A CLINICAL STUDY OF FETO-MATERNAL OUTCOME IN PREGNANCIES WITH ABNORMAL LIQUOR VOLUME

Dissertation submitted

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CERTIFICATE

This is to certify that the dissertation titled "**A CLINICAL STUDY OF FETO-MATERNAL OUTCOME IN PREGNANCIES WITH ABNORMAL LIQUOR VOLUME**" is a bonafide work done by **Dr.PUNITHAVATHI .J.** in the Institute of Obstetrics and Gynaecology (Madras Medical College) Egmore, Chennai in partial fulfillment of the university rules and regulations for the award of MS degree in Obstetrics and Gynaecology under my guidance and supervision during the academic year 2011-2014.

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I solemnly declare that this dissertation titled "“A CLINICAL STUDY OF FETO-MATERNAL OUTCOME IN PREGNANCIES WITH ABNORMAL LIQUOR VOLUME” was done by me at Institute of Obstetrics and Gynaecology , Madras Medical College during the year 2011 - 2014 under the guidance and supervision of **Prof.DR.UMASHANTHI M.D.,DGO. This dissertation is submitted to The Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.S. Degree in Obstetrics and Gynaecology (Branch -II)**

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ABSTRACT

A CLINICAL STUDY OF FETO-MATERNAL OUTCOME IN PREGNANCIES WITH ABNORMAL LIQUOR VOLUME

AIMS AND OBJECTIVES:

To study the obstetric and perinatal outcome in pregnancies complicated with abnormal liquor volume and to detect the etiological factors responsible for causing abnormal liquor volume.

MATERIALS AND METHODS:

This descriptive study was conducted at Institute Of Obstetrics and Gynaecology, egmore, Chennai from October 2012 to September 2013. In this study, pregnant women with singleton, gestational age between 28- 42 weeks with oligohydramnios (AFI $\leq$ 5) and polyhydramnios ($\geq$ 25) were taken as a study population. They were subjected to detailed history, clinical examination and ultrasound examination with Doppler and they were followed up through out the pregnancy and their fetal and maternal outcome were studied.

RESULTS:

- Most of the women with abnormal liquor volume were presented at term. Isolated oligohydramnios (37.33%) was the most common cause followed by post dated pregnancy (28.67%) in oligohydramnios group. Incidence of congenital anomalies were high in polyhydramnios (22%) than in oligohydramnios (4%). Incidence of induction of labour (65.33%), cesarean section(59.33%), fetal distress(76.4%), meconium stained liquor(57.33%), low 5 minutes APGAR, low birth weight(54%), IUGR(18.66%) and NICU admission(50.66%) were common in oligohydramnios group.

- Idiopathic polyhydramnios (58%) were the first common cause of polyhydramnios, the second were congenital anomalies(22%). Incidence of PROM(44.5%), preterm labour(14%), cord prolapse(6%),atonic PPH (4%), retained placenta(2%) were common in polyhydramnios group. Perinatal mortality were high in polyhydramnios than in oligohydramnios group.

CONCLUSION:

Isolated oligo and polyhydramnios in term gestation has better perinatal outcome compared to early onset and with associated conditions like hypertensive diseases of pregnancy, GDM, IUGR. A detailed history, clinical examination and relevant investigations should be done to identify the various etiological factors in all cases of abnormal liquor volume, to get better foetal outcome as well as to avoid the maternal complications.

KEYWORDS:

Pregnancy, Oligohydramnios, Polyhydramnios, congenital anomalies, perinatal outcome.

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INTRODUCTION

As our ancestors crawled out of the ocean to life on land, We too, float in the amniotic fluid until birth. The Amniotic fluid starts its origin from the maternal plasma by transudation as early as from the seventh week of gestation. Its amount varies throughout the pregnancy. The Amniotic fluid performs several functions during the intrauterine life. It helps to shape the fetal skeleton normally by creating the physical space, promotes fetal lung maturation and protects the umbilical cord from the compression during labour. Too much or too little amount of amniotic fluid is the most common clinically detectable intrinsic abnormality[1] which was the of basis of our study.

Before the era of the invent of ultrasound use in obstetrics, the amniotic fluid volume was assessed clinically by the bimanual palpation and symphysio-fundal height which was found to be unreliable subsequently. In 1950, Prof.sir. Ian donald[2] was the first to demonstrate and document the application of ultrasound to medical diagnosis. In modern obstetrics, ultrasound is an integral part of the obstetrician's armamentarium- almost an extension of the examining finger, because of its non invasive nature, accuracy and repeatability.

The Amniotic fluid volume assessment is an integral part of the antepartum fetal surveillance because of its abnormality is an indicator of poor perinatal outcome. Various ultrasound methos has been proposed for the detection of amniotic fluid, among which the amniotic fluid index(AFI) is the most widely used method. J.P. phelan[3] and colleagues in 1987 proposed this method. According to him, the amniotic fluid volume was categorized as follows,

Normal	8-24 cm
Borderline	5-8 cm
Oligohydramnios	≤ 5
Polyhydramnios	≥ 25

Oligohydramnios is recently defined as AFI below 5th percentile for the gestational age. Post dated pregnancy, uteroplacental insufficiency, congenital anomalies especially renal abnormalities, meconium passage, fetal heart rate abnormalities, low 5 minute APGAR and increased NICU admission are associated with Oligohydramnios[4]. Other studies are also shown that it is associated with increased perinatal morbidity and mortality. Hence antepartum fetal surveillance is mandatory in pregnant women with Oligohydramnios. Hence Oligohydramnios in term is considered as an indication for termination of pregnancy.

Polyhydramnios is defined as AFI > 95th percentile for gestational age. More than fifty percent of women with polyhydramnios, the etiology was unknown. Congenital fetal anomalies accouts for 20%, among which anencephaly occurs in 50% of the cases. Gestational diabetes, congenital infections also leads to the development of polyhydramnios. An increased risk of congenital abnormalities and perinatal mortality are associated with increasing severity of polyhydramnios[5]. Severe polyhydramnios (AFI ≥35 cm) is commonly associated with major congenital anomaly in 31% of cases.

So amniotic fluid volume assessment is an useful method to identify the fetus at risk for adverse obstetric and perinatal outcome.

Therefore the present study was conducted to find out the perinatal and maternal outcome and to identify the possible causes of abnormal liquor volume.

AIMS AND OBJECTIVES

1. To study the obstetric outcome in pregnancies with oligohydramnios and polyhydramnios.
2. To determine the perinatal outcome in pregnancies complicated with oligohydramnios and polyhydramnios.
3. To determine the possible factors causing oligohydramnios and polyhydramnios

REVIEW OF LITERATURE

AMNIOTIC FLUID

Sources and Circulation:

During the intrauterine development, the foetus is surrounded by the amniotic fluid . The precise site of origin of amniotic fluid is not well understood till now. Both maternal and foetal factors contributes to the development of liquor amnii[6,7]. It is produced from the sources listed below[6],

1. Transudation of maternal plasma across the amnion and chorion
2. Transudation from foetal circulation through umbilical cord and placental membranes
3. Transudation of foetal serum through the permeable foetal skin before keratinisation
4. Secretion from the amniotic epithelium
5. Foetal urine is the major source after 20 weeks of pregnancy
6. Foetal lung fluid that enters amniotic cavity
7. Secretions from foetal oral-nasal cavities also contributes to small extent

In first trimester inward transfer of solutes along with passive diffusion of water from extracellular fluid through the amnion and the permeable skin of the foetus is the likely source of amniotic fluid. After 20th week, increasing stratification and cornification of the skin prevents diffusion, the foetal urine becomes the main source of amniotic fluid thereafter. During 4th- 5th weeks of gestation, foetal kidneys start to develop, by 8th to 11th weeks it begin to excrete urine and by 20thweek produces most of the amniotic fluid. Daily urine production depends upon the weight of the foetus, approximately 30% of foetal weight. The excreted urine via the amniotic fluid is recycled back to the foetus by swallowing, it is approximately 25% of foetal weight, hence it will not serve real excretory or homeostatic function. Therefore foetal urine output should be adequate to maintain amniotic fluid volume. An another important contributor of AFV is foetal lung fluid[8].

Brace[9] et al 1997 described the factors involved in regulation of amniotic fluid volume,

Flow out of the amniotic sac,

1. Foetal swallowing (500-1000ml/ day)
2. Intramembranous flow across the placenta and umbilical cord (200-500 ml/day)

3. Transmembranous flow from amniotic cavity into the uterine circulation (10ml/day)

Flow into the amniotic sac,

1. Foetal urination (800-1200ml/day)
2. Foetal lung liquid secretion (170ml/day)
3. Oral-nasal secretions (25ml/day)

Various conditions which affects these factors results in abnormal liquor volume during pregnancy.

Volume of Amniotic Fluid[8]:

Amount of AFV varies throughout the pregnancy. It increases from 1ml at seven weeks to 25ml at ten weeks, 400ml at 20 weeks reaches about 1 litre at 36 weeks . Thereafter it decreases progressively to about 800ml at term, as the pregnancy continues post term, further reduction occurs to the extent of 200ml at 42 weeks.

Abhilash sandhyala and Radswiki et al studied the rate of change of amniotic fluid during each gestation. It raises from 10ml/ week at 8 weeks to 25 ml/week at 13 weeks reaches a maximum at 21 weeks of about 60 mls/week and then decreases and reached 0 at 33 weeks. AFV decreases at rate of 8%/week after term. Fetal weight is corresponds with

AFV during the first half of pregnancy. Upto 30 weeks of gestation, ratio of amniotic fluid to fetal volume increase and then declines

Queenan[10] et al 1991 also described the correlation of AFV with fetal and placental weight in grams.

Gestation age in weeks	16	28	36	40
Amniotic fluid in ml	200	1000	900	800
Fetas weight in grams	100	1000	2500	3300
Placenta in grams	100	200	400	500

Physical Features of Amniotic Fluid:

Amniotic fluid is slightly alkaline in nature with pH of 7-7.5. Lower electrolyte concentration of fetal urine makes it hypotonic and it contains more urea, creatinine and uric acid compared to maternal serum. With increasing gestational age, fetal urine osmolality decreases. Specific gravity of liquor amnii is low[6,7]

The colour of the amniotic fluid changes during the normal course of pregnancy. Before 20 weeks it ranges from a pale straw colour to deep yellow depending upon the amount of bilirubin. Before 20 weeks bilirubin is the normal constituent of amniotic fluid and does not indicate

the rhesus hemolytic disease in the fetus. After that the bilirubin concentration decreases. Normal amniotic fluid is colourless by 36 weeks of gestation. White floccules sometimes appear in the fluid during the last 4-5 weeks due to the presence of desquamated fetal cells and free lipid material(vernix caseosa)[6].

Abnormal colouring usually results from contamination with meconium or blood, but it may also be due to bilirubin. High bilirubin levels after 30 weeks is considered as abnormal[6].

Chemical Composition of Amniotic Fluid[6,7]:

The chemical composition of amniotic fluid is identical to maternal plasma in first half of pregnancy, as pregnancy advances it is changed markedly due to the addition of fetal urinary metabolites.

The main content of amniotic fluid is water constitute 98.1-99%, the solid part forms the minor component of about 1-2 %. Solid component includes organic, inorganic and other suspended particles

Organic Components:

Protein -0.5mg,

Non protein nitrogen-24mg

Uric acid-4-5 mg,

Sugar 19 mg,

Creatinne 2.2mg/ 100ml of amniotic fluid,

Urea-30 mg,

Total lipids- 50 mg,

Bilirubin,

Enzymes

Hormones-Cortisone, human chorionic gonodotrophin, human placental lactogen, pregnanediol, 17-OH corticosteroids, estriol.

Inorganic Components:

Sodium, potassium, chloride and calcium. Sodium and chloride concentration decreases as pregnancy advances but potassium remains unchanged.

Others:

Cells from bladder, vagina and respiratory tract

Vernix caseosa

Exfoliated squamous epithelial cell from fetal skin and lanugo hair

Amniotic cells

Evaluation of Amniotic Fluid:

A diagnosis of an amniotic fluid abnormality may be suspected by physical examination like uterine fundal height & dates variation, but the diagnosis is generally made by the examination of the fluid compartments. Ultrasound evaluation is widely used technique among the various tests available to detect AFV. Being a non invasive method, makes it ideal for large scale use and repeat AFV determination in suspected amniotic fluid abnormalities. AFV by USG is done either by simple visual estimation or by biometric assessment. It is a semiquantitative method, never represent a true quantitative method[11,12].

Various methods used are,

1. **Dye dilution test[13]:** It is considered as gold standard for assessment of amniotic fluid volume. However this is an invasive technique requiring amniocentesis and therefore not suitable for clinical practice which often needs repeated evaluation. In this technique a known volume of dye like aminohippurate sodium is injected into the amniotic cavity through amniocentesis. A sample of dye is taken after 20 mints which is analysed with spectrometry for degree of dilution. It reflects the actual AFV but invivo dye concentrations may undergo rapid changes.

2. Ultrasound evaluation of the amniotic fluid

- **Subjective method:** It is based on the visualisation of AF pockets without measurements. The results are reported as either normal, low or high[14]. Examination by an experienced sonographer is necessary to reduce the intraobserver variation which is common in this method[15].The results of this method is comparable with objective methods like AFI, SDVP, 2DP and dye dilution method.
- **Single deepest vertical pocket(SDVP):** Manning et al [16]in 1981 described the concept of measuring the depth of maximum vertical pocket(MVP).They defined severe oligohydramnios as MVP <1cm, reduced liquor as MVP 1-2 cm.

  In 1984 chamberline et al[16] defined the normal amount of amniotic fluid as the largest vertical pocket measuring 2-8cm, oligohydramnios as SDVP <2cm and polyhydramnios as SDVP >8 cm. While measuring SDVP ultrasound transducer probe should be right angle to the uterine contour without loops of cord structures and fetal parts.

- **Amniotic fluid index (AFI):** This method was proposed by phelan et al[3] in 1987. It is a more objective and reproducible method as it estimates the amniotic fluid in four quadrants. The uterus is arbitrarily divided into four quadrants by the umbilicus transversely and linea nigra vertically. The deepest vertical pocket with no loops of cord and free of foetal parts in each quadrant is measured and it is summed up to give the AFI. Pockets are measured perpendicular to the floor with the patient in supine position. An AFI of 5-18 cm is considered normal, AFI of 18cm or greater is polyhydramnios or less than 5cm is oligohydramnios. Recently oligohydramnios has been defined as less than 3rd and 5th percentile and hydramnios more than 95th and 97th percentile for gestational age[17]. The reliability of correctly identifying oligo- or polyhydramnios using the percentiles is similar to SDVP(2-8) and AFI (5-18).
- **Two diameter pocket method (2-DP):** It is an another semi quantitative method to assess the AFV which was described by magnan et al[18] in 1992. He multiplied the depth of largest vertical pocket to its transverse diameter. According to this method normal AFV is 2-DP 15.1-50cm2, 2-DP <15cm2 defined as oligohydramnios and 2DP > 50cm2 defined as hydramnios.

Though the accuracy of ultrasound indices is good to diagnose normal amount liquor amnii, the sensitivity for both oligohydramnios and polyhydramnios remains poor[19]. All these measurements suffer from methodological limitations of two dimensional ultrasound and interference from foetal movements and loops of cord.

Functions of Amniotic Fluid[6,7]:

- Amniotic fluid acts as a shock absorber to protect the growing foetus from any external injury
- Prevents adhesion formation between fetal parts and amniotic sac
- Supplying nutrients
- Facilitating growth and development of musculoskeletal system, lungs and gastrointestinal tracts
- Promotes surfactant synthesis
- Provides thermally stable environment
- During labour it helps in dilatation of cervix by forming a wedge the bag of membranes
- The amniotic fluid in the intact membranes prevents interference with placental circulation by preventing the umbilical cord compression during the uterine contraction.

- Antiseptic and bactericidal action of AF prevents ascending infection into the uterus

Clinical Importance of Amniotic Fluid[6,7]:

Amniocentesis has to be done to collect amniotic fluid for the following clinical purposes,

- For the detection of developmental abnormalities and genetic diseases in the fetus
- To assess the fetal lung maturity
- To check fetal renal maturity
- Hyaluronic acid which is rich in AF promotes bone healing
- Prostaglandins and hypertonic saline are injected in the amniotic cavity for the induction of abortion
- Artificial rupture of membranes is a one of the method for the induction and augmentation of labour
- Detection of abnormal liquor volume either excess or low by AFI, helps in identifying a fetus at risk

POLYHYDRAMNIOS:

Excessive amniotic fluid of more than 2000-2200ml is defined as polyhydramnios[1,6,7]. The incidence of polyhydramnios is 1%-2%, independant of race and ethinicity[20]. Multiparous women has increased risk to develop polyhydramnios than primi.

