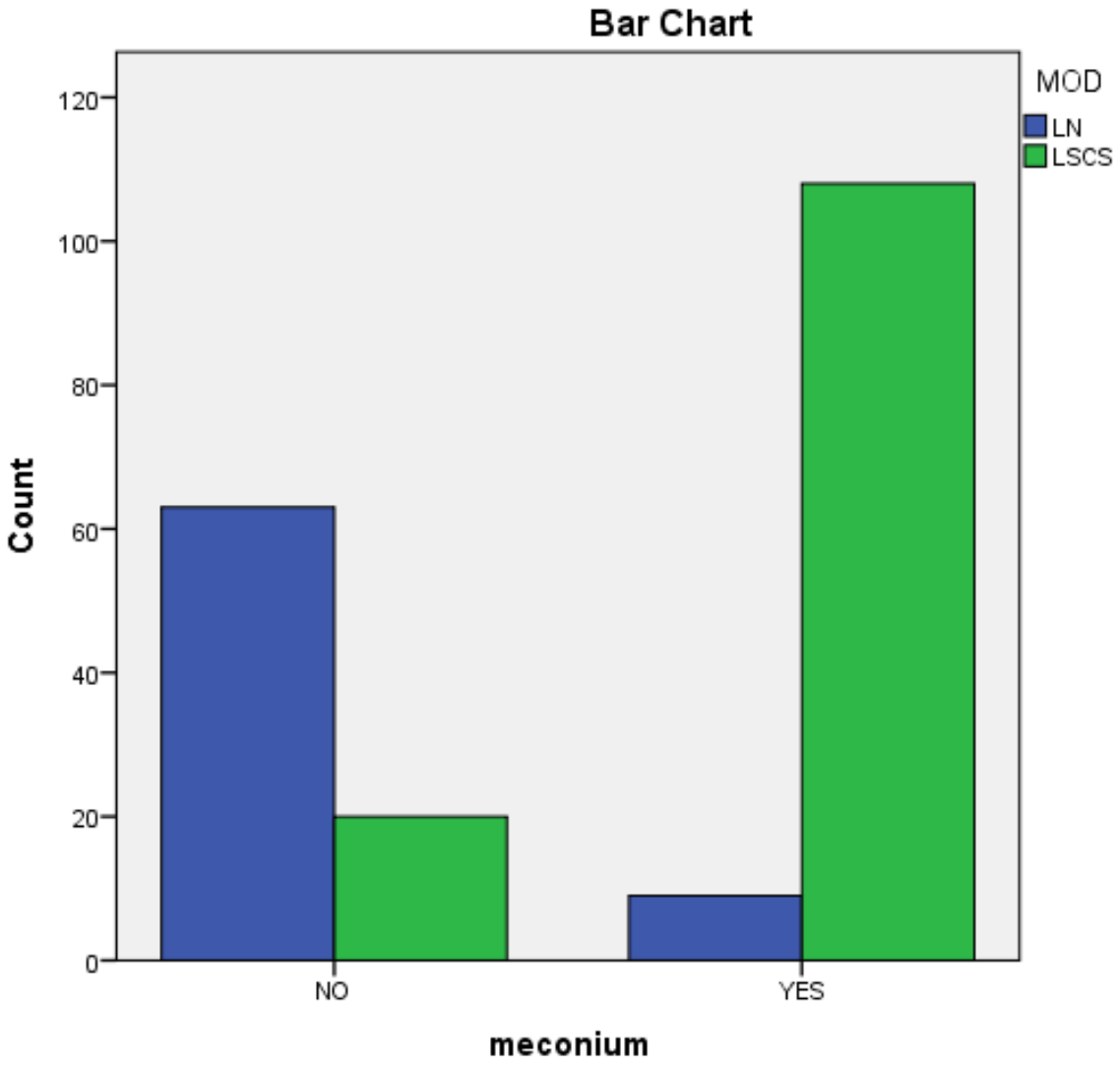
This table shows that there was nil Labour natural in the subset of AFI 1 to 2 in the study population and Caesarean section rate was higher in this group.

Table 8: MODE OF DELIVERY WITH RESPECT TO MECONIUM

			MOD		**Total**
			LN	**LSCS**	
meconium	NO	Count	63	20	83
		% of Total	31.5%	10.0%	41.5%
	YES	Count	9	108	117
		% of Total	4.5%	54.0%	58.5%
Total		**Count**	**72**	**128**	**200**

	% of Total	**36.0%**	**64.0%**	**100.0%**

P value: 0.001



Overall meconium stained amniotic fluid occurred in 58.5% of the study population. Of

which 36% had labour natural and 64% had cesarean section.It was statistically significant (p value <0.001).

Table 9: DISTRIBUTION OF STUDY POPULATION ACCORDING TO BIRTH WEIGHT

		Frequency	Percent
Valid	<2500	16	8.0
	>2500	184	92.0
	Total	200	100.0

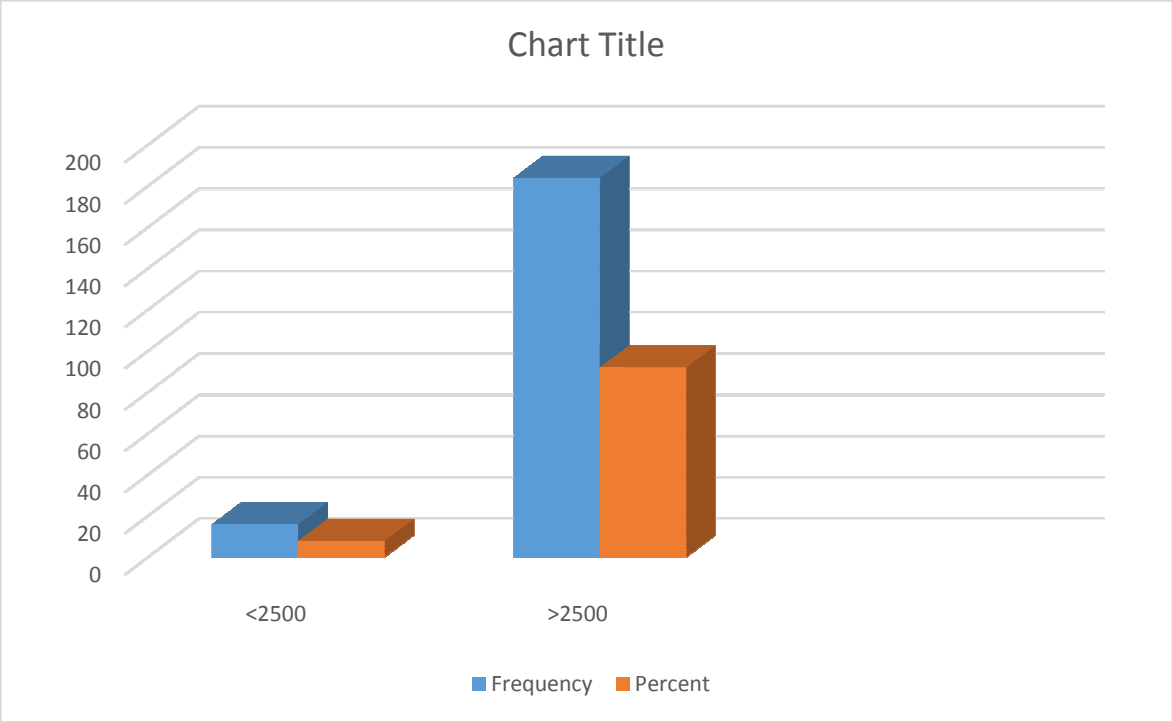
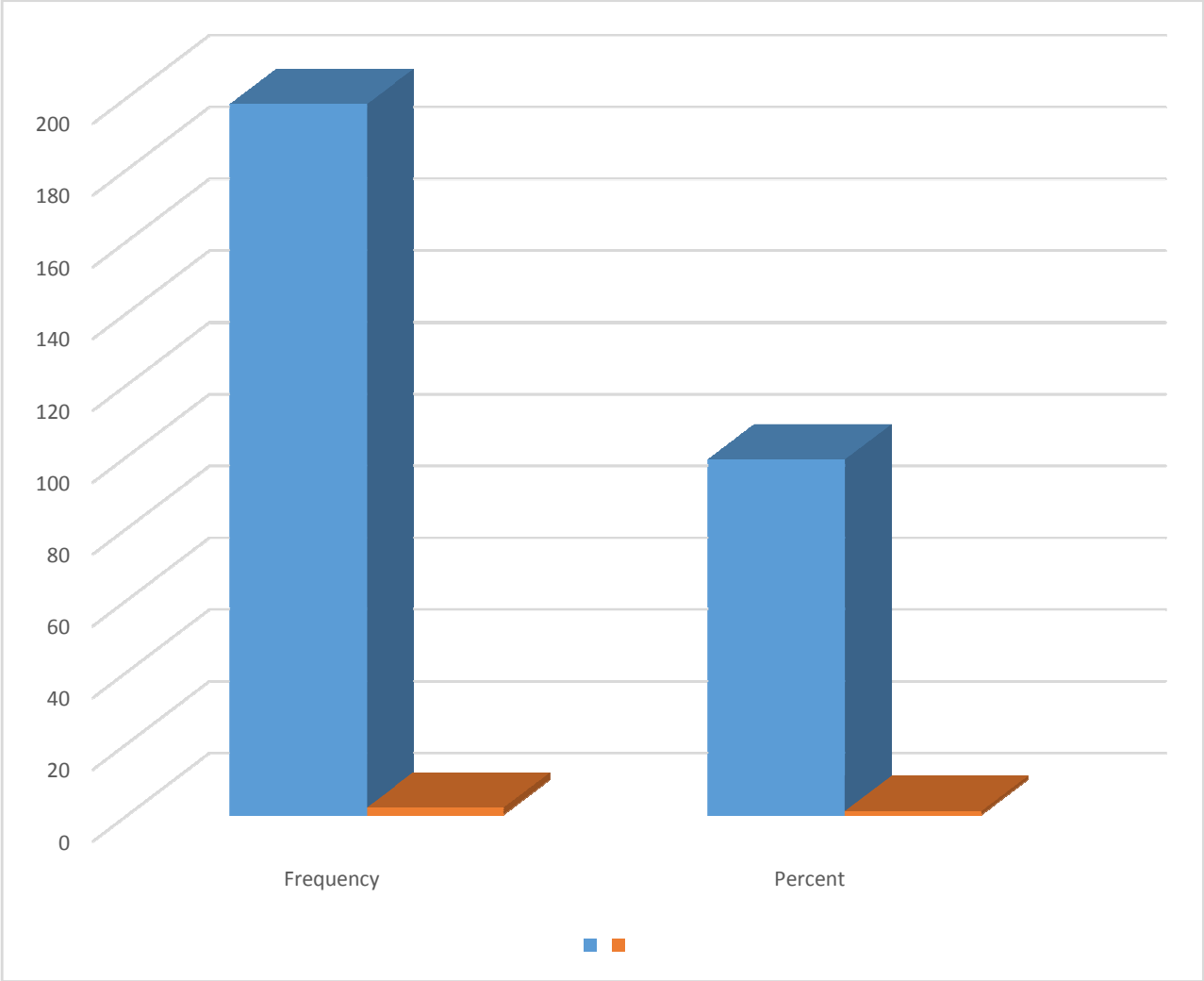