Definitions of hydramnios according to various study:

Chamberlin et al[21,22]	SDVP > 8 cm
Phelan et al[3]	AFI > 25 cm
Carlson et al[23]	AFI > 2SD of the mean for late 2nd and 3rd trimester (24cm)
Moore et al[17]	> 95th to 97th percentile for gestational age

Classification:

Based on the severity, Hill[24], Biggio[25] and Golan[26] classified the polyhydramnios as mild, moderate and severe. Harman CR[27] et al studied the perinatal mortality and anomalies associated with different types of polyhydramnios.

Types	SDVP in cm	AFI in cm	%	Perinatal Mortality in 1000	Anomalies(%)
Mild	8-11	25-30	80	50	≤ 6
Moderate	12-15	30-35	15	190	≤ 45
Severe	>16	>35	5	540	≤ 65

Based on the onset, it is further classified as acute and chronic[1,6].

Acute polyhydramnios: it is a rare condition with acute onset and the accumulation of fluid within a few days. It often manifests before 20 weeks, associated with monozygotic twins and chorioangioma of the placenta. Usually spontaneous abortion occurs, slow amnioreduction can be done for maternal distress. It often needs repeated amniocentesis.

Chronic polyhydramnios: It is the most common type with gradual increase in fluid over few weeks. It usually occurs after 32 weeks.

Causes of hydramnios:

Polyhydramnios can be due to excessive production of liquor amnii or due to defective absorption. The degree of hydramnios as well as its prognosis is often related the cause. Both maternal and fetal causes leads to the development of polyhydramnios.

Its various causes are as follows:

1. Idiopathic: In 66% of cases, cause is unknown
2. Fetal causes:

   Congenital anomalies[28] -

   - ✓ Anencephaly (50%) –It is a most common fetal congenital anomaly causing polyhydramnios. Increased urination caused by impaired ADH secretion, decreased swallowing reflex and increased transudation from the exposed meninges are the possible causes of hydramnios.
   - ✓ Open spina bifida- Increased transudation from the exposed meninges
   - ✓ Esophageal and duodenal atresia (15%) - Decreased swallowing of the liquor
   - ✓ Facial clefts and neck masses- by interfering with normal swallowing
   - ✓ Congenital diaphragmatic hernia
   - ✓ Fetal bartter syndrome
   - ✓ Fetal muscular dystrophy
   - ✓ Fetal sacrococcygeal teratoma
   - ✓ Fetal vein of galen aneurysm
   - ✓ Fetal infections

- ✓ Hydrops fetalis due to Rh isoimmunisation, cardiothoracic anomalies and fetal cirrhosis

Multiple pregnancy due to large placenta- 10 times the incidence, It is more commmon in monoamniotic twins affecting the second sac

3. Placental causes:
   - ✓ Placental chorioangioma due to increased transudation
4. Maternal causes:
   - ✓ Diabetes (30%)- Due to fetal hyperglycemia causing fetal diuresis and hydramnios
   - ✓ Cardiac or renal diseases due to increased transudation from edematous placenta

Clinical Presentation:

Symptoms[1,6,7]:

Depending upon the rapidity of its onset and degree of hydramnios, the clinical presentations will vary. Acute polyhydramnios will manifest like acute abdominal catastrophe like pain abdomen, nausea, vomiting. In gradual onset, the patient may present with increased abdominal girth, breathlessness on supine posture, digestive discomfort, swelling of the legs, varicosities in lower limb, occasionaly it can cause hyperemesis.

Mirror syndrome or ballantyne syndrome occurs in hydrops foetalis with hydramnios.

Signs[1,6,7]:

Dyspnoea on supine position

Signs of preeclampsia –hypertension, albuminuria, edema.

The foetus is freely ballottable

Fluid thrill is present

Foetal parts are difficult to palpate, foetal heart sounds are not easily audible

Malpresentations are common

Evaluation[29]:

Ultrasonography :

- ✓ It is helpful in the diagnosis of hydramnios
- ✓ To exclude the other causes of hydramnios.
- ✓ To detect associated congenital anomalies
- ✓ To know the lie and presentation of the foetus

Blood Investigations:

- ✓ Glucose tolerance test should be done to all women to exclude gestational diabetes.
- ✓ Blood grouping and typing. If USG shows foetal hydrops, maternal antibody screen for D, C, Kell and Duffy antigen should be done to exclude alloimmunisation. Further evaluation for non immune hydrops can be done if antibody testing is negative. These include serology testing for syphilis, IgG and IgM for rubella, toxoplasma, parvovirus and cytomegalovirus.

Invasive testing like amniocentesis can be performed for foetal karyotyping

Differential Diagnosis[6,7]:

1. Multiple pregnancy – it can be excluded from polyhydramnios by 1. Fundal height is more than the period of gestation 2. Too many foetal parts 3. Fluid thrill absent 4. USG will confirm the diagnosis
2. Large ovarian cyst complicating pregnancy – 1. The gravid uterus is felt separately from the cyst 2. The cervix is pushed down into the pelvis but in hydramnios the cervix is drawn up

3. Maternal ascites- 1. Presence of shifting dullness 2. Resonance in the midline due to floating gut whereas in hydramnios it is dull 3.Size of the uterus will be normal
4. Retoverted gravid uterus with full bladder
5. Hydatiform mole
6. Concealed abruption

Complications[1,6,7]:

Fetal Complications:

Perinatal morbidity and mortality is increased in polyhydramnios. Most cases of mild hydramnios are idiopathic and carry a low risk for undiagnosed anomalies compared to severe hydramnios. Premature delivery and congenital anomalies are the main foctors responsible for morbidity and mortality. Other factors are cord prolapse, hydrops foetalis, operative delivery and abruption

Maternal Complications:

During Pregnancy:

1. Abruptio placentae is most dreadly complication of hydramnios
2. Gestatioal hypertension
3. Abnormal foetal presentation

4. PROM
5. Premature delivery either spontaneous or induced
6. Cardio respiratory embarrassment

During Labour:

1. Increased incidence of cord prolapse
2. Dysfunctional labour
3. Uterine inertia
4. Increased operative delivery
5. Increased cesarean delivery
6. Postpartum hemorrhage
7. Retained placenta

Postpartum Period:

1. Subinvolution is common
2. Puerperal sepsis due to increased operative interference and blood loss

Management :

- Conservative Management with close observation will suffice in most of the cases of minor degree of Polyhydramnios

- Moderate type of Polyhydramnios can be managed until labour starts.

- Severe type often requires hospitalization, due to maternal respiratory distress, significant abdominal pain or premature uterine contractions. In this condition therapeutic amniocentesis is required.

- Serial amnioreduction is required in conditions with fetal abnormality or twin-twin transfusion syndrome with severe polyhydramnios.

- Amniocentesis: During amniocentesis 500 ml per hour (1500 to 2000ml per day) can be removed in single setting . Before the procedure placental localization should be done with ultra sound.

- Risks of Amniocentesis are fetal loss (1.2%), preterm labour, premature rupture of membranes, placental abruption, chorioamnionitis, Rh isoimmunisation and fetal pneumothorax.

- Prostaglandin synthetase inhibitors: Among the PG synthetase inhibitors, indomethacin is the most commonly used drug. It reduces the amniotic fluid volume by decreasing the urine production from the fetal kidneys, decreasing the production of lung fluid and increased removal of fluid from the lungs as well as increased movement across fetal membranes. Dose is 1.4-3 mg/kg daily. (25mg 4-6 hourly to 75 mg 12 hourly). Maternal side effects are GIT disturbances, rectal irritation, transient prenal insufficiency and cholestatic jaundice.

- Sulindac is another prostaglandin inhibitor used in the treatment of polyhydramnios.

- If it is decided to induce labour, liquor should be drained carefully in a controlled manner, either by amniocentesis or by a needle inserted into the forewater to prevent cord prolapse and abruption.

OLIGOHYDRAMNIOS:

Oligohydramnios is the condition in which the amount amniotic fluid is reduced to <200 ml at term. Incidence vary between 0.5 - 5%.

Definitions based on USG measurements are,

Manning[16] et al	MVP < 1cm
Chamberline [21,22]et al	SDVP <2cm
Phelan[3] et al	AFI <5cm
Jeng[30] et al	AFI <8cm

Conditions Associated with Oligohyadramnios[1,6,29]:

Maternal Causes:

1. Preterm premature rupture of membranes- 3-17 %
2. Uteroplacental insufficiency
3. Preeclampsia
4. Postdated pregnancy
5. Autoimmune disorders
6. Drugs like ACE inhibitors, PG synthesis inhibitors

Fetal Causes:

1. Chromosomal abnormalities- triploidy, turner syndrome, trisomy 18 - 4.4-30.7%
2. Intrauterine growth restriction
3. Intra uterine fetal demise
4. Fetal infections
5. Congenital anomalies- 7-37%

    Bilateral renal agenesis
    Multicystic dysplastic kidneys
    Bladder outflow tract obstruction
    Infantile polycystic kidney disease
    Musculoskeletal
    Cardiac
    Digestive tract anomalies.

Placental Causes:

1. Abruptio placentae
2. TTTS

Idiopathic: Failure of secretion from amnion cells

Clinical Implications[1,26]:

Maternal outcome is not affected adversely due to oligohydramnios per se. However maternal morbidity is indirectly increased by higher rate of induction and cesarean deliveries.

Perinatal mortality and morbidity is significantly increased in oligohydramnios particularly if it occurs remote from term. Congenital anomalies, pulmonary hypoplasia, concurrent early onset of preeclampsia and IUGR are the main contributors to this adverse perinatal outcome. Recent studies and reviews suggests that isolated oligohydramnios in pregnancies with appropriately grown fetuses, reassuring fetal heart pattern and no maternal disease, does not increase the incidence of adverse perinatal outcome and therefore does not justify induction of labour.

Diagnosis[1,6,26]:

1. Uterine size is smaller than expected
2. Decreased fetal movements
3. Fetal parts are easily palpable
4. USG is confirmatory

Management

It depends on

1. Gestational Age
2. Severity of oligohydramnios
3. Fetal status & well being
4. Etiology

Treatment includes adequate rest

Hydration therapy – either oral or intravenous fluids (2 litres per days) to improve amniotic fluid volume.

Amnioinfusion is done to prevent cord compression during labour & in case of meconium stained liquor to avoid meconium aspiration syndrome in the fetus.

Induction of labour / LSCS is based on fetal lung maturity, presence of congenital malformations, IUGR, severity of oligohydramnios.

MATERIALS AND METHODS:

This descriptive study was carried out in the Department of Obstetrics and Gynaecology at Institute of Obstetrics and Gynaecology, Egmore, Chennai from October 2012 to September 2013, to evaluate the feto-maternal outcome in pregnancies complicated with abnormal liquor volume. Pregnant women with abnormal liquor volume who attended the antenatal clinic regularly and those who fulfilled the following criteria were included in this study as a study population.

Cases were selected according to the following inclusion and exclusion criteria.

Inclusion Criteria for Oligohydramnios:

1. Singleton pregnancy
2. Ultrasound finding of AFI $\leq$ 5cm
3. Gestational age of 28-42 weeks
4. With intact membranes

Exclusion Criteria for Oligohydramnios

1. Intrauterine Fetal demise
2. Prelabour rupture of membranes
3. Post term pregnancy

Inclusion Criteria for Polyhydramnios:

1. Singleton pregnancy
2. Ultrasound finding of AFI ≥ 25cm
3. Gestational age of 28 - 42weeks

Exclusion Criteria for polyhydramnios

1. Multifetal gestation
2. Pregnancies before 28 weeks

Approval from the Institutional Ethical Committee was sort. After getting the informed consent from the pregnant women who fulfilling the above criteria were taken up for the study.150 cases of Oligohydramnios and 50 cases of Polyhydramnios were selected as a study population. The study population were subjected to a detailed history taking including age of the patient, parity, last menstrual period, previous menstrual history, obstetric history, past medical and surgical history, family history, personal history were taken. Followed by complete physical examination including general examination (including Ht, Wt, BMI, BP recorded at each visit, presence of anemia and pedal edema), cardiovascular and respiratory systemic examination, obstetric examination were done. Base

line investigations like urine albumin, sugar& deposits, Hb%, OGCT, blood grouping and typing, HIV,VDRL, and HbsAg test were done.

Obstetrical Ultrasound with Doppler was done for the study population using a real time scanner with 3.5-5MHz transducer by the same person to avoid the inter observer variation. Fetal presentation, gestational age, liquor status, placental localization, and anomalies if present were noted. Amniotic fluid index was measured by the Phelan's technique. In this technique uterus was divided into four quadrants by an imaginary lines drawn through the linea nigra and the umbilicus. Maximum deepest vertical pocket from the each quadrant was measured. Sum total of the four measurement gives the value of AFI. Women with AFI of ≥ 25 were taken as Polyhydramnios group and AFI of ≤ 5 were taken as Oligohydramnios group. These women were closely monitored throughout their antenatal, intrapartum and postpartum periods.

Age of the patient, distribution of parity, Gestational age at delivery, possible etiological factors causing abnormal liquor volume, rate of induction, liquor colour, No of vaginal, cesarean and instrumental deliveries, indications for the cesarean section, intrapartum complications like cord prolapse, preterm labour, PROM, malpresentation, abruptio placentae, immediate postpartum complications like atonic PPH, retained placenta were noted. Fetal outcome, five minutes apgar, birth weight,

Neonatal Intensive Care Unit (NICU) admission, perinatal morbidity and mortality were also noted. All these datas were entered in the preformed proforma and these different variables were tabulated and analyzed by SPSS 11.5.

STATISTICAL METHODS:

T test, chi square and fisher test were used for the statistical analysis. Critical value at 0.05 was considered as a significant level.

OBSERVATION AND RESULTS

TABLE-1

AGE DISTRIBUTION IN ABNORMAL LIQUOR VOLUME

AGE IN YEARS	GROUP			
	AFI ≤5		AFI ≥25	
	NO OF CASES N-150	%	NO OF CASES N-50	%
≤19	12	8.0	3	6
20 -25	86	57.3	17	34
26 – 30	42	28.0	21	42
31 -35	8	5.3	4	8
above 35	2	1.3	5	10.0

This table shows, the age distribution in the study population. Among the 150 oligohydramnios group, 57.3% were in the age of 20-25 yrs. Among the 50 polyhydramnios group, 42% were in the age of 26-30 yrs.

CHART 1

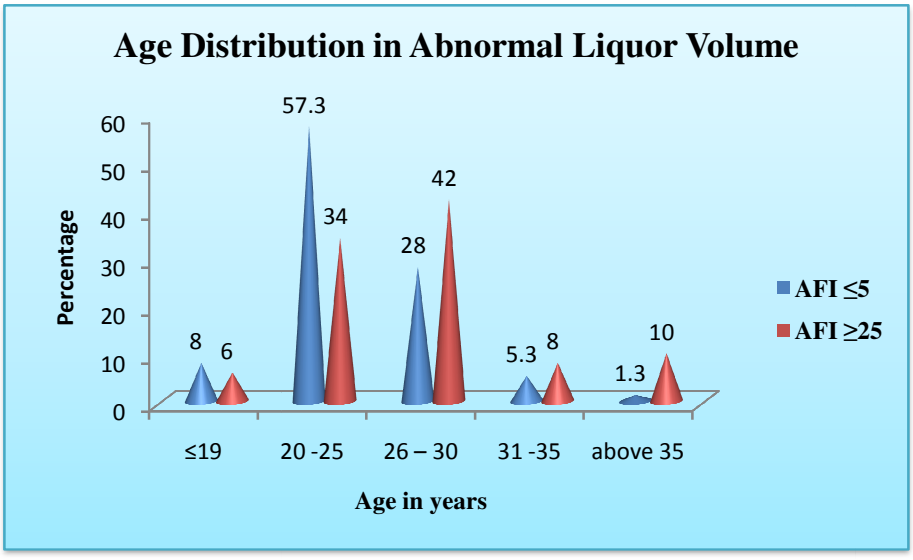


TABLE -2

PARITY DISTRIBUTION IN ABNORMAL LIQUOR VOLUME

OBSTETRIC SCORE	GROUP			
	AFI ≤ 5		AFI ≥ 25	
	NO OF CASES N-150	%	NO OF CASES N-50	%
Primi	99	66	16	32
G2 – 3	51	34	28	56
G4 -5	0	0	6	12

This table shows the parity distribution in abnormal liquor volume. 66% of the cases in oligohydramnios group were primigravida, compared to 56% were multigravida in polyhydramnios group.