Table 10: INCIDENCE OF STILL BIRTH

		Frequency	Percent
Valid	NO	198	99.0
	YES	2	1.0

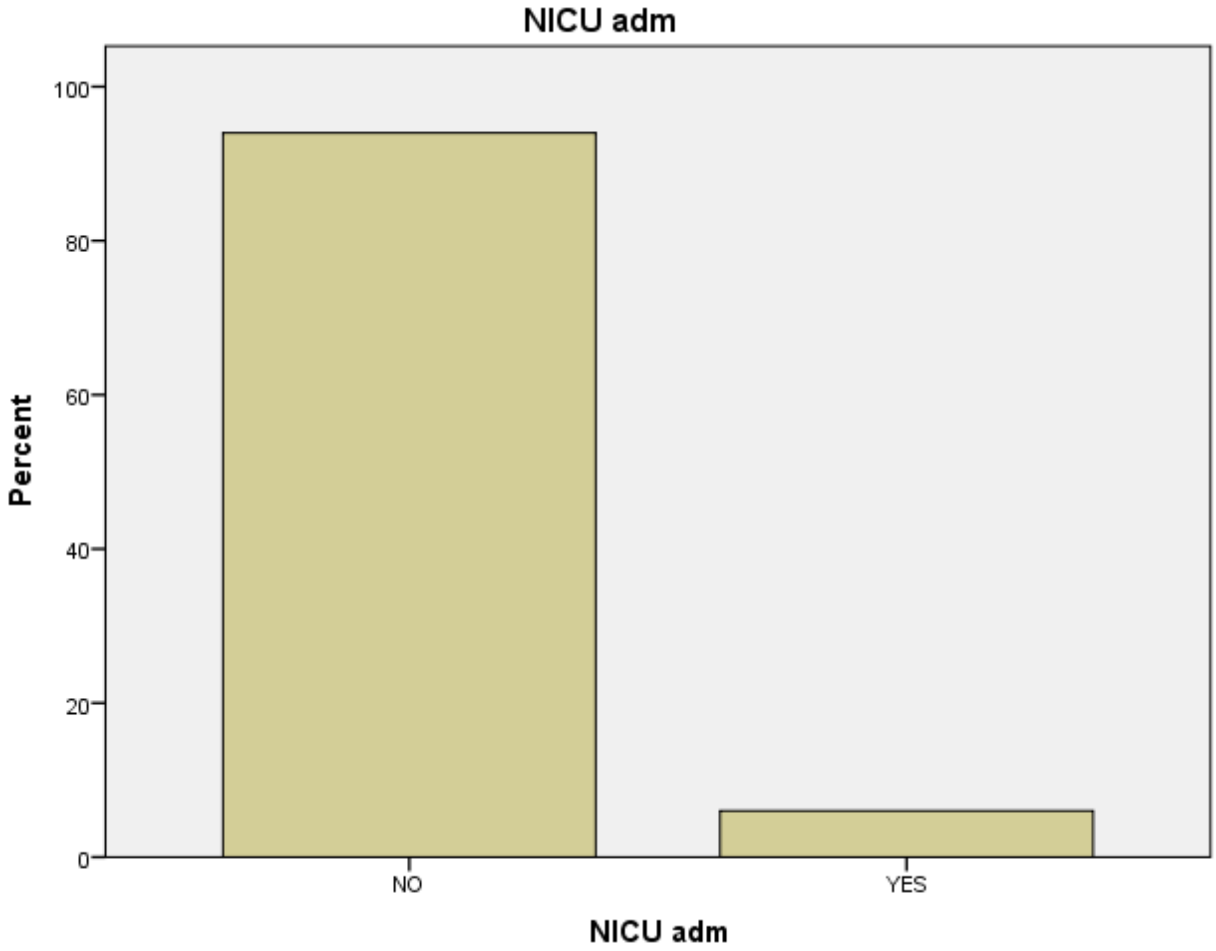
Total	**200**	**100.0**



Only 1% still birth was present in the study population.

Table 11: INCIDENCE OF NICU ADMISSION

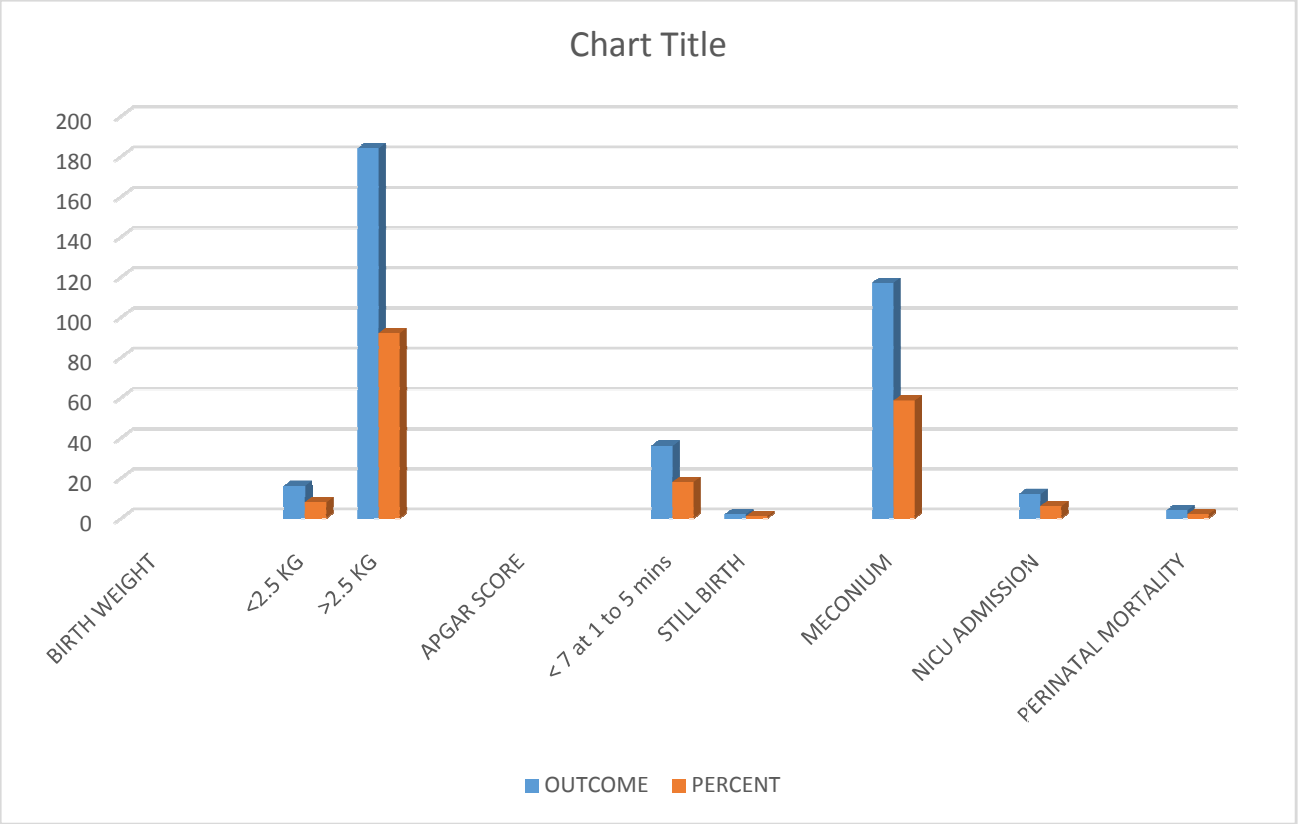
		Frequency	Percent
Valid	NO	188	94.0
	YES	12	6.0
	Total	200	100.0



This table shows that the incidence of NICU admission accounted to 6% of the study population.

Table 12 : PERINATAL OUTCOME

DISEASE	NUMBER	PERCENT
BIRTH WEIGHT		
<2.5 KG	16	8
>2.5 KG	184	92
APGAR SCORE		
<7 at 1 to 5 mins	36	18
STILL BIRTH	2	1
MECONIUM	117	58.5
NICU ADMISSION	12	6
PERINATAL MORTALITY	4	2



The above table shows the overall perinatal outcome with respect to birth weight (<2.5 kg-8% and >2.5 kg-92%), Apgar score (<7 at 1-5 mins:18%)., still birth (1%)., meconium (58.5%)., NICU admission(6%) and perinatal mortality (2%).

DISCUSSION

It is a known fact that severe oligohydramnios is associated with adverse perinatal outcome. On other hand, it still remains a poor predictor in detecting adverse outcome. But often oligohydramnios is used as an indication for operative delivery. Hence assessing amniotic fluid volume antenatally is essential in determining high and low risk groups.