CHART 2

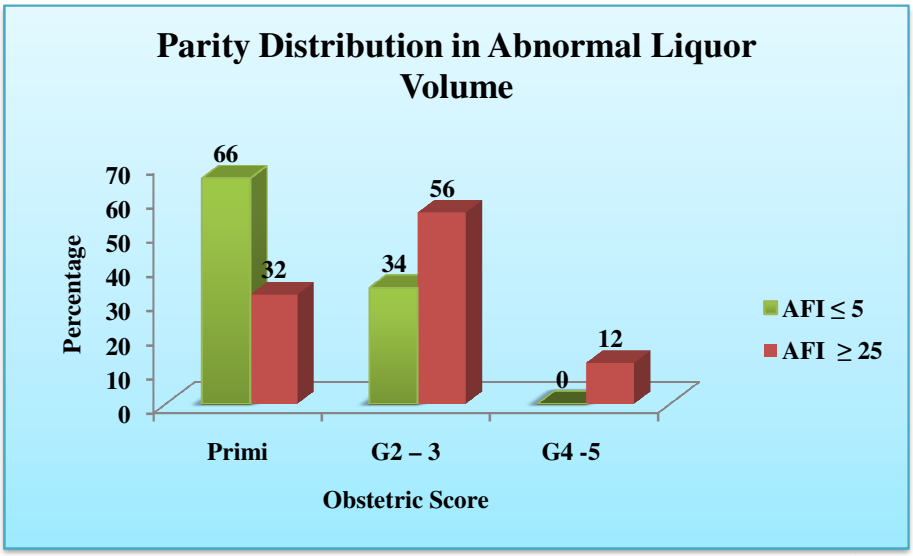


TABLE-3

GESTATIONAL AGE AT DELIVERY IN ABNORMAL LIQUOR VOLUME

	GROUP			
	AFI ≤ 5		AFI ≥ 25	
GESTATIONAL AGE IN WEEKS	NO OF CASES	%	NO OF CASES	%
28 -32 weeks	12	8	7	14
33 -37 weeks	44	29.33	11	22
>37 weeks	94	62.66	32	64

This table shows, the gestation age at delivery in the study population. In oligohydramnios group, 62.66% were in the gestation age of >37 weeks, 29.33% between 33-37 weeks and 8% between 28-32 weeks. In the polyhydramnios group also, 64% were in >37 weeks, 22% between 33-37 weeks, 14% between 28-32 weeks. According to this table, incidence of term and preterm delivery in abnormal liquor volume is not statistically significant with p value 0.338.

CHART 3

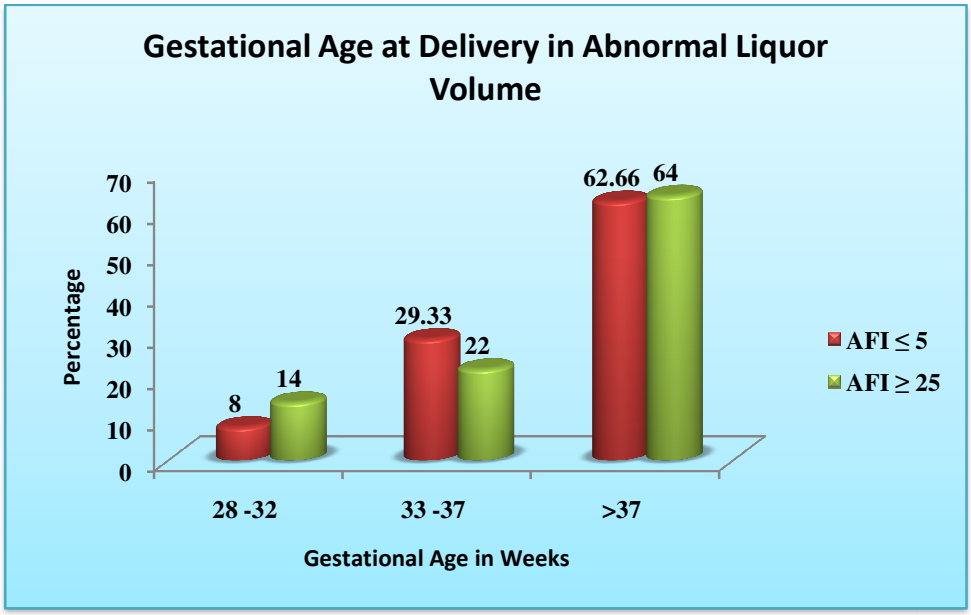

Gestational Age at Delivery in Abnormal Liquor Volume
Percentage
70
60
50
40
30
20
10
0
8
14
29.33
22
62.66
64
28 -32
33 -37
>37
Gestational Age in Weeks
AFI ≤ 5
AFI ≥ 25

TABLE-4

MATERNAL CONDITIONS ASSOCIATED WITH OLIGOHYDRAMNIOS

MATERNAL CONDITIONS	AFI≤ 5 NO OF CASES N-150	%
Postdated pregnancy	43	28.67
Hypertensive Diseases	26	17.34
Anemia	5	3.33
APLA	2	1.33

This table shows maternal conditions associated with oligohydramnios in which 28.67% were postdated pregnancy, 17.34% hypertensive disease (among which 15.38% were gestational hypertension, 84.61% were preeclampsia), 3.33% of anemia and 1.33% of APLA syndrome were present.

CHART 4

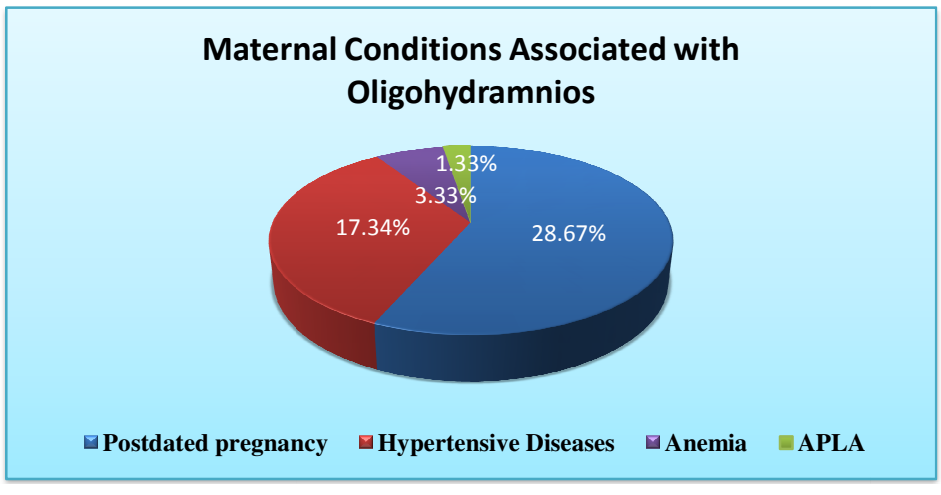


TABLE-5

MATERNAL CONDITIONS ASSOCIATED WITH POLYHYDRAMNIOS

MATERNAL CONDITIONS	AFI ≥ 25 NO OF CASES N- 50	%
GDM	8	16
Gestational Hypertension	2	4
Preeclampsia	3	6
Rh Negative	1	2
Chorioangioma of Placenta	1	2

GDM	NO. OF CASES N-60	%
Controlled	5	62.5
Uncontrolled GDM	3	37.5

This table shows the polyhydramnios associated maternal conditions.Among the 50 cases, GDM were present in 16% (out of which 62.5% were controlled GDM, 37.5% were uncontrolled), Preeclampsia in 6%, gestational hypertension in 4%, Rh negative pregnancy in 2% and chorioangioma of the placenta in 2%.

CHART 5

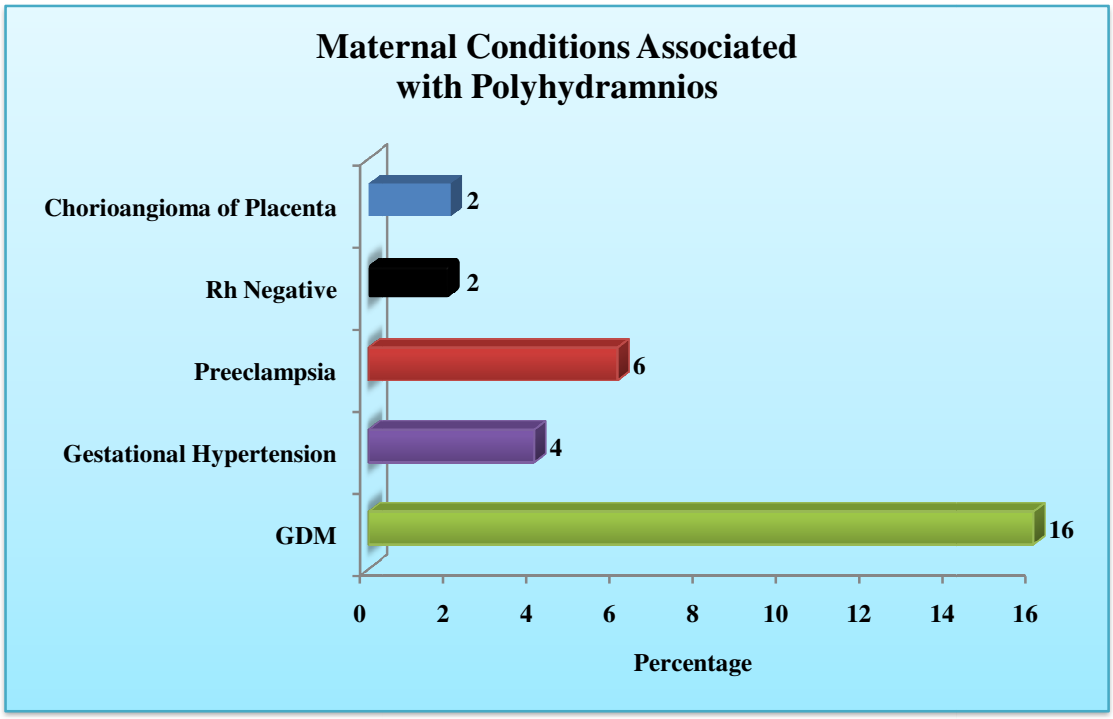

Maternal Conditions Associated with Polyhydramnios
Chorioangioma of Placenta
Rh Negative
Preeclampsia
Gestational Hypertension
GDM
2
2
6
4
16
0
2
4
6
8
10
12
14
16
Percentage

TABLE -6

CONGENITAL ANOMALIES ASSOCIATED WITH

OLIGOHYDRAMNIOS

CONGENITAL ANOMALIES	AFI ≤ 5 NO OF CASES N-6	%
Multicystic dysplastic kidney	1	0.67
Infantile PCKD	3	2.0
Single umblical artery	1	0.67
Micro cephaly	1	0.67

In 50 oligohydramnios patients, only 4% had congenital anomalies. Among which infantile polycystic kidney disease 2%, MCKD 0.67%, Single umbilical artery 0.67%, microcephaly 0.67%.

CHART 6

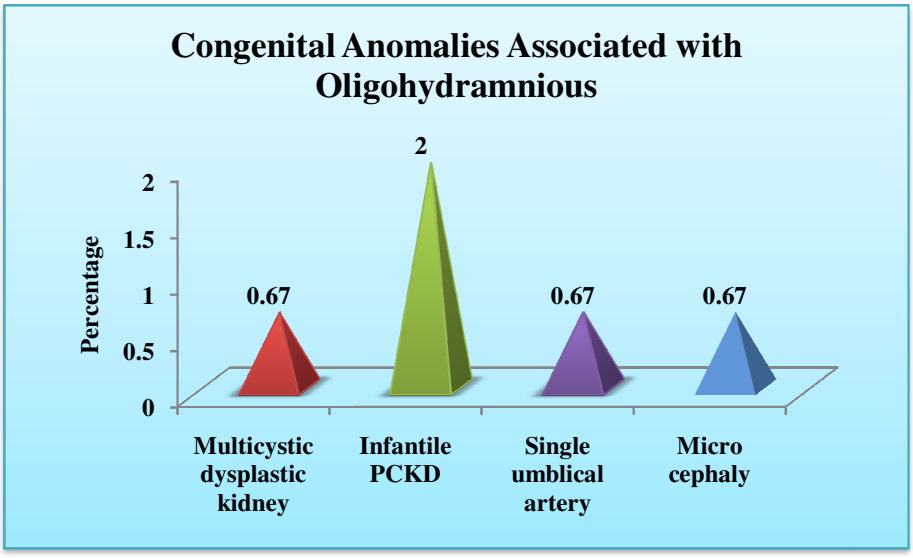


TABLE-7

CONGENITAL ANOMALIES ASSOCIATED WITH POLYHYDRAMNIOS

CONGENITAL ANOMALIES	AFI ≥ 25 NO OF CASES N-11	%
Anencaphaly	4	8
Diaphragmatic Hernia	1	2
Duodenal atresia	1	2
Non Immnue Hydrops	1	2
Spinabifida	2	4
Hydrocephalus with meningomyelocele	2	4

In 50 polyhydramnios cases, Total of 22% had congenital anomalies. Among which anencephaly was the common anomaly accounts 8% , spina bifida 4%, hydrocephalus with meningomyelocele 4%, Diaphragmatic hernia 2%, Duodenal atresia 2% and Non immune hydrops in 2% .

CHART 7

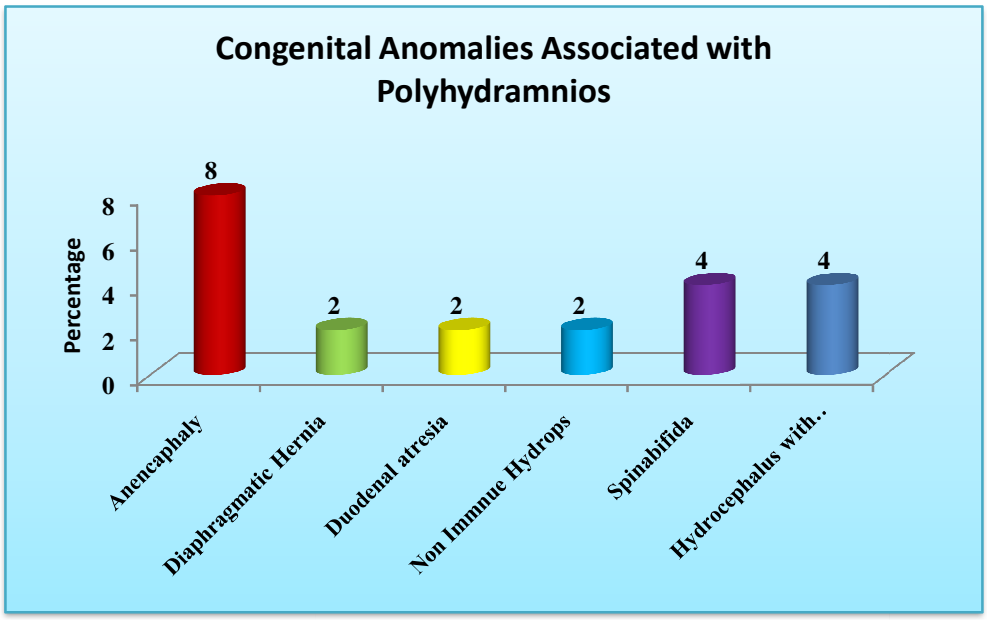

Congenital Anomalies Associated with Polyhydramnios
Percentage
0
2
4
6
8
8
2
2
2
4
4
Anencaphaly
Diaphragmatic Hernia
Duodenal atresia
Non Immnue Hydrops
Spinabifida
Hydrocephalus with..

TABLE -8

ETIOLOGICAL FACTORS IN OLIGOHYDRAMNIOS

ETIOLOGY	AFI ≤ 5 NO OF CASES N-150	%
Postdated Pregnancy	43	28.67
IUGR	19	12.66
Hypertensive Diseases	26	17.34
Congenital Anomalies	6	4.00
Idiopathic	56	37.33

This table shows the etiological factors in oligohydramnios group. According to this table 37.33% were isolated oligohydramnios with no identifiable cause, 28.67% were post dated pregnancy, 12.66% were IUGR, 17.34% were hypertensive diseases, 4% were congenital anomalies.

CHART 8

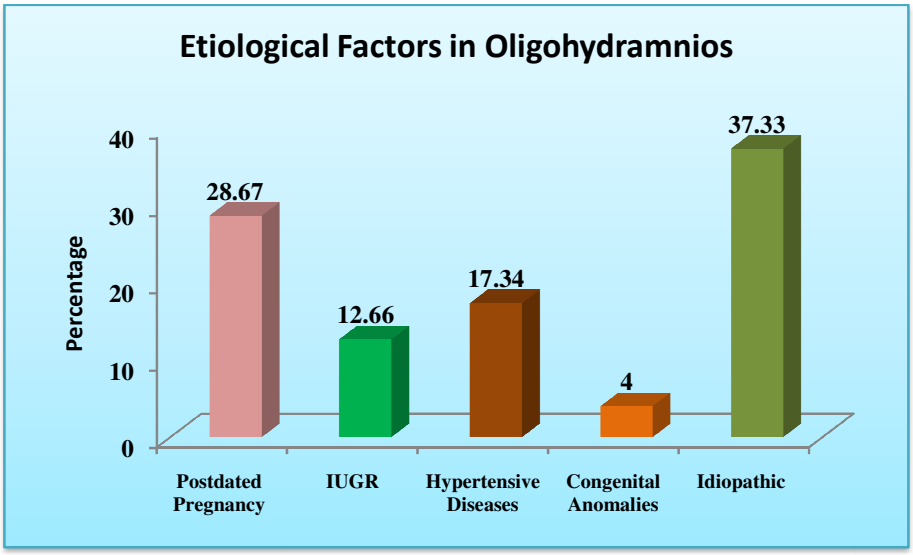

Etiological Factors in Oligohydramnios
Percentage
40
30
20
10
0
28.67
12.66
17.34
4
37.33
Postdated Pregnancy
IUGR
Hypertensive Diseases
Congenital Anomalies
Idiopathic

TABLE -9

ETIOLOGICAL FACTORS IN POLYHYDRAMNIOS

ETIOLOGY	AFI ≥ 25 NO OF CASES N-50	%
Idiopathic	29	58
Congenital Anomalies	11	22
GDM	8	16
Rh- Isoimmunisation	1	2
Chorioangioma of the placenta	1	2

This table shows the various etiological factors causing polyhydramnios in the study population. Exact cause of polyhydramnios were not detected in 58% of the cases. 22% had congenital anomalies, 16% had GDM, 2% had Rh isoimmunisation and 2% chorioangioma of the placenta.

CHART 9

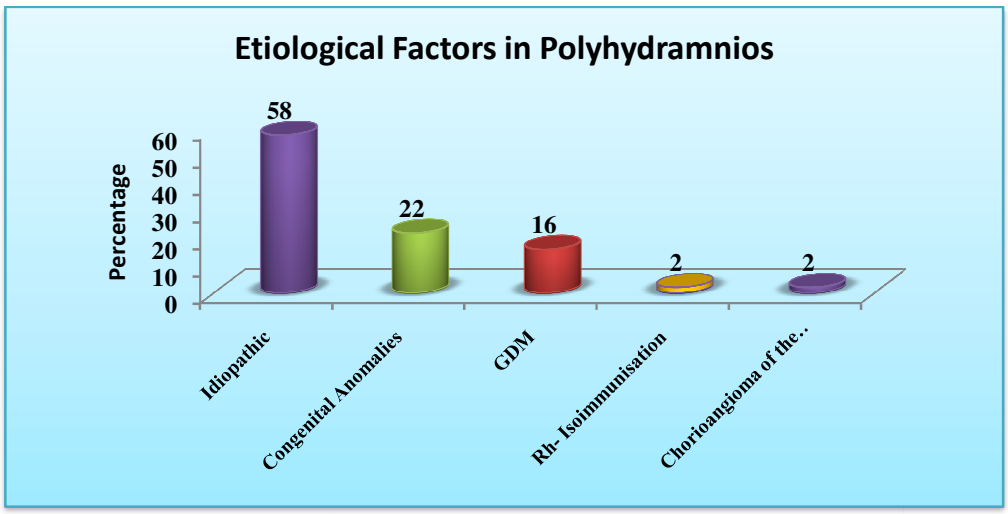

Etiological Factors in Polyhydramnios
Percentage
60
50
40
30
20
10
0
58
22
16
2
2
Idiopathic
Congenital Anomalies
GDM
Rh- Isoimmunisation
Chorioangioma of the..

TABLE -10

SEVERITY OF POLYHYDRAMNIOS

TYPES (AFI)	NO OF CASES N-50	%
Mild (25-30)	38	76
Moderate (31-35)	9	18
Severe (>35)	3	6

This table shows the severity of polyhydramnios. 76% were in mild polyhydramnios, 18% were moderate and 6% were in severe polyhydramnios group.

CHART 10

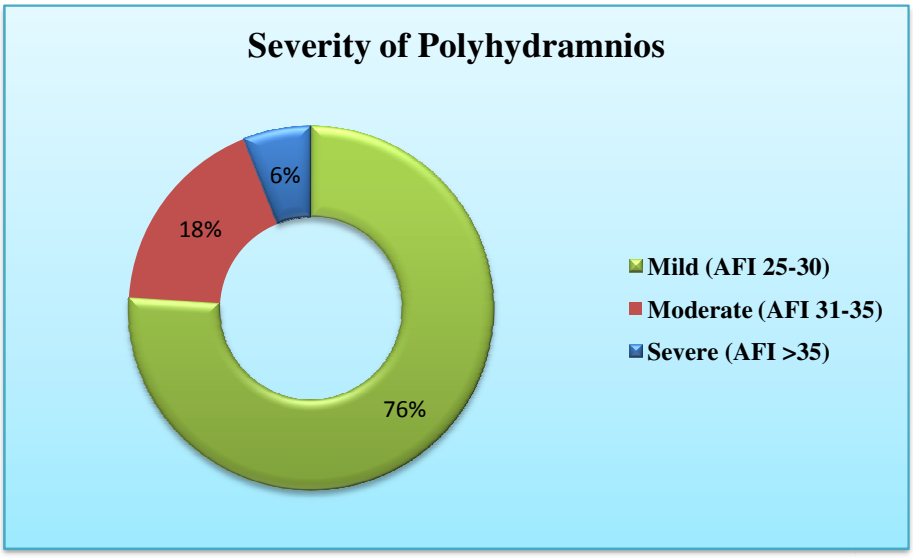


TABLE -11

ONSET OF LABOUR IN ABNORMAL LIQUOR VOLUME

ONSET OF LABOUR	GROUP			
	AFI ≤ 5		AFI ≥ 25	
	N- 150	%	N-50	%
Spontaneous	52	34.67	40	80
Induced	98	65.33	10	20

This table shows the onset of labour in abnormal liquor volume. Among the oligohydramnios group, 65.33% were induced compared to 20% in polyhydramnios group, which was highly significant with p value of 0.0001.

CHART 11

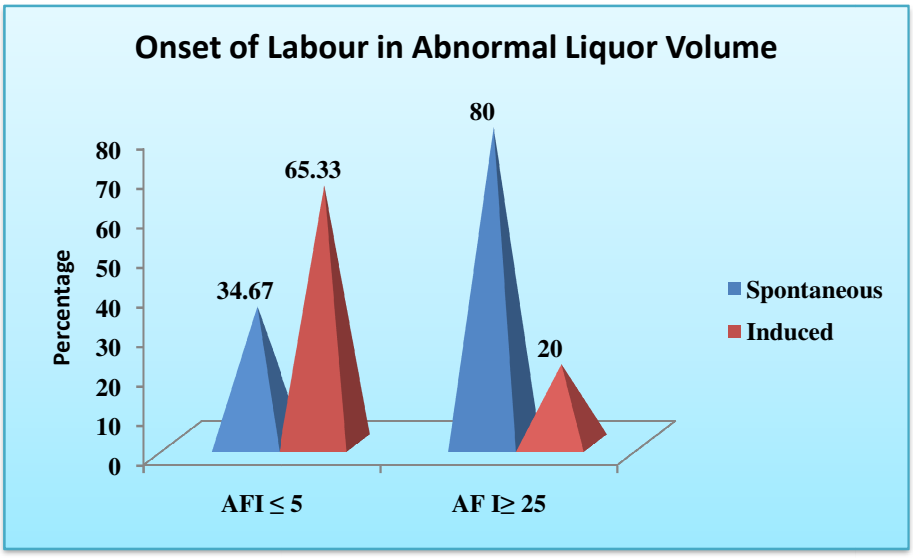


TABLE-12

MODE OF DELIVERY

MODE OF DELIVERY	GROUP			
	AFI ≤ 5		AFI ≥ 25	
	N- 150	%	N-50	%
SPVD	45	34	36	72
Instrumental delivery	10	6.66	5	10
cesarean	89	59.33	9	18

This table shows mode of delivery in abnormal liquor volume. In oligohydramnios group 59.33% were underwent cesarean section compared 18% in polyhydramnios group which is statistically significant with p value of 0.000. In polyhydramnios group 82% delivered vaginally compared to 40.66% in oligohydramnios group which is also statistically significant.

CHART 12

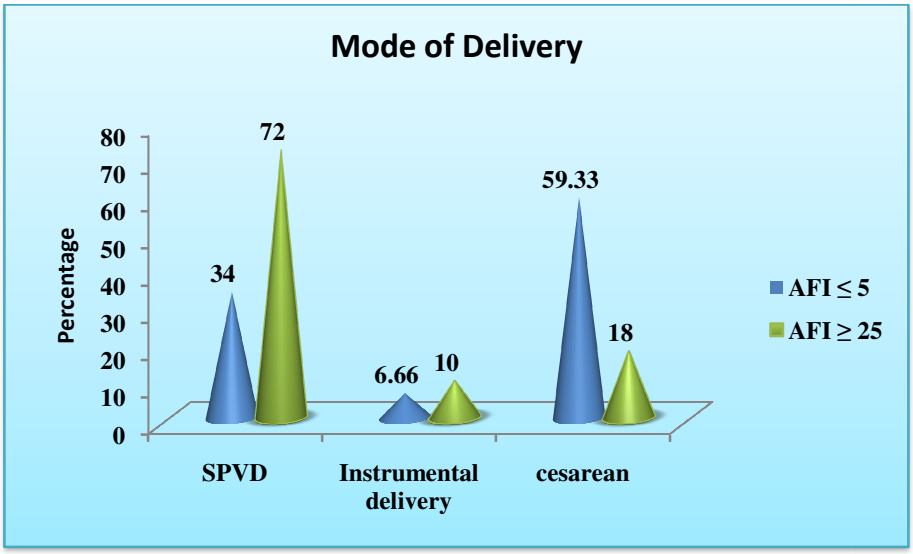

Mode of Delivery
Percentage
80
70
60
50
40
30
20
10
0
34
72
6.66
10
59.33
18
SPVD
Instrumental delivery
cesarean
AFI ≤ 5
AFI ≥ 25

TABLE-13

INDICATIONS OF CESAREAN SECTION

INDICATIONS	GROUP			
	AFI ≤ 5		AFI ≥ 25	
	N-89	%	N-9	%
Fetal Distress	68	76.40	2	22.22
CPD	10	11.23	3	33.33
Failed Induction	8	8.98	1	11.11
Mal presentation	3	3.37	2	22.22
Cord Prolapse	-	-	1	11.11

Among the indications for cesarean section in abnormal liquor volume, 76.40% were due to fetal distress, 11.23% due to CPD, 8.98% due to failed induction, 3.37% due to malpresentation in oligohydramnios group. In polyhydramnios group, 33.33% were due to CPD, 22.22% were due to malpresentation and fetal distress, 11.11% were due to cord prolapsed and failed induction.

CHART 13

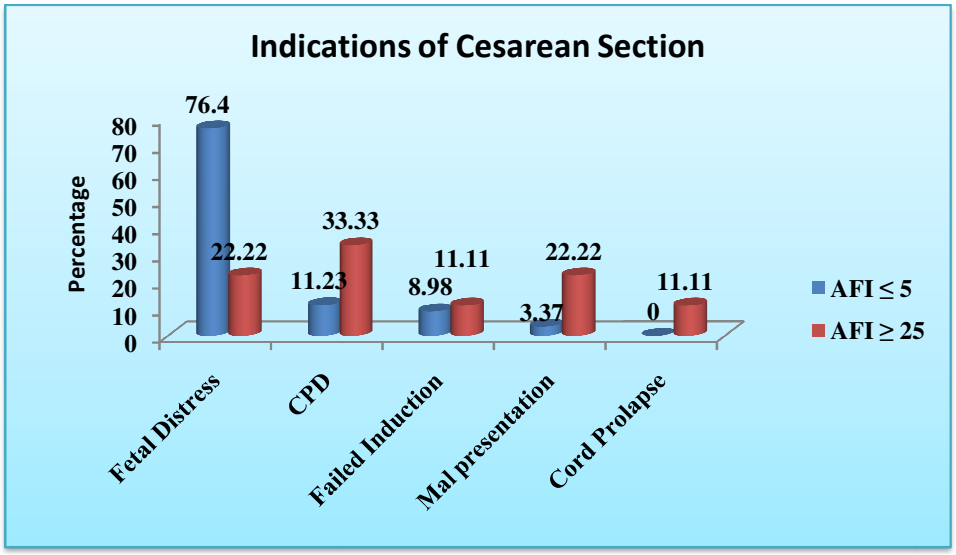

Indications of Cesarean Section
Percentage
80
70
60
50
40
30
20
10
0
76.4
22.22
11.23
33.33
8.98
11.11
3.37
22.22
0
11.11
Fetal Distress
CPD
Failed Induction
Mal presentation
Cord Prolapse
AFI ≤ 5
AFI ≥ 25

TABLE -14

COLOUR OF LIQUOR IN ABNORMAL LIQUOR VOLUME

COLOUR OF LIQUOR	GROUP			
	AFI ≤ 5		AFI ≥ 25	
	N- 150	%	N-50	%
Clear	64	42.66	44	88
Meconium	86	57.33	6	12

This table shows the colour of liquor in Abnormal liquor volume. 57.33% had meconium stained liquor in oligohydramnios group compared to 12% in polyhydramnios group, which is statistically significant with p value 0.000.

CHART 14

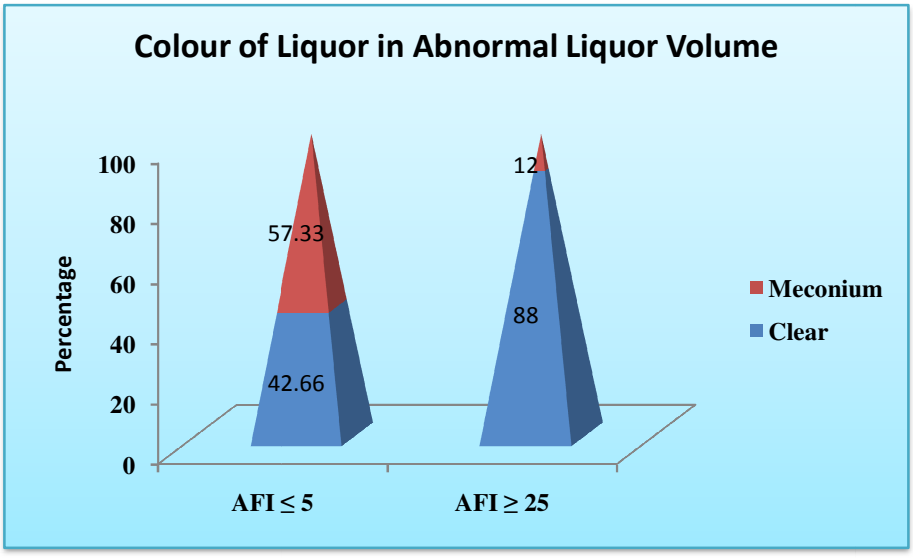


TABLE - 15

LABOUR COMPLICATIONS IN POLYHYDRAMNIOS

LABOUR COMPLICATIONS	AFI ≥ 25 NO OF CASES N-50	%
PROM	10	20
Cord Prolapse	3	6
Atonic PPH	2	4
Retained Placenta	1	2
Preterm Labour	7	14

This table shows intrapartum and postpartum complications in Polyhydramnios cases. Among which 20% were PROM , 14% Preterm labour, 6% Cord Prolapse, 4% Atonic PPH, 2% Retained Placenta.