In our study maximum number of women (n= 200) were in the age group of 21 to 25 years (n=111). Of which highest percentage of women (120) were primipara and 80 were multiparous. This was partially evident with that of Jagatia et al.,where they reported that incidence of oligohydramnios was more in primipara which is compatible with the study of Petrozella et al and Jandial et al.,

The present study revealed that, cesarean section rate was higher among non reactive CTG group. This goes with the study by Charu Jandial whish stated a non-reactive CTG + AFI< 5 cm indicated fetal jeopardy according to revised Biophysical profile scoring by Clerk e al,.The fetal jeopardy reflected an increased operative interference in this study.

This study showed high incidence of meconium stained liquor(58.5%) in oligohydramnios patient with AFI<5 cm. Meconium staining is an indicator of fetal distress and has its own complication in newborn. This finding is matching with many

research studies during various studies of Chandra et al., Sriya et al., Chate et al., and Ghike et al. In addition Jandial et al.,observed meconium stained liquor in 48% of women with oligohydramnios. Also, Youssef et al., found it in 40% of oligohydramnios group. This suggests that there is high incidence of meconium staining in oligohydramnios group.

The rate of NICU admission was found to be 6 % , of which perinatal mortality was 2%. In the study done by Wolff-F, 49.17% of newborns were referred to nearby pediatric hospital immediately following delivery. This difference may be due to the facilities encountered in the hospital set up.

LIMITATIONS OF THE STUDY

The limitations of study includes the following:

1. Only 200 cases were available during the study period which exactly satisfed inclusion and exclusion criteria which is less compared to other studies.

2. The diagnosis of fetal distress was made depending on FHR tracings. However, the fetal acidosis was not proved by fetal scalp blood sampling or other methods because of non-availability

3. The use of backup surveillance methods like scalp blood sampling and acoustic stimulation and amnioinfusion would have altered the outcome.

RECOMMENDATIONS

Determination of AFI should be used as an adjunct to other fetal surveillance methods.

It helps to identify those infants at risk of having poor perinatal outcome

It remains as a valuable screening test for predicting fetal distress in labour requiring cesarean section in oligohydramnios patient.

Continuous antepartum and intrapartum monitoring are mandatory for every women diagnosed with oligohydramnios to reduce the maternal and neonatal risks associated with oligohydramnios.

The suggested plan of action:

Development of health instruction brochure to raise the pregnant women′s awareness regarding oligohydramnios and its management.

2. The brochure should include knowledge related to the definition of oligohydramnios and its maternal and fetal outcome.

3. The brochure should be distributed to the pregnant women during their scheduled antenatal visit. 4. Meeting with the antenatal clinic nurses to encourage them to educate pregnant women about oligohydramnios, its maternal and fetal outcome and its plan of action.

5. Referral of the women who had oligohydramnios to the proper channels for further examination.

CONCLUSION

Oligohydramnios is commonly encountered and it necessitates extensive fetal surveillance and perfect antepartum and intrapartum care. Amniotic fluid volume is an important predictor of fetal level of tolerance during labour and its decrease is associated with increased risk of fetal distress and meconium staining of fluid. Due to unforeseen intrapartum complication and high incidence of perinatal mortality and morbidity, cesarean section rates are on the rise, but the decision between cesarean section and vaginal delivery must be well balanced so that unnecessary maternal morbidity could be prevented and timely decision can reduce perinatal mortality and morbidity.

To conclude, an AFI measurement of less than 5 cm detected after 37 completed weeks of gestation with a low risk pregnancy is found to be an indicator of adverse pregnancy outcome with higher cesarean section rate.

In our study, in presence of AFI <5 cm, the occurrence of non reactive CTG, incidence of meconium stained liquor and rate of LSCS are high.

Hence AFI assessment serves as an important tool and remains as an efective screening test in predicting fetal distress in labour that requires cesarean section.

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LIST OF ABBREVIATIONS

IUGR	**:**	**Intrauterine growth restriction**
HTN	**:**	**Hypertension**
PPROM	**:**	**Premature prelabour rupture of membranes**
APH	**:**	**Antepartum hemorrhage**
CTG	**:**	**Cardiotocography**
C/S	**:**	**Cesarean section**
EM/C/S	**:**	**Emergency C/S**
EL/C/S	**:**	**Elective C/S**
USS	**:**	**Ultrasound scan**
VD	**:**	**Vaginal delivery**
SVD	**:**	**Spontanous VD**
NICU	**:**	**Neonatal intensive care unit**
PIH	**:**	**Pregnancy induced hypertension**
Kg	**:**	**Kilogram**
Wt	**:**	**Weight**
GA	**:**	**Gestational age**
FSB	**:**	**Fresh stillbirth**
MSB	**:**	**Macerated stillbirth**
AFI	**:**	**Amniotic fluid index**
AFV	**:**	**Amniotic fluid volume**
ACE	**:**	**Angiotensin - converting enzyme**
Fig	**:**	**Figure**
IUFD	**:**	**Intrauterine fetal death**
MSAFP	**:**	**Maternal serum alpha-feto protein**
PG	**:**	**Phosphatidyl glycerol**

PROFORMA

Name: **Husband name:**

Age: **Case no:**

IP no.: **Occupation:**

Obs code: **Date of admission:**

LMP : **Date of delivery:**

EDD: **Socio economic status:**

Period of gestation:

Chief complaints:

History of presenting illness:

Menstrual history:

Marital history :

Obstetric history:

Past:

Present:

I trimester

II trimester

III trimester

Past history:

H/O similar illness

H/O hypertension

H/O diabetes/epilepsy/ thyroid disorder/ heart disease/ asthma

H/O renal or liver disease/ auto immune disorder

H/O chronic drug intake/ trauma

Family history:

GENERAL EXAMINATION:

Height: **Breast:**

Weight: **Thyroid:**

PR: **Spine:**

BP: **Pedal Edema:**

SYSTEMIC EXAMINATION:

CVS:

RS:

PA:

Inspection:

Palpation:

Auscultation:

Investigations:

Hb%

Blood grouping typing:

Blood sugar:

Serum urea:

Serum creatinine:

LFT:

Urine albumin:

Urine sugar:

Urine microscopic exam.:

USG OBS:

Singleton:

Viability:

GA:

Placenta:

Presentation:

AFI:

EFW:

Congenital Anomalies:

Maternal outcome:

Mode of delivery:

Vaginal:

Assisted vaginal:

LSCS: Ind:

Intra natal course and complication if any:

Fetal outcome:

Live birth/ Still birth/ IUD:

Term/ Preterm:

Male/ Female:

APGAR :

Weight of the baby:

HC of the baby:

IUGR:

Ponderal index:

Neonatal complication if any:

Placenta:

Colour of the liquor:

INFORMATION SHEET

- We are conducting a study on **"**Outcome of pregnancy and perinatal outcome in Amniotic Fluid Index less than or equal to 5 in term low risk pregnancy**"**
- The purpose of the study is to find whether antepartum oligohydramnios is associated with adverse perinatal outcome and to determine Obstetric outcome in low risk term patients with AFI less than or equal to 5.
- The privacy of the patient in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research , no personally identifiable information will be shared.
- Taking part in the study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time. Your decision will not result in any loss of benefits to which you are otherwise entitled.
- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator **Signature of patient /guardian**

Date :

NO	NAME	AGE	PARITY	NUMBER	GA	AFI NO	AFI	REACTIVE	meconium	FHR	IUGR	INDICATION	MOD	STILL BIRT	FETAL DIS	B.WT	APGAR1	APGAR 5	NICU adm	perinatmo
1	tamil	22	multi	3	39	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.5	NORMAL	NORMAL	NO	NO
2	Rosi	27	multi	2	38	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	3	NORMAL	NORMAL	NO	NO
3	subadra	26	primi	3	39	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.9	NORMAL	NORMAL	NO	NO
4	anandi	20	primi	2	38	4 TO 5	4	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	3.2	NORMAL	NORMAL	NO	NO
5	hema	24	primi	1	37	4 TO 5	4	reactive	NO	NORMAL	NO		LN	NO	NO	3	NORMAL	NORMAL	NO	NO
6	madhavi	25	multi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.8	NORMAL	NORMAL	NO	NO
7	princy	23	primi	2	38	1 TO 3	3	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
8	parvathy	25	primi	2	38	4 TO 5	4	reactive	NO	NORMAL	NO		LN	NO	NO	2.5	NORMAL	NORMAL	NO	NO
9	vivita	23	primi	2	38	4 TO 5	5	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.5	NORMAL	NORMAL	NO	NO
10	saindhavi	28	primi	1	37	4 TO 5	5	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.5	NORMAL	NORMAL	NO	NO
11	Sudha	25	primi	2	38	1 TO 3	3	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	3.1	NORMAL	NORMAL	NO	NO
12	Sudha	23	multi	1	37	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	3.2	ABNORM	ABNORM	YES	NO
13	amudham	26	multi	2	38	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	3.1	NORMAL	NORMAL	NO	NO
14	radha	26	primi	1	37	4 TO 5	5	reactive	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.5	ABNORM	NORMAL	NO	NO
15	padma	22	primi	1	37	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	3	NORMAL	NORMAL	NO	NO
16	jeeva	28	primi	2	38	4 TO 5	5	non react	NO	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
17	shamala	21	multi	3	39	4 TO 5	5	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.6	ABNORM	NORMAL	NO	NO
18	supriya	22	primi	4	40	4 TO 5	5	reactive	YES	NORMAL	NO		LN	NO	NO	2.7	NORMAL	NORMAL	NO	NO
19	devi	20	multi	1	37	4 TO 5	5	reactive	YES	NORMAL	NO		LN	NO	NO	2.5	NORMAL	NORMAL	NO	NO
20	Evangeline	26	multi	3	39	4 TO 5	5	reactive	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	4	NORMAL	NORMAL	NO	NO
21	barathy	26	primi	4	40	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.6	NORMAL	NORMAL	NO	NO
22	devaki	25	primi	2	38	4 TO 5	4	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	3.1	NORMAL	NORMAL	NO	NO
23	vanathi	24	primi	2	38	4 TO 5	5	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
24	suganya	23	primi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.8	NORMAL	NORMAL	NO	NO
25	subha	24	primi	3	39	1 TO 3	2	non react	NO	ABNORM	NO	FETAL DISTRESS	LSCS	YES	YES	2	ABNORM	ABNORM	NO	YES
26	malini	21	multi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.6	NORMAL	NORMAL	NO	NO
27	tamil	22	primi	2	38	4 TO 5	4	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
28	niranjana	26	multi	1	37	4 TO 5	4	reactive	NO	NORMAL	NO		LN	NO	NO	2.7	NORMAL	NORMAL	NO	NO
29	ragavi	22	primi	3	39	4 TO 5	5	reactive	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.4	NORMAL	NORMAL	NO	NO
30	Ajidha	29	primi	2	38	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.4	NORMAL	NORMAL	NO	NO
31	lakshmi	23	primi	3	39	4 TO 5	4	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.8	NORMAL	NORMAL	NO	NO
32	naga priya	25	primi	1	37	4 TO 5	4	non react	NO	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
33	shamala	21	primi	3	39	4 TO 5	4	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.6	NORMAL	ABNORM	YES	NO
34	Indumathi	26	primi	1	37	4 TO 5	4	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.4	NORMAL	NORMAL	NO	NO
35	sangavi	27	primi	2	38	4 TO 5	4	reactive	NO	NORMAL	NO		LN	NO	NO	2.9	NORMAL	NORMAL	NO	NO
36	delphin	24	primi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.9	NORMAL	NORMAL	NO	NO
37	swetha	26	primi	3	39	4 TO 5	4	non react	NO	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.7	NORMAL	NORMAL	NO	NO
38	Kayani	20	primi	2	38	4 TO 5	5	reactive	YES	ABNORM	NO		LN	NO	NO	3	ABNORM	NORMAL	YES	NO

39	shiva	27	multi	2	38	4 TO 5	4	non react	NO	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.5	NORMAL	NORMAL	NO	NO
40	shivam	21	primi	2	38	4 TO 5	4	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.5	NORMAL	NORMAL	NO	NO
41	sunitha	25	primi	1	37	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.8	NORMAL	NORMAL	NO	NO
42	sanjeevana	24	primi	2	38	4 TO 5	5	non react	NO	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.8	NORMAL	NORMAL	NO	NO
43	vedha	28	multi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	3	NORMAL	NORMAL	NO	NO
44	janaki	26	multi	3	39	4 TO 5	5	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.3	NORMAL	NORMAL	NO	NO
45	pallavi	24	multi	2	38	4 TO 5	4	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	3.4	NORMAL	NORMAL	NO	NO
46	moulika	22	primi	1	37	4 TO 5	5	reactive	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	3.1	NORMAL	NORMAL	NO	NO
47	vinitha	23	multi	2	38	4 TO 5	5	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.7	NORMAL	NORMAL	NO	NO
48	samanthi	23	multi	2	38	4 TO 5	4	reactive	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	3.2	NORMAL	NORMAL	NO	NO
49	gaythri	29	multi	1	37	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.9	NORMAL	NORMAL	NO	NO
50	Rani	24	multi	2	38	4 TO 5	4	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.8	NORMAL	NORMAL	NO	NO
51	nandhini	21	multi	3	39	4 TO 5	5	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.9	NORMAL	NORMAL	NO	NO
52	chandra	22	primi	2	38	1 TO 3	3	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.8	NORMAL	NORMAL	NO	NO
53	saroja	21	multi	3	39	4 TO 5	4	reactive	NO	NORMAL	NO		LN	NO	NO	2.7	NORMAL	NORMAL	NO	NO
54	lavanya	27	primi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.7	NORMAL	NORMAL	NO	NO
55	sathya	19	primi	2	38	4 TO 5	5	reactive	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	3	NORMAL	NORMAL	NO	NO
56	latha	19	multi	1	37	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.6	NORMAL	NORMAL	NO	NO
57	Ganga	25	multi	2	38	4 TO 5	5	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.9	NORMAL	NORMAL	NO	NO
58	Alagammai	28	primi	1	37	4 TO 5	5	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.5	NORMAL	NORMAL	NO	NO
59	soundarya	26	primi	2	38	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
60	kaleeshwari	21	primi	1	37	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.6	ABNORM	NORMAL	NO	NO
61	Yamuna	24	primi	2	38	4 TO 5	5	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
62	Jeya	24	primi	1	37	4 TO 5	5	reactive	YES	NORMAL	NO		LN	NO	NO	2.5	NORMAL	NORMAL	NO	NO
63	prasanna	24	multi	1	37	4 TO 5	4	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
64	sharmila	27	multi	2	38	4 TO 5	4	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.3	NORMAL	NORMAL	NO	NO
65	amudha	22	primi	3	39	4 TO 5	4	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
66	shanthi	23	primi	2	38	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.6	ABNORM	NORMAL	NO	NO
67	preethi	23	multi	3	39	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.6	NORMAL	NORMAL	NO	NO
68	girija	26	primi	2	38	4 TO 5	4	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.5	NORMAL	NORMAL	NO	NO
69	selvi	23	primi	1	37	4 TO 5	4	reactive	NO	NORMAL	NO		LN	NO	NO	2.4	NORMAL	NORMAL	NO	NO
70	sunny	27	multi	2	38	4 TO 5	4	reactive	NO	NORMAL	NO		LN	NO	NO	2.7	NORMAL	NORMAL	NO	NO
71	sundari	22	primi	4	40	4 TO 5	4	non react	NO	NORMAL	NO		LN	NO	NO	2.6	NORMAL	NORMAL	NO	NO
72	shenba	27	primi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.6	NORMAL	NORMAL	NO	NO
73	evangeline	28	primi	1	37	4 TO 5	5	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.7	ABNORM	NORMAL	NO	NO
74	kavitha	21	multi	3	39	1 TO 3	3	reactive	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.7	NORMAL	NORMAL	NO	NO
75	Tamil lniaya	24	multi	1	37	4 TO 5	4	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
76	deepa	24	primi	3	39	4 TO 5	5	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.7	NORMAL	NORMAL	NO	NO
77	jeya	26	primi	1	37	4 TO 5	4	reactive	YES	NORMAL	NO		LN	NO	NO	2.4	NORMAL	NORMAL	NO	NO
78	Punitha	28	multi	2	38	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	3	NORMAL	NORMAL	NO	NO
79	Renuka	27	multi	1	37	4 TO 5	4	reactive	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.9	NORMAL	NORMAL	NO	NO

80	nimi	26	multi	3	39	4 TO 5	5	non react	NO	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.7	NORMAL	NORMAL	NO	NO
81	sudar	25	primi	2	38	4 TO 5	5	reactive	YES	NORMAL	NO		LN	NO	NO	2.5	NORMAL	NORMAL	NO	NO
82	Pushpa	28	primi	2	38	4 TO 5	5	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	3.1	NORMAL	NORMAL	NO	NO
83	bhavya	20	primi	1	37	4 TO 5	4	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.7	NORMAL	NORMAL	NO	NO
84	leela	22	multi	1	37	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.8	NORMAL	NORMAL	NO	NO
85	shankari	21	primi	2	38	4 TO 5	4	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	3	NORMAL	NORMAL	NO	NO
86	Rani	24	primi	3	39	1 TO 3	3	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
87	janani	25	multi	2	38	1 TO 3	3	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
88	rita	23	primi	2	38	1 TO 3	3	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.9	ABNORM	NORMAL	NO	NO
89	loganayaki	26	primi	3	39	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.6	NORMAL	NORMAL	NO	NO
90	sheeba	27	primi	1	37	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.8	NORMAL	NORMAL	NO	NO
91	parvathy	25	primi	1	37	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.5	NORMAL	NORMAL	NO	NO
92	deepika	25	multi	3	39	4 TO 5	4	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.7	NORMAL	NORMAL	NO	NO
93	sita	23	multi	1	37	4 TO 5	4	reactive	NO	NORMAL	NO		LN	NO	NO	2.4	NORMAL	NORMAL	NO	NO
94	susila	26	multi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.6	NORMAL	NORMAL	NO	NO
95	indu	26	primi	2	38	4 TO 5	4	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
96	fathima	27	primi	3	39	1 TO 3	3	non react	YES	ABNORM	YES	MECONIUM	LSCS	YES	YES	1.9	ABNORM	ABNORM	NO	YES
97	venda	25	multi	1	37	4 TO 5	4	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.7	NORMAL	NORMAL	NO	NO
98	parvathy	22	multi	2	38	4 TO 5	5	non react	NO	ABNORM	NO	MECONIUM	LSCS	NO	YES	3.2	NORMAL	NORMAL	NO	NO
99	regina	26	primi	2	38	4 TO 5	4	reactive	NO	NORMAL	NO		LN	NO	NO	2.5	NORMAL	NORMAL	NO	NO
100	chandra	25	primi	2	38	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
101	varnam	19	multi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	3.1	NORMAL	NORMAL	NO	NO
102	subadra	26	primi	1	37	4 TO 5	4	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
103	mary	32	primi	1	37	4 TO 5	5	reactive	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	3.2	ABNORM	NORMAL	YES	NO
104	merlin	24	primi	1	37	4 TO 5	4	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.5	NORMAL	NORMAL	NO	NO
105	hephzibah	22	multi	3	39	4 TO 5	5	reactive	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.5	NORMAL	NORMAL	NO	NO
106	lekha	25	multi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.5	NORMAL	NORMAL	NO	NO
107	indira	27	primi	2	38	4 TO 5	4	reactive	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
108	lakshmi	27	primi	1	37	4 TO 5	5	non react	NO	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.4	NORMAL	NORMAL	NO	NO
109	namitha	26	primi	2	38	4 TO 5	5	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.8	NORMAL	NORMAL	NO	NO
110	shenbagam	22	primi	1	37	4 TO 5	5	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	3.5	NORMAL	NORMAL	NO	NO
111	parameshwari	24	multi	3	39	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.9	NORMAL	NORMAL	NO	NO
112	Jagadhambal	24	primi	1	37	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.8	NORMAL	NORMAL	NO	NO
113	saranya	26	primi	1	37	4 TO 5	4	reactive	NO	NORMAL	NO		LN	NO	NO	2.6	NORMAL	NORMAL	NO	NO
114	sukanya	24	primi	4	40	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	3.1	NORMAL	NORMAL	NO	NO
115	bharathy	20	multi	1	37	4 TO 5	5	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	3.5	ABNORM	NORMAL	YES	NO
116	nethra	26	primi	2	38	4 TO 5	4	reactive	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
117	Hannah	25	multi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.8	NORMAL	NORMAL	NO	NO
118	anitha	25	multi	1	37	4 TO 5	5	non react	NO	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
119	menaga	22	primi	1	37	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.6	NORMAL	NORMAL	NO	NO
120	hepzibah	25	primi	2	38	4 TO 5	4	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO

121	reshma	19	multi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.6	NORMAL	NORMAL	NO	NO
122	barani	26	primi	2	38	4 TO 5	5	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	3.5	NORMAL	NORMAL	NO	NO
123	chandra	32	primi	3	39	4 TO 5	5	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.8	ABNORM	NORMAL	NO	NO
124	viji	22	primi	2	38	4 TO 5	5	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.8	NORMAL	NORMAL	NO	NO
125	Hema Malini	22	multi	2	38	4 TO 5	5	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.8	ABNORM	NORMAL	YES	NO
126	sanjana	30	multi	1	37	4 TO 5	5	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.8	NORMAL	NORMAL	NO	NO
127	sreeranjani	27	primi	2	38	4 TO 5	5	reactive	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.7	NORMAL	NORMAL	NO	NO
128	kannagi	29	primi	3	39	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.7	NORMAL	NORMAL	NO	NO
129	celine	20	primi	1	37	4 TO 5	4	non react	NO	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
130	meena	26	multi	2	38	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.5	NORMAL	NORMAL	NO	NO
131	lavanya	24	multi	1	37	4 TO 5	5	non react	NO	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.8	NORMAL	NORMAL	NO	NO
132	kavya	25	multi	3	39	4 TO 5	4	reactive	NO	NORMAL	NO		LN	NO	NO	2.4	NORMAL	NORMAL	NO	NO
133	tina	26	multi	3	39	4 TO 5	4	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.5	NORMAL	NORMAL	NO	NO
134	Gayathri	27	multi	1	37	4 TO 5	4	non react	NO	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.5	NORMAL	NORMAL	NO	NO
135	vasuki	24	primi	1	37	4 TO 5	5	non react	NO	ABNORM	NO	MECONIUM	LSCS	NO	YES	3.1	NORMAL	NORMAL	NO	NO
136	shankari	27	primi	2	38	4 TO 5	4	non react	NO	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.8	ABNORM	ABNORM	YES	NO
137	neelavathy	24	multi	2	38	4 TO 5	5	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
138	sheela	24	primi	3	39	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.9	NORMAL	NORMAL	NO	NO
139	preethi	23	multi	1	37	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.7	ABNORM	NORMAL	YES	NO
140	surekha	28	primi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.7	NORMAL	NORMAL	NO	NO
141	hanshitha	22	primi	1	37	4 TO 5	4	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	3	NORMAL	NORMAL	NO	NO
142	preetha	28	multi	2	38	4 TO 5	5	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.5	ABNORM	NORMAL	NO	NO
143	shakila	26	primi	2	38	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.7	ABNORM	NORMAL	NO	NO
144	Sangamithra	25	multi	1	37	4 TO 5	5	reactive	YES	NORMAL	NO		LN	NO	NO	2.7	NORMAL	NORMAL	NO	NO
145	ambuja	24	primi	1	37	4 TO 5	5	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.5	NORMAL	NORMAL	NO	NO
146	Reshma Beevi	22	multi	3	39	4 TO 5	5	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.7	NORMAL	NORMAL	NO	NO
147	quincy	23	primi	2	38	4 TO 5	4	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.8	NORMAL	NORMAL	NO	NO
148	bakya	28	primi	2	38	4 TO 5	5	non react	NO	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.5	NORMAL	NORMAL	NO	NO
149	suganthi	28	multi	1	37	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.6	NORMAL	NORMAL	NO	NO
150	shambu	24	primi	1	37	4 TO 5	4	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.8	ABNORM	NORMAL	NO	NO
151	maraim	28	primi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.4	NORMAL	NORMAL	NO	NO
152	sneha	28	multi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	3	ABNORM	NORMAL	NO	NO
153	dhanam	23	primi	1	37	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.9	ABNORM	NORMAL	NO	NO
154	ajina	25	multi	3	39	1 TO 3	3	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
155	suman	25	primi	3	39	4 TO 5	4	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.9	NORMAL	NORMAL	NO	NO
156	usha	26	multi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.5	NORMAL	NORMAL	NO	NO
157	geetha	22	multi	2	38	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.7	ABNORM	ABNORM	YES	NO
158	riya	23	primi	1	37	4 TO 5	4	reactive	NO	NORMAL	NO		LN	NO	NO	2.5	NORMAL	NORMAL	NO	NO
159	sita	24	multi	2	38	4 TO 5	4	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.6	ABNORM	NORMAL	NO	NO
160	bargavi	22	multi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.7	NORMAL	NORMAL	NO	NO
161	shantha	27	primi	4	40	4 TO 5	4	reactive	NO	NORMAL	NO		LN	NO	NO	2.5	NORMAL	NORMAL	NO	NO