CHART - 15

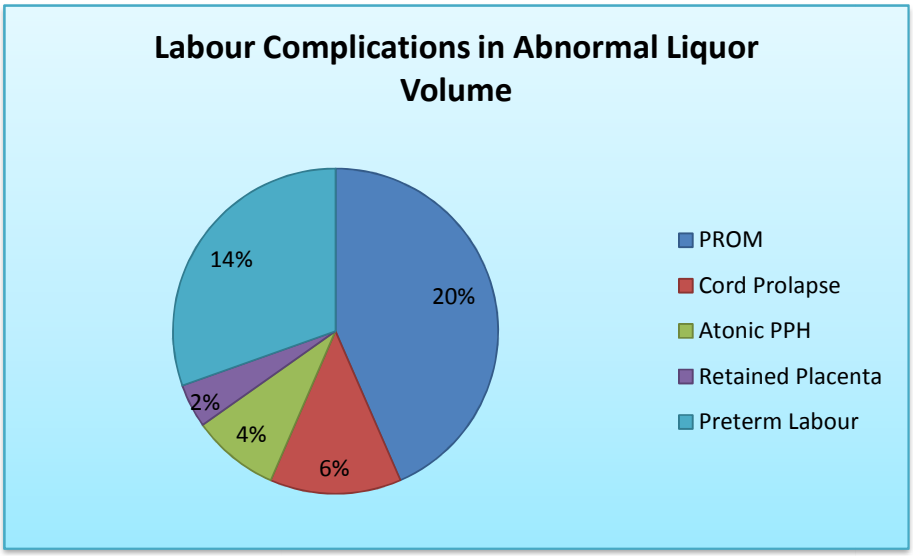


TABLE -16

FETAL OUTCOME IN ABNORMAL LIQUOR VOLUME

FETAL OUTCOME	GROUP			
	AFI ≤ 5		AFI ≥ 25	
	N- 150	%	N-50	%
Alive	139	92.7	36	72
Perinatal Death	11	7.3	14	28

This table shows Fetal Outcome in pregnancies with Abnormal Liquor Volume. In Oligohydramnios group 92.70% were alive compared to 72% in polyhydramnios group. Perinatal death was 28% in polyhydramnios group compared to 7.3% in oligohydramnios group which is statistically significant p value of 0.0001.

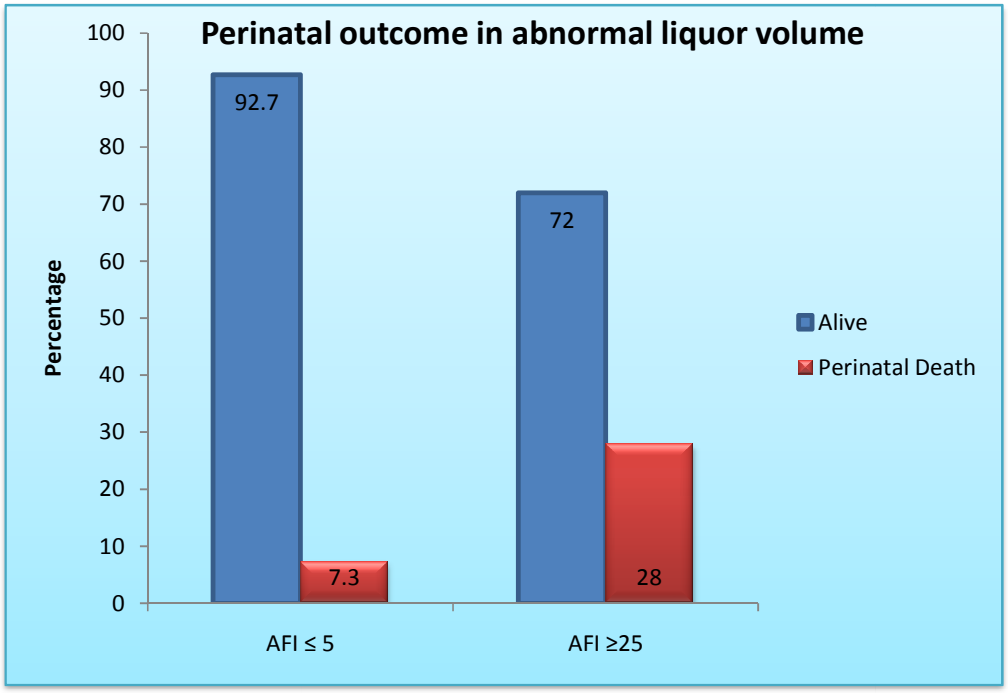

Perinatal outcome in abnormal liquor volume
100
90
80
70
60
50
40
30
20
10
0
Percentage
92.7
7.3
72
28
AFI ≤ 5
AFI ≥25
Alive
Perinatal Death

TABLE -17

5 MINUTES APGAR IN ABNORMAL LIQUOR VOLUME

5 MINUTES APGAR	GROUP			
	AFI ≤ 5		AFI ≥ 25	
	N- 150	%	N-39	%
<7	58	38.66	11	28.20
≥7	92	61.33	28	71.79

This table shows the 5 minutes apgar status in abnormal liquor volume. 5 minutes apgar score in oligohydramnios group was <7 in 38.66% compared to 28.20% in polyhydramnios group which is not statistically significant.

TABLE -18

BIRTH WEIGHT IN ABNORMAL LIQUOR VOLUME

BIRTH WEIGHT IN kg	GROUP			
	AFI ≤ 5		AFI ≥ 25	
	N- 150	%	N-39	%
≤ 2.5	81	54	6	15.38
2.6-3	45	30	10	25.64
3.1-3.5	19	12.66	15	38.46
3.6-4	5	3.33	5	12.82
> 4	-	-	3	7.69

This table shows the birth weight distribution in abnormal liquor volume.In oligohydramnios group, 54% were ≤ 2.5 kg, 45.99% were between 2.6-4 kg, no babies were born above 4 kg. In contrast polyhydramnios group delivered 76.92% babies with birth weight between 2.6-4 kg, 7.69% with >4 kg and 15.38% with ≤2.5kg.

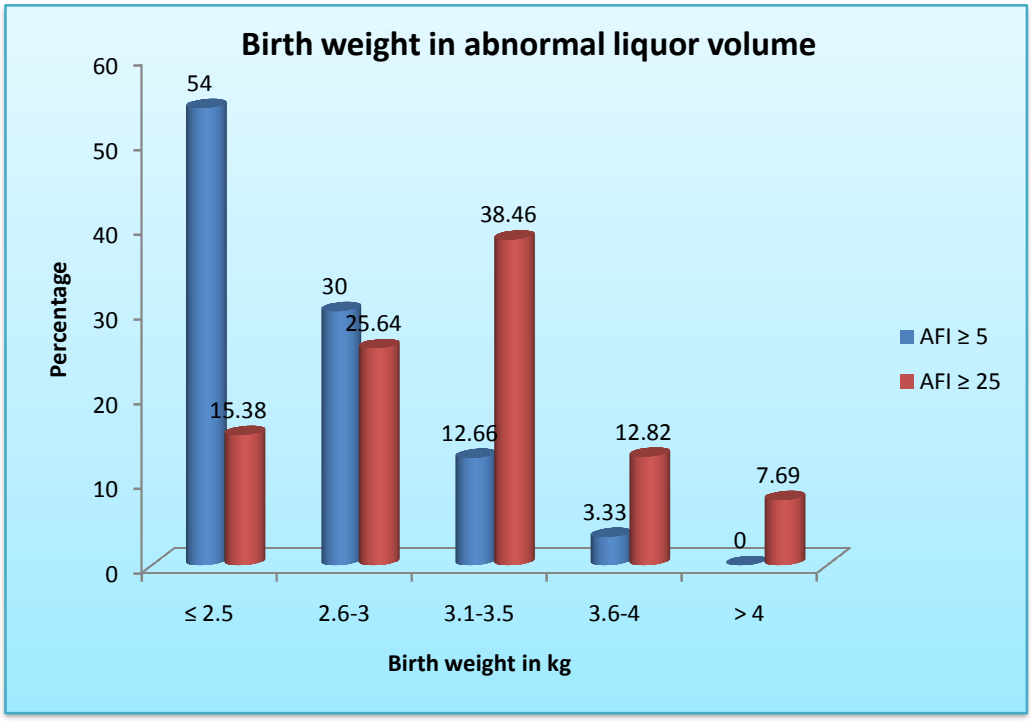

Birth weight in abnormal liquor volume
Percentage
60
50
40
30
20
10
0
54
15.38
30
25.64
12.66
38.46
3.33
12.82
0
7.69
≤ 2.5
2.6-3
3.1-3.5
3.6-4
> 4
Birth weight in kg
AFI ≥ 5
AFI ≥ 25

TABLE -19

IUGR IN ABNORMAL LIQUOR VOLUME

	GROUP			
	AFI ≤ 5		AFI ≥ 25	
	N- 150	%	N-50	%
IUGR	28	18.66	-	-

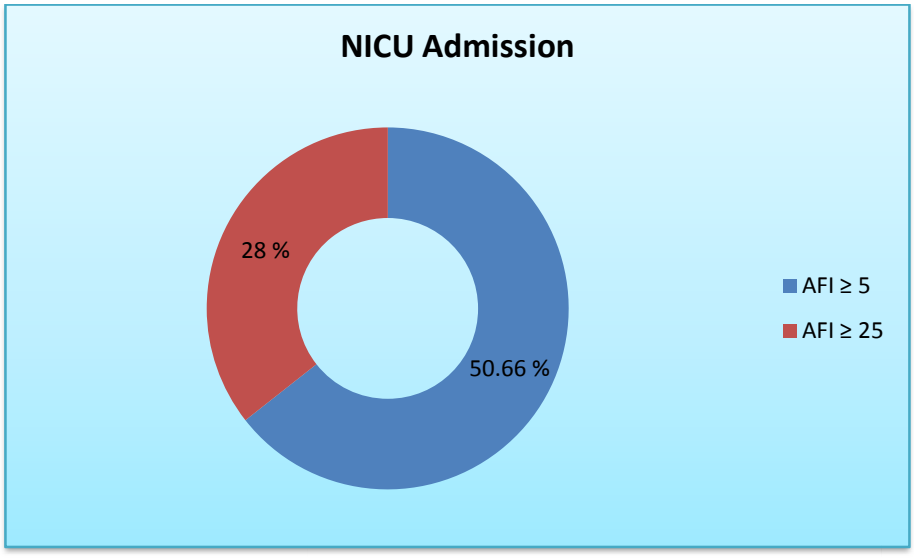
According to this table, 18.66% of babies were IUGR in oligohydramnios group. No cases of IUGR was present in polyhydramnios group.

TABLE -20

NICU ADMISSION IN ABNORMAL VOLUME

	GROUP			
	AFI ≤ 5		AFI ≥ 25	
	N- 150	%	N-39	%
NICU Admission	76	50.66	14	28

This table shows the Number of NICU admission in abnormal liquor volume. Compared to polyhydramnios group , 50.66% of babies were admitted in NICU in oligohydramnios group which is statistically significant with p value of 0.0001.



DISCUSSION

Various studies have been presented to know the perinatal morbidity and mortality in pregnancy with abnormal liquor volume. In the same way our study was tried to reveal the fetomaternal outcome in abnormal liquor volume in our Institute of Obstetrics and Gynaecology, Egmore, Chennai.

In our study, 57.3% were in the age of 20-25 yrs in oligohydramnios group, 42% were in the age of 26-30 yrs in polyhydramnios group. This is comparable to Guin et al[4] study in 2011

In our study, among the parity distribution, 66% of the cases in oligohydramnios group were primigravida, but there was no significant relation of age and parity with oligohydramnios according to the study done by Casey et a[31,] Chauhan et al[32,33], Magann et al[15]. In polyhydramnios group majority of the women were multigravida which is comparable to study by Guin et al[4] .

In our study, majority of the women in the gestation age of >37 weeks in both oligo and polyhydramnios group.

Maternal conditions associated with abnormal liquor volume:

Oligohydramnios:

In our study, 28.67% were postdated pregnancy as compared to 10.7% in Guin et al[4] study.

In our study, 17.34% were hypertensive disease of pregnancy as compared to 38.46% in Chandra et al[34] study, 3.5% in Guin et al[4] study and 8% in Preshit et al[35] study.

In our study, anemia were present in 3.33% of cases. APLA were present in 1.33%.

Polyhydramnios:

In our study, GDM were present in 16% as compared to Guin et al[4] study where 20% cases were GDM and 5% cases were GDM in Vaid et al[36] study.

In our study, 10% cases were hypertensive diseases as compared to 17.7% in Guin et al[4] study and 13% in Vaid et al[36] study.

In our study, 2% cases were Rh negative pregnancy as compared to Guin et al[4] where Rh -ve pregnancy were 4.4% , 1% in Lyndon M Hill et al[37] study.

In our study, 2% cases were chorioangioma of the placenta

Congenital anomalies in abnormal liquor volume:

Oligohydramnios

In our study, total of 4% cases had congenital anomalies as compared to 12.9% in Guin et al study, 5.8% in Anil Shetty et al[38].

In our study, infantile PCKD were 2% as compared to Guin et al[4] study where 7.5% were PCKD. In our study 0.67% were MCKD, single umbilical artery and microcephaly.

Polyhydramnios.

In our study, total of 22% had congenital anomalies which was comparable to Guin et al[4] study where 31.1% were associated with congenital anomalies.

In our study, anencephaly was the common anomaly that account for 8% as compared to 6% in Guin et al[4] study and 65.96% in Vaid et al[36] study.

Spina bifida were present in 4% cases in our study as compared to 4% in Guin et al[4] study.

Hydrocephalus with meningomyelocele were in 4% cases in our study as compared to Guin et al[4] study where 10% cases were hydrocephalus, 10.63% in Vaid[36] et al study.

Diaphragmatic hernia were present in 2% in our study, Duodenal atresia were 2% in our study as compared 4% in Guin et al[4] study.

In our study, Non immune hydrops were present in 2% cases as compared to 7% in Nicole Damato et al[39] study.

Etiological factors in abnormal liquor volume:

Oligohydramnios

In our study, 37.33% were isolated oligohydramnios with no identifiable cause as compared to 52% in Krishna jagatia et al.

In our study, 28.67% were postdated pregnancy as compared to 10.7% in Guin et al[4] study.

In our study, 17.34% were hypertensive disease of pregnancy as compared to 38.46% in Chandra et al[34] study, 3.5% in Guin et al[4] study and 8% in Preshit chate et al[35] study.

In our study, 12.66% cases were IUGR which was comparable with Guin et al[4] study of 14.2% and 25% in Anil Shetty et al[38] study.

In our study, 4% cases were congenital anomalies as compared to 12.9% in Guin et al[4] study, 5.8% in Anil Shetty et al[38] study.

Polyhydramnios:

In our study, exact cause of polyhydramnios were not detected in 58% which is comparable with Brady et al[40] study.

In our study, total of 22% had congenital anomalies which was comparable to Guin et al[4] study where 31.1% were associated with congenital anomalies.

16% had GDM in our study which was comparable to 20% in Guin et al[4] study.

In our study, 2% had Rh isoimmunisation as compared to 4.4% in Guin et al[4] study, 1% in Lyndom M Hill et al[27].

In our study, chorioangioma of the placenta were present in 2% of cases.

Severity of polyhydramnios:

In our study , 76%, 18%, 6% patients were mild, moderate and severe polyhydramnios respectively, which was comparable to Lyndon M. Hill et al[37] where 77.4%, 18.6%, 4% were mild ,moderate and severe

polyhydramnios respectively. Majority of mild polyhydramnios were detected at term in our study.

In our study, induction rate was higher in oligohydramnios group of about 65.33% as compared to Guin et al[4] where 56.5% cases were induced.

In polyhydramnios group only 20% cases were induced as compared to 13.6% in Guin et al[4] study.

The rate of cesarean section was 59.33% in oligohydramnios group which is comparable to 42.8% in Guin et al[4] study, 64% in Preshit chate et al[35] and 76.92% in Chandra P et al[34] study. This increased rate of cesarean was due to fetal distress. In our study 76.4% cases had fetal distress which in turn due to increased meconium stained liquor (57.33%) and IUGR. (18.66%) in our study.

In our study, 38.66% of babies had low 5 minute APGAR score in oligohydramnios group as compared to 23.7% in Chandra et al[34], 16% in Preshit chate et al[35].

In polyhydramnios group only 18% underwent cesarean section as compared to 22.2% in Guin et al[4] . Instrumental delivery rate was not significant in both oligo and polyhydramnios group in our study.

In our study, with polyhydramnios group, 20% cases were PROM as compared to 44.5% in Guin et al[4], 14% were preterm labour which was comparable to 40% in Guin et al[4], 6% had cord prolapse as compared to 4.4% in Guin et al[4] study.

In our study, atonic PPH were occurred in 4% of cases as compared to 4.4% in Guin et al[4] study. Retained placenta were seen in 2% cases in our study.

In our study, polyhydramnios group had high perinatal mortality rate of 28% as compared to oligohydramnios group which is 7.3%. This was comparable with Guin et al[4] study where perinatal mortality were 42.25% in polyhydramnios and 12.9% in oligohydramnios group. This high perinatal mortality in polyhydramnios group was due to increased fatal congenital anomalies.

In our study, with oligohydramnios group, 54% were $\leq$ 2.5 kg as compared to Chandra et al[34] and Preshit Chate et al[35], 45.99% were between 2.6-4 kg, No babies were born above 4 kg.

In contrast polyhydramnios group delivered 76.92% babies with birth weight between 2.6-4 kg, 7.69% with >4 kg and 15.38% with $\leq$2.5kg in our study.

In our study, 50.66% babies were admitted in NICU in oligohydramnios group which is comparable to 46.15% in Chandra et al[34] and 42% in Preshit Chate et al[36] study. In polyhydramnios group 28% of babies were admitted in NICU.

SUMMARY

1. Majority of the oligohydramnios cases were primigavida and polyhydramnios cases were multigravida.
2. Most of the patients with abnormal liquor volume were diagnosed at term.
3. Mild polyhydramnios was the most common type.
4. Isolated oligohydramnios (37.33%) was the most common cause followed by post dated pregnancy (28.67%) and third being the hypertensive diseases of pregnancy(17.34%) in oligohydramnios group.
5. Idiopathic polyhydramnios (58%) were the first common cause of polyhydramnios, the second were congenital anomalies(22%) and the GDM (16%) was the third one .
6. Incidence of congenital anomalies were high in polyhydramnios (22%) than in oligohydramnios (4%).
7. Most common congenital anomaly in oligohydramnios group were Infantile polycystic kidney disease. In polyhydramnios group, anencephaly was the most common anomaly followed by spina bifida, hydrocephalus with meningomyelocele.

8. Induction of labour was high in oligohydramnios group (65.33%) than in polyhydramios (20%) group.
9. 59.33% were underwent cesarean section in oligohydramnios group compared to 18% in polyhydramnios group.
10. Fetal distress (76.4%) was the leading cause of cesarean in oligohydramnios , CPD (33.33%) was the common cause in polyhydramnios group.
11. There is No significant difference in instrumental delivery in both groups
12. Meconium stained liquor were common in oligohydramnios group.
13. In polyhydramnios group, the major labour complications were PROM and preterm labour.
14. No maternal mortality was present in our study.
15. In oligohydramnios group, the alive babies rate were 92.7% and perinatal death was 7.3%. In polyhydramnios group, the alive and perinatal death rate were 72%, and 28% respectively.
16. In the oligohydramnios group, congenital anomaly was not the cause of any perinatal death but in polyhydramnios group majority of the death were due to lethal congenital anomalies.
17. Birth weight <2.5 kg were high in oligohydramnios group (54%).

18. IUGR cases were common in oligohydramnios group.

19. NICU admission was highest in oligohydramnios group (50.66%) than polyhydramnios (28%)group.

CONCLUSION

- Development of abnormal liquor volume during pregnancy signals danger to the foetus. It is associated with an increased incidence of caesarean section, labour complications and adverse perinatal outcome.
- But isolated oligo and polyhydramnios in term gestation has better perinatal outcome compared to early onset and with associated conditions like hypertensive diseases of pregnancy, GDM, IUGR.
- Ultrasound is the ideal method to detect any abnormality in liquor volume. If any abnormality is detected during the ultrasound, a careful search should be made to detect any identifiable congenital anomalies.
- A detailed history, clinical examination and relevant investigations should be done to identify the various etiological factors in all cases of abnormal liquor volume, to get better foetal outcome as well as to avoid the maternal complications.

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PROFORMA

NAME: **AGE:** **IP NO:**

UNIT:

ADDRESS: **DOA:**

SOCIO ECONOMIC STATUS:

BOOKED/UNBOOKED:

LMP:

EDD:

OBSTETRIC FORMULA : **G P L A**

PRESENTING COMPLAINTS:

H/O Amenorrhea

H/O Bleeding pv/ Draining pv

H/O Perception of fetal movements.

PRESENT OBSTETRIC HISTORY:

PAST OBSTETRIC HISTORY :

MENSTRUAL HISTORY:

Age at Menarche:

Menstrual Cycles:

MARITAL HISTORY:

Married Since:

Consanguineous/ Non Consanguineous

PAST HISTORY:

History of DM / HT / TB / Rheumatic Heart Disease / Epilepsy / Thyroid / Bronchial asthma

FAMILY HISTORY:

PERSONAL HISTORY: Diet, Sleep, Bladder & Bowel habits

GENERAL EXAMINATION:

HT: WT: BMI:

TEMP: BP: PR: RR:

PALLOR / ICTERUS / PEDAL EDEMA

CVS: BREAST:

RS: THYROID:

PER ABDOMEN:

Size of Uterus (In Weeks):

Acting/Not Acting:

Presenting Part:

Engaged/ Unengaged:

FHR:

Amount of liquor

PER VAGINAL EXAMINATION:

INVESTIGATIONS:

- URINE INVESTIGATIONS**:**
  Urine albumin: Sugar: Deposits:

- BLOOD INVESTIGATIONS**:**
  Blood grouping and typing

  HIV & VDRL

  HB%

  Random blood sugar

  OGCT

  Admission CTG

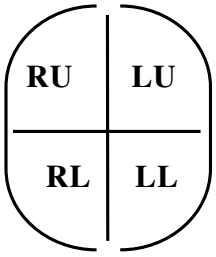
ULTRASOUND OBSTETRICS WITH DOPPLER:

Presentation

GA

Placenta

AFI



Congenital Anomalies if any

DETAILS OF DELIVERY:

Mode of delivery: Spontaneous/ Induced

Labour natural:

Operative vaginal Delivery : Vaccum / Forceps

Indication

LSCS : Indication

LIQUOR : Scanty / Average / Excess

Meconium Stained / Clear

BABY DETAILS :

Live / still born

Term / Preterm / IUGR /SGA / LGA

Date and time of birth

Birth weight

Apgar

Any congenital anomalies

NICU Admission**:** Yes / No

Cause for admission

No of days in NICU

Iv antibiotics: Yes / No

CXR: Yes / No

PERINATAL MORTALITY (if any)**:**

NEONATAL OUTCOME: Healthy / Neonatal death

COMPLICATIONS DURING DELIVERY:

MATERNAL :

Uterine inertia

APH

PPH

Preterm labour

PROM

Cord prolapse

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI –3

EC RegNo:ECR/270/Inst./TN/2013 Telephone No : 04425305301
Fax : 044 25363970
Date:10.09.2013

CERTIFICATE OF APPROVAL

To
Dr.J.Punithavathi,
MD OG post Graduate,
Institute of Obstetrics and Gynaecology, Egmore,
Chennai - 8

Dear Dr.J.Punithavathi,

The institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "A clinical study of feto maternal outcome in pregnancies with abnormal liquor volume" No.02092013.

The following members of Ethics Committee were present in the meeting held on 10.09.2013 conducted at Madras Medical College, Chennai -3.

1. Dr.G.SivaKumar, MS FICS FAIS --- Chairperson
2. Prof. R. Nandhini MD -- Member Secretary
   Director, Instt. of Pharmacology ,MMC, Ch-3
3. Prof. Shyamraj MD -- Member
   Director i/c , Instt. of Biochemistry , MMC, Ch-3
4. Prof. P. Karkuzhali. MD -- Member
   Prof., Instt. of Pathology, MMC, Ch-3
5. Prof. Kalai Selvi -- Member
   Prof of Pharmacology, MMC, Ch-3
6. Prof. Siva Subramanian, -- Member
   Director, Instt. of Internal Medicine, MMC, Ch-3
7. Thiru. S. Govindsamy. BABL -- Lawyer
8. Tmt. Arnold Saulina MA MSW -- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

R Nandini

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

PATIENT CONSENT FORM

Title of the Project

A CLINICAL STUDY OF FETO-MATERNAL OUTCOME IN PREGNANCIES WITH ABNORMAL LIQUOR VOLUME

Institution : **INSTITUTE OF OBSTETRICS & GYNAECOLOGY,**
Egmore, Chennai-600008.

Name : Date :

Age : IP No. :

Sex : Project Patient No. :

The details of the study have been provided to me in writing and explained to me in my own language.

I confirm that I have understood the above study and had the opportunity to ask questions.

I understood that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without the medical care that will normally be provided by the hospital being affected.

I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).

I have been given an information sheet giving details of the study.

I fully consent to participate in the above study regarding abnormal liquor volume

_____ _____ _____

Name of the subject Signature Date

INFORMATION TO PARTICIPANTS

Title : **A CLINICAL STUDY OF FETO-MATERNAL OUTCOME IN PREGNANCIES WITH ABNORMAL LIQUOR VOLUME**

Principal Investigator : **Dr.PUNITHAVATHI.J**

Name of Participant :

Site : Institute of obstetrics and gynaecology

You are invited to take part in this study. The information in this document is meant to help you decide whether or not to take part. Please feel free to ask if you have any queries or concerns.

WHAT IS THE PURPOSE OF RESEARCH?

An increased (or) decreased liquor volume, in pregnancies between 28-42 weeks of gestation, is associated with an increased incidence of complication in labour, cesarean section and adverse perinatal outcome. The purpose of this study is to evaluate the obstetrics outcome and to find the cause wherever is possible in pregnancies with abnormal liquor volume.

THE STUDY DESIGN

Pregnant women admitted at IOG, Egmore with abnormal liquor volume (Oligohydromnios or polyhydromnios) or diagnosed during their stay at IOG are followedup and their pregnancy outcome evaluated. They are subjected to Ultrasound Examination and necessary routine investigations are done.

STUDY PROCEDURES

Pregnant women with gestational age between 28-42 weeks with abnormal liquor volume are subjected to ultrasound examination. Necessary routine investigations are done to find the cause. The effect of abnormal liquor volume in labour (increased incidence of caesarian section, instrumental deliveries) and perinatal outcome is evaluated.

Possible risks to you – Nil

Possible benefits to you - you will be under regular follow up till discharge.

Possible benefits to other people

The results of the research may provide information regarding the factors associated with abnormal liquor volume and we can limit the serious maternal and fetal complication.

தகவல் படிவம்

தலைப்பு	:	**கர்ப்பக் காலத்தில் பனிக்குட நீரின் அளவு வழக்கத்திற்கு மாறான நிலையில் கூடுதலாகவோ அல்லது குறைவாகவோ இருப்பதனால் தாய்க்கும், சேய்க்கும் ஏற்படும் பாதிப்பு பற்றிய ஆய்வு.**
முக்கிய ஆராய்ச்சியாளர்	:	**மரு. புனிதவதி. J.**
பங்கேற்பாளர்	:	
இடம்	:	**தாய் சேய் நல மருத்துவமனை, எழும்பூர்.**

இந்த ஆவணம், நீங்கள் ஆய்வில் பங்கேற்கலாமா அல்லது வேண்டாமா என்பதை முடிவு செய்வதற்கு உதவும். உங்களுக்கு ஏதேனும் கேள்விகள் அல்லது கருத்துக்கள் இருந்தால் தயங்காமல் கேட்கலாம்.

ஆய்வின் நோக்கம்:

28-42 வாரத்திற்குள் இருக்கும் கர்ப்பிணிகளுக்கு பனிக்குட நீரின் அளவு வழக்கத்திற்கு மாறான நிலையில் கூடுதலாகவோ அல்லது குறைவாகவோ இருப்பதனால் தாய்க்கும், சேய்க்கும் ஏற்படும் பாதிப்பு பற்றியும், அதற்கான காரணத்தை பற்றியும் கண்டறிவதே இந்த ஆராய்ச்சியின் நோக்கமாகும்.

ஆய்வின் நடைமுறைகள்:

இந்த ஆய்வில் எங்கள் மருத்துவமனையின் உள்நோயாளியாக அனுமதிக்கப்பட்டு இருக்கும் கர்ப்பிணிகளின் பனிக்குட நீரின் அளவு அல்ட்ரா சவுண்ட் ஸ்கேன் கருவி மூலம் பரிசோதிக்கப்படும் அதில் பனிக்குட நீர் அளவு அதிகமாகவோ அல்லது குறைவாகவோ உள்ளவர்கள் இந்த ஆய்வில் பங்கேற்பார்கள். அதற்கான காரணத்தை அறிய தேவையான ஆய்வக பரிசோதனை மேற்கொள்ளப்படும். இவ்வாறு பனிக்குட நீரின் அளவு வழக்கத்திற்கு மாறாக அதிகமாகவோ அல்லது குறைவாகவோ இருப்பதினால் கர்ப்பகாலத்தின் போதும், பிரசவத்தின் போதும் மற்றும் பிறக்கும் குழந்தைக்கு ஏற்படும் பாதிப்புகள் பற்றி ஆராயப்படும்.

மேலும் உங்களது நோயின் தன்மை ஆய்வு செய்யப்படும். நீங்கள் மருத்துவமனைக்கு மீண்டும் வரவேண்டி இருக்கலாம்.

ஆய்வினால் தங்களுக்கு ஏற்படும் கெடுதல்கள்: ஒன்றுமில்லை

ஆய்வினால் தங்களுக்கு ஏற்படும் பலன்கள்: நீங்கள் எங்களது தொடர் கண்காணிப்பில் இருப்பதால் உங்களுக்கு பலன்கள் ஏற்படும்.

ஆய்வினால் மற்றவருக்கு ஏற்படும் பலன்கள்: இந்த ஆய்வின் முடிவுகளால் மருத்துவ அறிவை வலப்படுத்திக் கொள்ளவும், மேலும் பல நோயாளிகளுக்கு உதவியாகவும் அமையும்.

தங்களை பற்றிய தகவல்கள் மற்றும் அடையாளங்களையோ வெளியிடமாட்டோம் என்பதை தெரிவித்துக் கொள்கிறோம். இந்த ஒப்புதல் படிவத்தினை கையெழுத்திடுவதனால் தங்களை பற்றிய தகவல்களை ஆராய்ச்சியாளர்கள் மற்றும் அது சம்பந்தப்பட்டவர்கள், துறை சார்ந்த நெறிமுறை குழு உறுப்பினர்கள் ஆகியோர் தேவைப்பட்டால் தெரிந்து கொள்வதற்கு சம்மதிக்கிறீர்கள். இந்த ஆய்வின் தகவல்கள் விஞ்ஞான புத்தகங்களிலோ அல்லது கருத்தரங்களிலோ வெளியிடப்பட்டால் அதில் தங்களது அடையாளங்கள் வெளியிடப்படமாட்டாது.

இந்த ஆய்வில் தாங்கள் கலந்துக்கொள்ளாமை, தங்களது மருத்துவ கவனிப்பையோ அல்லது இந்த ஆய்வு நடத்துபவரின் உறவையோ, நிறுவனத்தினரோடு உள்ள உறவையோ பாதிக்காது. நீங்கள் நன்றாகவே கவனிக்கப்படுவீர்கள் மற்றும் உங்களுக்கு கிடைக்கும் பலன்கள் பாதிக்கப்படாது.