162	sangavi	27	primi	2	38	4 TO 5	4	reactive	NO	NORMAL	NO		LN	NO	NO	2.8	NORMAL	NORMAL	NO	NO
163	mani	29	primi	2	38	4 TO 5	4	reactive	NO	NORMAL	NO		LN	NO	NO	2.6	NORMAL	NORMAL	NO	NO
164	adhirai	25	primi	2	38	4 TO 5	5	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	3	NORMAL	NORMAL	NO	NO
165	pavithra	27	primi	2	38	4 TO 5	5	reactive	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.7	NORMAL	NORMAL	NO	NO
166	maria	22	primi	3	39	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.5	NORMAL	NORMAL	NO	NO
167	malathy	20	multi	1	37	4 TO 5	4	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.5	NORMAL	NORMAL	NO	NO
168	anu	22	multi	3	39	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.5	NORMAL	NORMAL	NO	NO
169	prabha	23	primi	2	38	1 TO 3	3	non react	NO	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.5	NORMAL	NORMAL	NO	NO
170	anandhi	26	multi	3	39	4 TO 5	4	reactive	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.5	ABNORM	NORMAL	NO	NO
171	deepika	23	multi	2	38	4 TO 5	5	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.7	NORMAL	NORMAL	NO	NO
172	sheela	27	primi	2	38	4 TO 5	4	reactive	NO	NORMAL	NO		LN	NO	NO	3.2	NORMAL	NORMAL	NO	NO
173	surya	24	multi	2	38	4 TO 5	5	reactive	YES	NORMAL	NO		LN	NO	NO	2.7	NORMAL	NORMAL	NO	NO
174	fortuna	26	primi	4	40	4 TO 5	4	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.5	NORMAL	NORMAL	NO	NO
175	rekha	26	multi	1	37	4 TO 5	4	reactive	YES	NORMAL	NO		LN	NO	NO	2.5	NORMAL	NORMAL	NO	NO
176	vijaya	24	multi	2	38	4 TO 5	5	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	3	NORMAL	NORMAL	NO	NO
177	divya	28	primi	1	37	4 TO 5	4	reactive	NO	NORMAL	NO		LN	NO	NO	2.9	NORMAL	NORMAL	NO	NO
178	sumathy	24	primi	3	39	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.7	NORMAL	NORMAL	NO	NO
179	Devaki	23	primi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.8	NORMAL	NORMAL	NO	NO
180	Garpaga Lakshmi	26	primi	4	40	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	3.1	NORMAL	NORMAL	NO	NO
181	uma	29	primi	4	40	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.6	NORMAL	NORMAL	NO	NO
182	manali	27	multi	2	38	1 TO 3	3	non react	NO	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	3.2	ABNORM	ABNORM	YES	NO
183	saranya	24	multi	1	37	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.6	ABNORM	ABNORM	YES	YES
184	geetha	25	primi	2	38	4 TO 5	4	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.8	NORMAL	NORMAL	NO	NO
185	aruna	24	primi	1	37	4 TO 5	5	non react	NO	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.7	NORMAL	NORMAL	NO	NO
186	deena	25	multi	2	38	4 TO 5	4	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.5	NORMAL	NORMAL	NO	NO
187	nithya	22	multi	2	38	4 TO 5	4	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.5	NORMAL	NORMAL	NO	NO
188	ajitha	21	primi	3	39	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.6	NORMAL	NORMAL	NO	NO
189	ragini	24	primi	2	38	4 TO 5	5	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.6	NORMAL	NORMAL	NO	NO
190	Bhavani	23	primi	1	37	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.9	NORMAL	NORMAL	NO	NO
191	Sowmya	23	primi	3	39	4 TO 5	5	non react	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	3	NORMAL	NORMAL	NO	NO
192	lina	22	multi	2	38	4 TO 5	4	non react	NO	ABNORM	NO	MECONIUM	LSCS	NO	YES	3.3	NORMAL	NORMAL	NO	NO
193	priya	19	primi	3	39	4 TO 5	4	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.4	ABNORM	ABNORM	YES	YES
194	thenmozhi	24	multi	2	38	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.7	NORMAL	NORMAL	NO	NO
195	regina	23	multi	1	37	4 TO 5	5	reactive	NO	NORMAL	NO		LN	NO	NO	2.4	NORMAL	NORMAL	NO	NO
196	keerthi	31	multi	2	38	4 TO 5	4	reactive	NO	NORMAL	NO		LN	NO	NO	2.6	NORMAL	NORMAL	NO	NO
197	neeta	29	primi	2	38	4 TO 5	5	reactive	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	2.4	NORMAL	NORMAL	NO	NO
198	bojana	28	primi	3	39	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.8	NORMAL	NORMAL	NO	NO
199	poornima	28	primi	3	39	4 TO 5	4	non react	YES	ABNORM	NO	FETAL DISTRESS	LSCS	NO	YES	3.2	NORMAL	NORMAL	NO	NO
200	Yazhini	26	primi	1	37	4 TO 5	5	reactive	YES	ABNORM	NO	MECONIUM	LSCS	NO	YES	2.7	NORMAL	NORMAL	NO	NO

சுயஒப்புதல்படிவம்

ஆய்வுசெய்யப்படும்தலைப்பு : "A STUDY ON THYROID DYSFUNCTION AMONG PATIENT UNDERGOING AMIODARONE THERAPY IN TERTIARY CARE CENTRE"

ஆய்வுசெய்யப்படும்இடம்:

பங்குபெறுபவரின்பெயர்:

பங்குபெறுபவரின்வயது: **பங்குபெறுபவரின்எண் :**

மேலேகுறிப்பிட்டுள்ளமருத்துவஆய்வின்விவரங்கள்எனக்குவிளக்கபட்டுள்ளது.நான்இவ்வாய்வில்தன்னிச்சையாகபங்கேற்கின்றேன்.எந்தகாரணத்தினாலோ, எந்தசட்டசிக்கலுக்கும்உட்படாமல்நான்இவ்வாய்வில்இருந்துவிலகிக்கொள்ளலாம் என்றும்அறிந்துகொண்டேன்.

இந்தஆய்வுசம்பந்தமாகவோ,
இதைசார்ந்துமேலும்ஆய்வுமேற்கொள்ளும்போதும்இந்தஆய்வில்பங்குபெறும்மருத்துவர்,
என்னுடையமருத்துவஅறிக்கைகளைபார்ப்பதற்குஎன்அனுமதிதேவைஇல்லைஎன அறிந்துகொள்கிறேன்.
இந்தஆய்வின்மூலம்கிடைக்கும்தகவலையோ,முடிவையோபயன்படுத்திக்கொள்ள மறுக்கமாட்டேன்.

இந்தஆய்வில்பங்குகொள்ளஒப்புக்கொள்கிறேன்.இந்தஆய்வைமேற்கொள்ளும்மருத்துவஅணிக்குஉண்மையுடன்இருப்பேன்என்றுஉறுதியளிக்கிறேன்.

பங்கேற்பவரின்கையொப்பம்: **சாட்சியாளரின்கையொப்பம்**

இடம்: **இடம்:**

தேதி: **தேதி :**

பங்கேற்பவரின்பெயர்மற்றும்விலாசம்:

ஆய்வாளரின்கையொப்பம்:

இடம்: **தேதி:**