நீங்கள் இந்த ஆய்வில் கலந்துக்கொள்வது முற்றிலும் உங்களது விருப்பத்தின் பேரில் தான். மேலும் நீங்கள் எந்நேரத்திலும் இந்த ஆய்விலிருந்து காரணம் ஏதுமின்றி விலகிக்கொள்ளலாம். ஆனால் நீங்கள் விலகுவதற்கான முடிவெடுக்கும் முன்பு இந்த ஆய்வு குழுவினரோடு பேசி முடிவெடுத்தல் நலம்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்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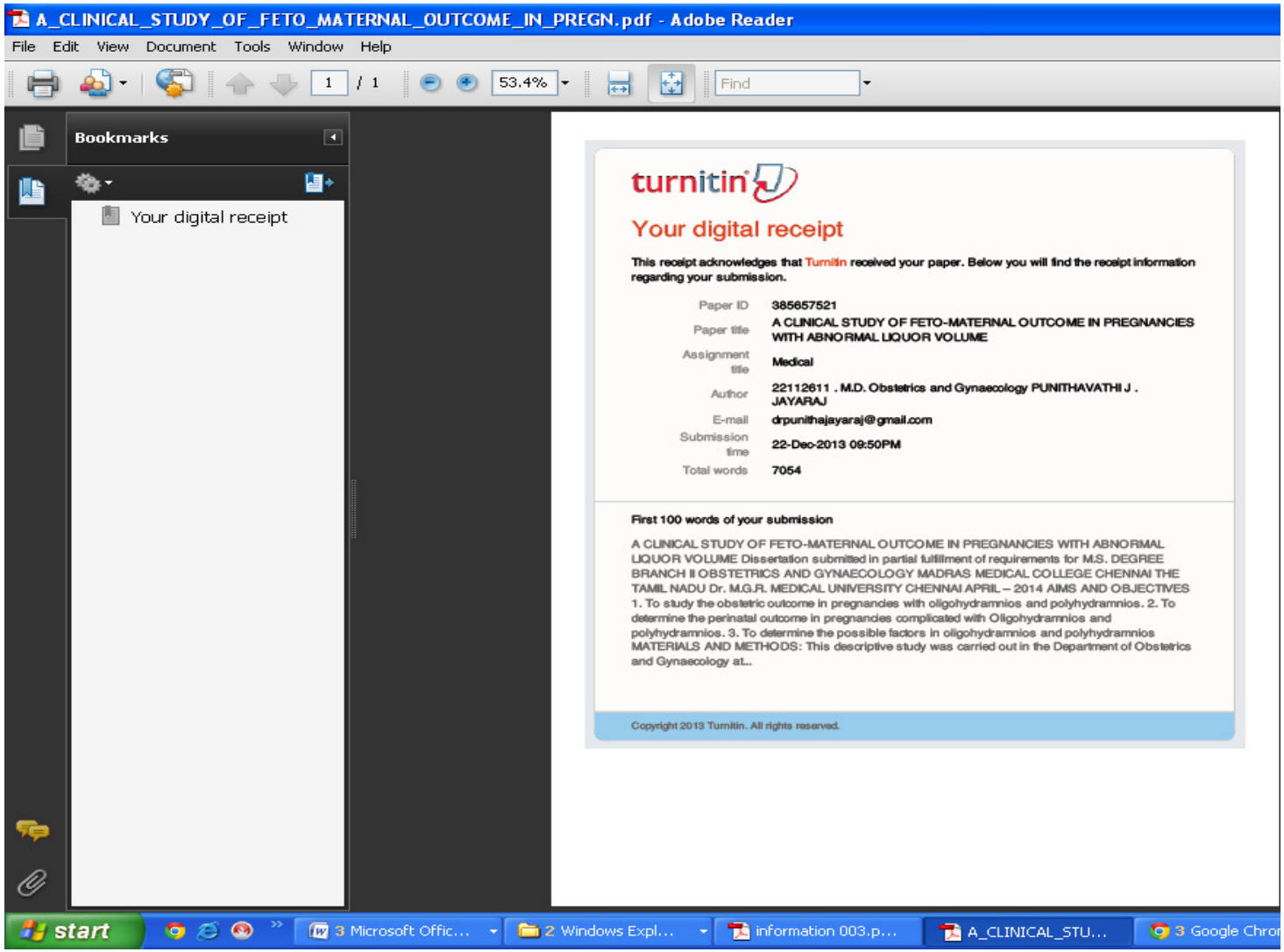
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A CLINICAL STUDY OF FETO-MATERNAL OUTCOME IN PREGNANCIES WITH ABNORMAL LIQUOR VOLUME Dissertation submitted in partial fulfillment of requirements for M.S. DEGREE BRANCH II OBSTETRICS AND GYNAECOLOGY MADRAS MEDICAL COLLEGE CHENNAI THE TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY CHENNAI APRIL – 2014 AIMS AND OBJECTIVES 1. To study the obstetric outcome in pregnancies with oligohydramnios and polyhydramnios. 2. To determine the perinatal outcome in pregnancies complicated with Oligohydramnios and polyhydramnios. 3. To determine the possible factors in oligohydramnios and polyhydramnios MATERIALS AND METHODS: This descriptive study was carried out in the Department of Obstetrics and Gynaecology at...
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OLIGO HYDRAMNIOS

S.NO	NAME	AGE	IP NO	OBSTETRIC SCORE	GESTATION AGE IN WEEKS	ASSOCIATED MATERNAL CONDITIONS	AFI	ANOMALIES NOTED IN USG	ETIOLOGICAL FACTORS	SPONTANEOUS/ INDUCED LABOUR	COLOUR OF THE LIQUOR	MODE OF DELIVERY	INDICATIONS FOR CESAREAN	LABOUR COMPLICATIONS	FETAL OUTCOME	APGAR AT 5 MINTS	BIRTH WEIGHT IN Kg	NICU ADMISSION	PERINATAL DEATH
1	Lavanya	25	28074	Primi	38	Anemia	4		Idiopathic	I	C	OUTLET			A/T AGA	>7	3.2		
2	Vijayalakshmi	22	27999	G2A1	38	PE	3.2		PE	S	C	CS	FD		A/T/SGA	<7	2.9	AD	
3	Selvi	31	28096	G3P2L2	30		3		Idiopathic	I	M	VD			A/PT/SGA	>7	2.4	AD	
4	Chandrakala	21	27200	G2P1L1	40+2		4.2		PD	I	C	OUTLET			A/T/AGA	>7	3.4		
5	Saranya	20	26344	Primi	32	GHT	5		GHT	S	C	VD			A/PT/AGA	<7	1.9	AD	
6	Chitrapriya	24	27976	G2P1L1	39		4		Idiopathic	S	C	VACCUM			A/T/SGA	<7	2.49	AD	
7	Yasmin fathima	21	28119	primi	35		3.4		Idiopathic	I	M	CS	FD		A/PT/AGA	>7	2.4		
8	sangeetha	28	28111	primi	31		NIL	B/L MCKD	Anomaly	I	M	VD			A/PTSGA	<7	1.4	AD	D
9	Sreelekha	29	28891	Primi	36		4.9		Idiopathic	S	M	VD			A/PT/SGA	<7	2.51		
10	Nandhini	23	28901	Primi	40+1		5		PD	I	C	CS	FI		A/T/SGA	<7	2.6		
11	suganya	19	28890	primi	31		2		Idiopathic	I	M	VD			A/PT/AGA	<7	1.2	AD	D
12	Saranya	25	28702	G3P1L1A1	40+3		3.6		PD	I	C	CS	FI		A/T/AGA	>7	3.17		
13	Padmavathi	28	27202	primi	40+2		2		PD	I	M	VD	FD		A/PT/AGA	<7	2.45	AD	

14	Sharmila	24	29920	G1P1L1	32		4.8		Idiopathic	I	M	CS	FD		A//T/AGA	<7	1.2	AD	
15	Fradeena robinson	21	23360	Primi	29	PE	1.9		PE	I	C	VD			A/PT/AGA	>7	1.1	AD	
16	Sudha	20	28101	Primi	40+2		3.7		PD	S	C	CS	FI		A/T/SGA	<7	2.6		
17	Devika	23	29792	G3A2	37		5		Idiopathic	S	C	VD	FI		A/T/SGA	>7	2.54		
18	Kavitha	24	28896	primi	38		4		Idiopathic	I	M	CS	FD		A/T/IUGR	<7	2.39		
19	Parveen banu	21	28890	primi	37		4.9		Idiopathic	S	M	CS	CPD		A/T/SGA	>7	2.5		
20	Sakila	30	30620	Primi	40	Anemia	5		IUGR	I	C	VD			A/T/IUGR	<7	2.23	AD	
21	Hemalatha	23	30596	G2P1L1	35		3.8		Idiopathic	I	M	CS	FI		A/PT/AGA	<7	2.9		
22	Kalaivani	25	30641	primi	39		4		Idiopathic	I	M	CS	FD		A/T/AGA	>7	3.5		
23	Aishwarya	27	31370	primi	36		2.9		Idiopathic	S	M	CS	Breech		A/PT/AGA	<7	2.19	AD	
24	Tamilselvi	24	31311	G2P1L0	40+1		3.8		PD	I	C	VD			A/T/AGA	>7	3.3		
25	Sasirekha	26	30598	Primi	40	PE	4.6		PE	I	M	CS	FD		A/T/SGA	<7	2.8		
26	Indira	23	29278	Primi	32		NIL		Idiopathic	I	M	CS	FD		A/PT/AGA	>7	2	AD	
27	Kuttiammal	32	29819	G3P2L1	40+3		4		PD	I	M	CS	CPD		A/T/AGA	<7	3.2		
28	Lavanya	23	31741	primi	39		5	Microcephaly	Anomaly	S	C	VD			A/T/SGA	>7	2.6	AD	
29	Arulmozhi	26	31963	primi	34		1.4		Idiopathic	S	M	CS	FD		A/PT/AGA	<7	2	AD	
30	Muniyammal	23	31227	Primi	40		2.9		Idiopathic	I	M	CS	FD		A/T/AGA	>7	2.6		
31	Thangam	25	32208	G2P1L1	40+3	Anemia	4.7		PD	I	M	CS	CPD		A/T/SGA	<7	2.49	AD	
32	Roopa	29	31381	primi	33		3.6	Infantile PCKD	Anomaly	I	C	VD			A/PT/SGA	>7	2.1	AD	
33	sangeetha	22	32223	primi	39		3.1		Idiopathic	S	M	CS	FD		A/T/SGA	<7	2.47	AD	
34	Nargis banu	28	32228	G2A1	39	PE	4.2		PE	I	M	CS	CPD		A/T/AGA	>7	3.4		
35	Anitha	21	32219	Primi	32		2.3		Idiopathic	I	M	CS	FD		A/PT/AGA	>7	1.4	AD	
36	sumathy	27	32828	Primi	40+2		4.6		PD	I	M	CS	FD		A/T/SGA	<7	2.7	AD	
37	Bhavani	25	30598	primi	29	PE	3.4		PE	S	C	VD			A/PT/SGA	<7	960 gms	AD	D
38	Ambika	24	31367	G2P1L0	37		4.5		Idiopathic	I	C	VD			A/T/AGA	>7	2.5		
39	Jayanthi	33	32210	Primi	38		2.5		Idiopathic	I	C	VD			A/T/SGA	>7	2.48		

40	Ammu	21	30596	Primi	40+2		5		PD	I	M	CS	FD		A/T/SGA	<7	2.61		
41	sarala	27	33509	G3A2	30	APLA	4.1		APLA	I	C	VD			A/PT/SGA	>7	1	AD	D
42	Anandhi	25	33831	primi	40+2		5		PD	S	M	CS	FI		A/T/SGA	<7	2.49		
43	kanniyammal	30	33818	G2P1L1	35	GHT	4.9		GHT	S	M	CS	FD		A/PT/AGA	>7	2.16	AD	
44	shilpa	20	34690	Primi	40+1		4		PD	I	C	CS	FD		A/T/SGA	<7	2.475	AD	
45	Revathy	25	34693	Primi	37		3.9		Idiopathic	I	M	CS	FD		A/T/AGA	>7	3		
46	Karpagam	26	35019	G2P1L0	40+1		3.2		PD	I	M	CS	FD		A/T/iUGR	>7	2.3	AD	
47	Jothi	22	35698	prim	36	PE	5		PE	S	C	VD			A/PT/SA	<7	2.29	AD	
48	Dhivya	26	34980	G3A2	40		4.8		Idiopathic	S	C	OUTLET			A/T/AGA	>7	3.24		
49	Rosy	24	36054	primi	37		3.9		IUGR	I	M	CS	FD		A/T/IUGR	<7	2.21	AD	
50	chitra	22	36418	primi	38		3.1		Idiopathic	I	M	CS	FD		A/T/AGA	>7	3.12		
51	Kavitha	28	35707	G3P1L1A1	33		2.7		Idiopathic	S	C	VD			A/PT/IUGR	<7	1.98	AD	D
52	Murugeshwari	21	36370	primi	40+5		5		PD	I	M	CS	CPD		A/T/SGA	<7	2.6	AD	
53	Sundarapriya	26	34180	primi	36	Anemia	4.9		IUGR	S	C	VD			A/PT/IUGR	>7	1.99	AD	D
54	Kasthuri	23	35695	primi	39		4.8		Idiopathic	I	C	VD			A/T/SGA	>7	2.8		
55	Parvathy	34	29276	G2P1L1	40	PE	3.2		PE	I	M	CS	FD		A/T/AGA	<7	3.43		
56	Jameela	25	36372	primi	37		4		Idiopathic	S	M	CS	FD		A/T/SGA	>7	2.12	AD	
57	Gomathy	27	38311	primi	40+4		3.1		PD	I	M	CS	FD		A/T/AGA	>7	3.6		
58	Asha	28	38344	primi	35		4.7		Idiopathic	S	C	VD			A/PT/AGA	<7	1.89	AD	
59	Sundari	22	38302	G3P1L1A1	35	PE	3.5		PE	I	C	VD			A/PT/SGA	<7	2	AD	
60	Andal	20	36364	primi	40+1		5		PD	I	M	CS	FD		A/T/AGA	>7	2.6		
61	Muthumari	23	37029	G2A1	34		3.5		Idiopathic	S	C	VD			A/PT/SGA	<7	1.765	AD	D
62	Jasmin	27	37684	primi	40	GHT	4		GHT	I	M	CS	oblique lie		A/T/AGA	>7	2.46		
63	Uma	24	32207	primi	36		2.9		IUGR	S	C	VD			A/PT/IUGR	>7	2	AD	
64	Dharani	18	3829	primi	40+3		2.7		PD	I	M	CS	FD		A/T/AGA	<7	3.5	AD	
65	Porkodi	31	537	primi	37	Anemia	4		Idiopathic	S	M	CS	FD		A/T/SGA	>7	2.9		

66	Bindhu	22	1060	G2P1L0	40+1		3		PD	I	C	VD			A/T/SGA	>7	3		
67	Kanaga	29	1582	primi	36	PE	2.7		PE	I	M	CS	FD		A/PT/SGA	<7	2.2		
68	Poongodi	24	2201	primi	40+3		3.1		PD	I	M	CS	FD		A/T/AGA	>7	2.38		
69	Mangammal	19	2185	primi	38	PE	3.5		PE	I	C	CS	CPD		A/T/SGA	>7	2.13	AD	
70	Ganageetham	23	2716	primi	35		2.9		Idiopathic	S	M	CS	FD		A/PT/AGA	<7	2.1	AD	
71	Sabeena	30	3442	G3P1L0A1	31	APLA	1.7		APLA	I	C	VD			A/PT/SGA	<7	1	AD	D
72	Malliga	35	3261	primi	40+6		3.7		PD	I	M	CS	FD		A/T/IUGR	>7	2.29	AD	
73	Laisha	23	4412	primi	40		5		Idiopathic	I	M	CS	FI		A/T/AGA	>7	3.35		
74	Jayanthi	28	3260	G2P1L1	36		3		Idiopathic	S	C	OUTLET			A/PT/SGA	>7	2.3		
75	Udhayakumari	21	5948	primi	40+3		2.4		PD	I	M	CS	FD		A/T/IUGR	<7	2.48		
76	Deepadevi	27	6387	primi	38		4.6		Idiopathic	I	M	CS	CPD		A/T/AGA	>7	3.2		
77	Selviammal	25	5898	primi	37	PE	3.9		PE	I	C	CS	FD		A/T/IUGR	<7	2.47	AD	
78	Rajathi	20	5888	primi	40+2		4.8		PD	I	M	CS	FD		A/T/SGA	<7	2.7	AD	
79	samundeeshwari	28	6556	G2A1	34		4.1		Idiopathic	I	C	VD			A/PT/SGA	>7	2		
80	Meenatchi	24	6762	primi	39		4		Idiopathic	I	M	CS	FD		A/T/AGA	<7	3	AD	
81	Vahithabanu	27	7336	primi	40	PE	5		PE	I	M	CS	FD		A/T/AGA	>7	2.5		
82	Sathya	25	7582	primi	30	PE	3.3		PE	I	C	VD			A/PT/SGA	<7	782gms	AD	D
83	Malar	19	7590	primi	40		4.9		IUGR	I	M	CS	FD		A/T/IUGR	>7	2.49	AD	
84	Santhosini	21	7858	G2A1	40+1		4.4		PD	I	M	CS	FD		A/T/AGA	<7	3.1		
85	Jamuna	26	8160	primi	36		3.5		IUGR	S	M	CS	FD		A/PT/IUGR	>7	2.2	AD	
86	Ameenabee	33	9694	G3P2L2	38		4.2		Idiopathic	I	C	VD			A/T/AGA	>7	3.7		
87	Deepa	25	10700	primi	39	PE	4.4		PE	I	M	CS	FD		A/T/SGA	<7	2.46	AD	
88	Venkatama	30	11032	G2P1L1	37		5		Idiopathic	S	M	CS	FD		A/T/AGA	<7	3.15	AD	
89	Geetha	22	10936	primi	40+1		3.7		PD	I	C	VD			A/T/SGA	>7	2.6		
90	Sudhasanthi	27	13004	primi	40+5		1.6		PD	I	C	VD			A/T/IUGR	<7	2.48	AD	
91	Kalaiselvi	24	13108	G2A1	38		4.6		Idiopathic	S	M	CS	CPD		A/T/SGA	<7	2.63		

92	Rasathi	29	13555	primi	39		3.7	Infantile PCKD	Anomaly	I	M	CS	CPD		A/T/SGA	>7	2.1		
93	Parimala	16	14881	primi	28	PE	5		PE	I	C	VD			A/PT/IUGR	<7	796gms	AD	D
94	Latha	21	14844	G2P1L1	40+2		2.7		PD	I	M	CS	FD		A/T/SGA	<7	2.6	AD	
95	Sabeenabegam	19	16823	primi	37		4.2		Idiopathic	S	M	CS	FD		A/T/AGA	<7	3		
96	Renuka	25	18043	G2A1	40+5		4.8		PD	I	C	CS	FD		A/T/SGA	>7	2.2	AD	
97	Padmakumari	27	16470	primi	37		4.6		IUGR	S	M	VD			A/T/SGA	<7	2.4	AD	
98	Umadevi	24	19316	primi	38		5		Idiopathic	I	M	CS	FD		A/T/AGA	>7	2.47	AD	
99	Ragal	29	21065	G3P1L1A1	32		5		Idiopathic	S	C	CS	FD		A/PT/AGA	<7	1.7	AD	
100	Mala	21	21975	primi	39	PE	4.9		PE	I	M	CS	FD		A/T/AGA	>7	2.49		
101	Poonam	23	21056	primi	40	GHT	2.7		GHT	S	C	VD			A/PT/SGA	<7	2.4	AD	
102	Parveen	28	19310	primi	38		5		IUGR	S	M	CS	FD		A/T/IUGR	>7	2.487	AD	
103	sivaroshini	21	20635	primi	40+5		3.6		PD	I	C	CS	FD		A/T/SGA	<7	2.4	AD	
104	Muthulakshmi	32	15963	G3A2	39		5		Idiopathic	S	M	CS	FD		A/PT/AGA	>7	3		
105	Suganya	21	16405	primi	36		5		IUGR	S	C	VD			A/T/IUGR	<7	1.9	AD	
106	Deepa	18	14816	primi	40		5		IUGR	I	M	CS	FD		A/T/IUGR	>7	2.4	AD	
107	Rajeshwari	27	16063	primi	37		4.9		Idiopathic	S	C	CS	CPD		A/T/SGA	<7	2.37		
108	Dhanalakshmi	24	16823	G2P1L1	40+1		3.4		PD	I	M	CS	FD		A/T/SGA	<7	2.8	AD	
109	manimala	26	17173	primi	34		5		IUGR	S	C	VD			A/T/IUGR	>7	2	AD	
110	zahera	25	16410	primi	40+4		3.9		PD	I	M	CS	FD		A/T/SGA	<7	2.7	AD	
111	Vimala	30	18043	G2P1L1	40		1.5		IUGR	I	M	CS	FD		A/T/IUGR	<7	2.46	AD	
112	Thenmozhi	23	17107	primi	38	PE	4.2		PE	I	C	VD			A/PT/AGA	<7	2.6	AD	
113	Priyanka	29	16470	G3P1L1A1	39		1.5		IUGR	S	M	CS	FD		A/T/IUGR	>7	2.39		
114	Datchayani	17	16299	primi	40+4		4.6		PD	I	C	VACCUM			A/T/AGA	<7	3		
115	Sumathy	21	17169	primi	32	PE	3.7		PE	I	C	VD			A/PT/IUGR	<7	997gms	AD	D
116	kalpana	21	15603	G2A1	39		2		IUGR	I	M	CS	FD		A/T/IUGR	<7	2.3	AD	
117	Rajalakshmi	18	20533	primi	37		4.6		Idiopathic	S	C	VD			A/T/SGA	>7	2.5		

118	Mageshwari	26	20676	G2P1L1	38		4.2		IUGR	I	M	CS	FD		A/T/IUGR	<7	2.39	AD	
119	Sudhendra	21	20759	primi	36		4.8		Idiopathic	S	C	CS	FD		A/PT/AGA	<7	2.6	AD	
120	Reka	30	20753	primi	40+1		3.5		PD	I	M	CS	FD		A/T/AGA	>7	3.4		
121	Sindhubharathi	25	20874	G2P1L0	34	PE	3.2		PE	I	M	CS	FD		A/PT/SGA	<7	2.3		
122	Meena	22	20979	primi	40+6		5		PD	I	C	VD			A/T/AGA	>7	3.5		
123	Yuvarani	20	21065	primi	37		2.5		IUGR	S	M	CS	FD		A/T/IUGR	<7	1.9	AD	
124	Sundari	25	20826	G3P2L1	40+1		3		PD	I	M	CS	FD		A/T/IUGR	<7	2.6	AD	
125	Logeshi	18	21329	primi	37		2.9		Idiopathic	S	M	CS	FD		A/T/AGA	<7	2.1	AD	
126	Anusia	24	21534	G2P1L1	38		4.5	single UA	Anomaly	I	C	VD			A/T/sGA	>7	2.67		
127	Rama	31	21615	primi	33	PE	2.3		PE	S	M	CS	FD		A/PT/SGA	>7	2		
128	Porselvi	22	21975	primi	40+1		4.5		PD	I	C	VD			A/T/AGA	<7	2.98		
129	Sofia	24	18538	G3A2	39		5		IUGR	S	M	CS	FD		A/T/IUGR	<7	2.45	AD	
130	Rasmiya	28	18999	primi	40+1		4.3		PD	I	M	CS	FD		A/T/AGA	<7	3		
131	Kumari	21	19310	G2P1L1	36		4.1		IUGR	S	C	VD			A/PT/IUGR	<7	2.51	AD	
132	Pooja	19	19294	primi	40		2.8		Idiopathic	I	M	CS	FD		A/T/AGA	<7	3.2		
133	Amirthavalli	25	17814	G2A1	37		4.3		Idiopathic	I	M	CS	FD		A/T/SGA	<7	2.54		
134	Muniyammal	26	19298	G3P1L1A1	39		4.3		IUGR	S	C	VACCUM			A/T/IUGR	<7	2.74		
135	Therasa	24	19361	primi	40+2		3.9		PD	I	M	CS	FD		A/T/AGA	<7	3.1	AD	
136	Laksmi	21	19276	primi	40+4		5		PD	I	M	CS	FD		A/T/SGA	>7	2.86		
137	Sumathy	20	19832	primi	38		3.2		IUGR	I	C	VD			A/PT/IUGR	<7	1	AD	
138	Priya	23	19974	G2P1L1	40+3		3.6		PD	S	C	OUTLET			A/T/AGA	<7	3.13		
139	Manjula	28	19316	G2P1L1	35	PE	5		PE	I	C	VD			A/PT/IUGR	>7	820gms	AD	
140	Dhanalakshmi	21	20084	primi	40+1		3.2		PD	I	M	CS	FD		A/T/SGA	<7	2.76		
141	Divya	21	19167	primi	40		3.4		Idiopathic	S	C	VD			A/T/SGA	>7	2.68		
142	suganya	27	20335	G2P1L0	36	PE	5		PE	I	M	VD			A/PT/SGA	<7	2.4	AD	
143	Kanmani	24	19876	G2P1L1	39		4.3	Infantile PCKD	Anomaly	S	C	VD			A/T/IUGR	>7	2.72		

144	Ramani	20	19453	primi	39		4.9		Idiopathic	S	C	OUTLET			A/PT/SGA	<7	1.76	AD	
145	Rasathi	22	19492	primi	40+3		3.7		PD	I	M	CS	FD		A/T/AGA	>7	3.4		
146	Tamilselvi	19	20341	primi	38		4.2		Idiopathic	S	C	OUTLET			A/T/SGA	>7	2.54		
147	Growser	23	20781	G2A1	40+6		5		PD	I	C	CS	FI		A/T/AGA	<7	2.6		
148	Nagammal	35	21873	primi	37		3.2		Idiopathic	S	C	CS	FD		A/T/SGA	<7	2.51	AD	
149	Sivasankari	21	21762	primi	40+5		4.1		PD	I	M	CS	FD		A/T/AGA	<7	3.1		
150	Sankari	25	20845	D2P1L1	39		4.4		Idiopathic	I	M	CS	oblique lie		A/T/SGA	>7	2.6	AD	

POLYHYDRAMNIOS

S.NO	NAME	AGE	IP NO	OBSTETRIC SCORE	GA IN WEEKS	ASSOCIATED MATERNAL CONDITIONS	AFI	ETIOLOGICAL FACTORS	ASSOCIATED ANOMALIES	SPONTANEOUS/INDUCED	COLOUR OF LIQUOR	MODE OF DELIVERY	INDICATIONS FOR CESAREAN	LABOUR COMPLICATIONS	FETAL OUTCOME	5 MINT APGAR	BIRTH WEIGHT IN Kg	NICU ADMISSION	PERINATAL DEATH
1	Nabeesha	21	21990	Primi	36	PE	25	Idiopathic		S	C	VD		PTL	A/ PT/AGA	<7	2.6	AD	
2	Muniyammal	26	22552	G2P1L1	39		29	Idiopathic		S	C	VD			A/T/AGA	>7	3		
3	sakthi	20	21853	primi	29	Rh neg	34	Rh neg		I	C	VD		PTL	D				D
4	kalaiyarasi	22	22569	primi	40	GDM	25	GDM		S	M	CS	FD		A/T/AGA	>7	3.4		
5	sundari	31	22790	G3P1L1A1	37		26	Idiopathic		S	C	VD			A/T/AGA	>7	2.8		
6	Deepika	25	22892	Primi	31		25	Idiopathic		S	C	VD		PTL	A/PT/AGA	<7	1.2	AD	
7	sandhiya	21	22983	G2P1L1	38	GHT	27	Idiopathic		S	C	VD		PROM	A/T/AGA	>7	3.4		
8	Rasheeda	25	22917	G5P2L1A2	39		25	Idiopathic		S	C	CS	Cord prolapse	Cord prolapse	A/T/LGA	>7	3.2	AD	
9	lalitha	36	23296	Primi	36		32	Anencephaly	Anencephaly	I	C	VD			D				D
10	Vijayalakshmi	26	23455	G3A2	41		30	Idiopathic		S	M	CS	Oblique lie		A/T/AGA	>7	3.84		
11	Laksmi	29	23646	G2P1L1	40	PE	25	Idiopathic		S	C	VD			A/T/SGA	<7	3.14	AD	
12	devi	24	23657	Primi	30		25	Idiopathic		S	C	VD		PTL	A/PT/AGA	<7	1.1	AD	D
13	Kalpana	27	23771	G2P1L0	37		26	Idiopathic		S	C	Vaccum forceps		PROM	A/T/AGA	>7	2.8		
14	Amuthavalli	33	23686	Primi	37		30	Idiopathic		S	C	CS	CPD		A/T/AGA	>7	3.1		

15	prema	30	23553	G3P1L1A1	38	GDM	28	GDM		S	M	VD			A/T/AGA	>7	3.61			
16	Revathy	35	23836	Primi	34	GDM	30	GDM		S	C	VD		PTL	A/T/AGA	>7	2			
17	Kanniyammal	28	23973	G3P2L2	39		35	Diaphragmatic hernia	Diaphragmatic hernia	S	C	VD			A/T/SGA	<7	3.07	AD	D	
18	Angammal	18	24012	G3P2L1	40		25	Idiopathic		S	C	VD			A/T/AGA	>7	3.1			
19	Yuvarani	30	23945	G5P4L3	32		29	Idiopathic		S	C	VD		Cord prolapse	A/PT/AGA	>7	1.3	AD	D	
20	Nagalakshmi	36	24149	G4p3L3	37		26	Idiopathic		S	C	VD		PROM	A/T/AGA	>7	2.6			
21	Nandhini	31	23619	Primi	38		31	Anencephaly	Anencephaly	I	C	VD		ATONIC PPH	D				D	
22	jayakodi	27	24118	G2P1L0	36	GDM	25	GDM		S	C	VD			A/PT/AGA	>7	2.4			
23	Vedavalli	22	29418	G2P1L1	35		29	Idiopathic		S	C	Outlet forceps		PTL	A/PT/AGA	>7	2.2			
24	Sandhiya	26	23993	G2P1L1	32		25	Spina bifida	Spina bifida	I	C	VD		PTL	D				D	
25	Therasa	20	23333	Primi	39	GHT	26	Idiopathic		S	C	CS	FI	PROM	A/T/AGA	>7	3.09			
26	Rama	38	24359	G4P2L2A1	37		27	Idiopathic		S	C	VD		Cord prolapse	A/T/AGA	<7	3	AD		
27	Archana	29	24359	G3P1L1A1	33		29	Anencephaly	Anencephaly	I	C	VD			D				D	
28	Selvi	34	24829	G3P2L1	40		36	Duodenal atresia	Duodenal atresia	S	C	VD			A/T/SGA	<7	3.8	AD		
29	Parveen	17	24912	Primi	41		25	Idiopathic		S	C	VD		ATONIC PPH	A/T/AGA	>7	4.1			
30	Mary	21	25012	G2A1	39	GDM	32	GDM		S	C	VD			A/T/LGA	>7	3.8			
31	Alamelu	28	25176	G2P1L1	38		28	Idiopathic		S	M	VD			A/T/AGA	>7	2.8			
32	Srisha	23	25337	G4P3L1	40		26	Idiopathic		S	C	Outlet forceps		PROM	A/T/AGA	>7	3.2			
33	Guruvamma	25	25586	Primi	39		25	Hydrocephalus	Hydrocephalus	S	C	VD			D				D	
34	Sigappi	27	25952	G2P1L1	32	Chorioangioma	37	Chorioangioma		S	C	VD		Retained placenta	D				D	
35	Arivumalar	26	26111	Primi	38	GDM	29	GDM		S	M	Vaccum forceps			A/T/AGA	<7	4.1	AD		
36	Mala	19	26300	G2P1L1	41		30	Idiopathic		S	C	VD		PROM	A/T/LGA	<7	3.26	AD		
37	Kavitha	29	26868	G3P1L1A1	40		27	Idiopathic		S	C	VD		PROM	A/TAGA	>7	3.2			

38	Rani	35	26993	Primi	38	GDM	26	GDM		S	C	CS	FD		A/T/AGA	<7	3.46	AD	
39	Shanthi	25	26997	G2P1L1	40		25	Idiopathic		I	C	CS	CPD		A/T/LGA	>7	3.18		
40	Thendral	26	27016	G2P1L0	40	PE	25	Idiopathic		S	C	VD		PROM	A/T/SGA	>7	3.1		
41	Rajasundari	21	26933	Primi	42		30	Idiopathic		I	C	Outlet forceps			A/T/AGA	<7	3.46	AD	
42	Velankani	24	23672	G2P1L0	38		27	Idiopathic		S	C	CS	CPD		A/T/AGA	>7	3.39		
43	Usha	29	24781	G3P1L0A1	40	GDM	29	GDM		S	C	VD		PROM	A/T/AGA	>7	4.1	AD	
44	Karpagam	30	25810	G2P1L1	39		26	Non immune hydrops	Non immune hydrops	I	C	VD			D				D
45	mathew meera	23	21763	G3P2L2	38		27	Idiopathic		S	M	VD			A/T/AGA	>7	3.11		
46	venketramani	22	23908	G2A1	29		25	Hydrocephalus	Hydrocephalus	S	C	VD			D				D
47	Deepa	26	24654	G4P3L2	39		33	Idiopathic		S	C	VD			A/T/AGA	>7	3.95		
48	Vidhyavathi	29	27983	Primi	39		26	Spina bifida	Spina bifida	I	C	VD			D				D
49	Indhu	27	22990	G2A1	40		26	Idiopathic		S	C	CS	Oblique lie	PROM	A/T/AGA	>7	3.9		
50	Vinodhini	30	24321	G2P1L1	39		31	Anencephaly	Anencephaly	I	C	VD			D				D

ABBREVIATIONS

A	**--**	**ALIVE**
AD	**-**	**ADMISSION**
AFI	**-**	**AMNIOTIC FLUID INDEX**
AGA	**-**	**APPROPRIATE FOR GESTATIONAL AGE**
C	**-**	**CLEAR**
M	**-**	**MECONIUM**
T	**-**	**TERM**
PT	**-**	**PRETERM**
CPD	**-**	**CEPHALO PELVIC DISPROPOTION**
FI	**-**	**FAILED INDUCTION**
FD	**-**	**FETAL DISTRESS**
GDM	**-**	**GESTATIONAL DIABETES MELLITUS**
GHT	**-**	**GESTATIONAL HYPERTENSION**
PE	**-**	**PREECLAMPSIA**
VD	**-**	**VAGINAL DELIVERY**
CS	**-**	**CESAREAN SECTION**
S	**-**	**SPONTANEOUS**
I	**-**	**INDUCED**
PTL	**-**	**PRETERM LABOUR**
PROM	**-**	**PREMATURE RUPTURE OF MEMBRANES**
PPH	**-**	**POST PARTUM HEMORR HAGE**
SGA	**-**	**SMALL FOR GESTATION**
IUGR	**-**	**INTRA UTERINE GROWTH RESTRICTION**
D	**-**	**DEATH**
PD	**-**	**POST DATED**
APLA	**-**	**ANTIPHOSPHOLIPID ANTIBODY SYNDROME**